H
×

Duplicate.

This book was exchanged for
a book of greater value.
S.C. Ranner
Librarian

KING'S COLLEGE HOSPITAL
MEDICAL SCHOOL.
-5 DEC 1918

WEC
King's
collection:
Cheyne

200840228 X
KING'S COLLEGE LONDON

512460

KING'S
College
LONDON

KCSMD R129 ALL

Library

ALLBUTT, THOMAS CLIFFORD
A SYSTEM OF MEDICINE
1905-1911

KING'S COLLEGE HOSPITAL
LIBRARY
Medical School

A SYSTEM OF MEDICINE

BY MANY WRITERS

8vo. Half-bound.

Second Edition, edited by Sir CLIFFORD ALLBUTT, *K.C.B., M.D., F.R.S., and* H. D. ROLLESTON, *M.D., F.R.C.P.*

Vol. I. Prolegomena and Infectious Diseases. 25s. net.

II. Part I. Infectious Diseases (*continued*), Intoxications. 25s. net.

II. Part II. Tropical Diseases and Animal Parasites. 25s. net.

III. Certain General Diseases, Diseases of the Alimentary Canal and Peritoneum. 25s. net.

IV. Part I. Diseases of the Liver, Pancreas, Ductless Glands, and Kidneys. 25s. net.

IV. Part II. Diseases of the Nose, Pharynx, Larynx, and Ear. 25s. net.

V. Diseases of the Respiratory System, Disorders of the Blood. 25s. net.

VI. Diseases of the Heart and Blood-Vessels. 25s. net.

VII. Diseases of the Muscles, the Trophoneuroses, Diseases of the Nerves, Vertebral Column, and Spinal Cord. 25s. net.

VIII. Diseases of the Brain and Mental Diseases. 25s. net.

IX. Diseases of the Skin. 25s. net.

MACMILLAN AND CO., LTD., LONDON.

A SYSTEM OF MEDICINE

MACMILLAN AND CO., LIMITED
LONDON · BOMBAY · CALCUTTA
MELBOURNE

THE MACMILLAN COMPANY
NEW YORK · BOSTON · CHICAGO
ATLANTA · SAN FRANCISCO

THE MACMILLAN CO. OF CANADA, LTD.
TORONTO

K G'S C LEGE HOSPITAL
MEDICAL SCHOOL.

A
SYSTEM OF MEDICINE

BY MANY WRITERS

KING'S COLLEGE HOSPITAL
LIBRARY
MEDICAL SCHOOL

EDITED BY

SIR CLIFFORD ALLBUTT, K.C.B.
M.A., M.D., LL.D., D.SC., F.R.C.P., F.R.S., F.L.S., F.S.A.
REGIUS PROFESSOR OF PHYSIC IN THE UNIVERSITY OF CAMBRIDGE
FELLOW OF GONVILLE AND CAIUS COLLEGE

AND

HUMPHRY DAVY ROLLESTON
M.A., M.D., F.R.C.P.
SENIOR PHYSICIAN, ST. GEORGE'S HOSPITAL; PHYSICIAN TO THE VICTORIA HOSPITAL
FOR CHILDREN; SOMETIME FELLOW OF ST. JOHN'S COLLEGE, CAMBRIDGE

VOLUME VIII

DISEASES OF THE BRAIN AND MENTAL DISEASES

MACMILLAN AND CO., LIMITED
ST. MARTIN'S STREET, LONDON
1911

512460
KCSMD

KING'S COLLEGE

First Edition 1899
Second Edition 1910, 1911

WEC King's collection - Cheyne

H
x
2119 x
27.4.56

From the library of
Dr. Willoughby Lyle.

KING'S COLLEGE HOSPITAL
LIBRARY
Medical School

A

PREFACE

THIS volume contains the Diseases of the Brain and Mental Diseases, and differs from Vol. VIII. of the first edition in not containing the Diseases of the Skin, which will form the subject-matter of the concluding volume (IX.) of this *System.*

Under the heading Meningitis Dr. Batten has contributed a new article dealing with all forms of meningitis except the tuberculous; in it he has made use of the statistics and illustrations given in Dr. Lees and Sir Thomas Barlow's elaborate article on Simple ("posterior-basic") Meningitis in the first edition. Other new articles in this volume are those on Acute Polioencephalitis by Dr. F. E. Batten, on Recurrent Paralysis by Professor J. Michell Clarke, and on Apraxia and Agnosia by Dr. James Collier, who has also revised Dr. Bastian's article on Aphasia. Dr. Head has rewritten the account of Occupation-Neuroses formerly contributed by the late Dr. Vivian Poore. The article on Neurasthenia has been largely rewritten and expanded.

To Dr. Henry Head and to Dr. Savage the Editors are under a deep obligation for their generous help and advice in the sections on Nervous Diseases and Mental Diseases respectively. As in the past Dr. A. J. Jex-Blake has given the Editors much assistance in the detection of misprints.

CLIFFORD ALLBUTT.
H. D. ROLLESTON.

CONTENTS

DISEASES OF THE NERVOUS SYSTEM *(continued)*

PAGE
EXPERIMENTAL PATHOLOGY OF THE CEREBRAL CIRCULATION. Dr. Leonard Hill 3
THE REGIONAL DIAGNOSIS OF CEREBRAL DISEASE. Sir D. Ferrier, M.D. . 37

DISEASES OF THE CEREBRAL MENINGES

MENINGITIS. Dr. F. E. Batten 165
TUBERCULOUS MENINGITIS. Sir T. Barlow, Bt., M.D. 201

DISEASES OF THE BRAIN

SOME MILDER FORMS OF CONCUSSION. Sir William Bennett 231
ACUTE POLIOENCEPHALITIS. Dr. F. E. Batten 241
ABSCESS. Dr. Byrom Bramwell 250
INTRACRANIAL TUMOURS. Dr. Byrom Bramwell 266
OCCLUSION OF THE CEREBRAL VESSELS. Dr. James Taylor . . . 290
HAEMORRHAGE. Dr. Howard H. Tooth 307
INTRACRANIAL SYPHILIS. Dr. W. Aldren Turner 334
GENERAL PARALYSIS OF THE INSANE. Dr. G. H. Savage, Dr. E. Goodall . 346

APHASIA AND OTHER SPEECH DEFECTS. Dr. H. Charlton Bastian. Revised by Dr. James Collier 385
APRAXIA AND AGNOSIA. Dr. James Collier 447

CEREBRAL PALSIES OF CHILDREN. Dr. James Taylor 452
AMAUROTIC FAMILY IDIOCY. Dr. J. S. Risien Russell 468

PARALYSIS AGITANS. Sir William R. Gowers, M.D. 473
EPILEPSY. Sir William R. Gowers, M.D. 483
CHOREA. Dr. J. S. Risien Russell 519

PAGE
Migraine. Dr. James Mackenzie 558
Impediments of Speech. Dr. W. S. Colman 569
Tetany. Dr. J. S. Risien Russell 578
Recurrent Paralysis. Prof. J. Michell Clarke, M.D. . . . 604
Tics. Dr. J. S. Risien Russell 614
Facial Spasm. Dr. J. S. Risien Russell 638
Paramyoclonus Multiplex. Dr. J. S. Risien Russell 649
Eclampsia Nutans. Dr. J. S. Risien Russell 659
Head-Nodding. Dr J. S. Risien Russell 661
Occupation-Neuroses. Dr. H. Head 667
Hysteria. Dr. J. A. Ormerod 687
Neurasthenia. Sir Clifford Allbutt, M.D. 727
Disorders of Sleep. Prof. J. B. Bradbury, M.D. 791
Night-Terrors. Dr. Leonard G. Guthrie 804

MENTAL DISEASES

Introduction. Dr. Savage 823
Vice, Crime, and Insanity. Dr. Mercier 842
Idiocy and Imbecility. Dr. Shuttleworth and Dr. Fletcher Beach. Revised
by Dr. Shuttleworth 874
Mania. Dr. Savage 892
Acute Delirium. Dr. R. Percy Smith 899
Toxic Insanities. Dr. Savage 904
Alcoholic Insanity. Dr. Hyslop 912
Insanity and Surgical Operations. Mr. Clinton Dent 921
Epochal Insanities. Dr. Clouston 926
Insanity and Epilepsy. Dr. Robert Jones 941
Recurrent Insanity. Dr. Urquhart 961
Melancholia and Hypochondriasis. Dr. H. Rayner 964
Mental Stupor and Catalepsy. Dr. Savage 984
States of Mental Weakness. Dr. Urquhart 989
Systematised Delusional Insanity. The late Dr. Conolly Norman. With
an Addendum by Dr. R. Percy Smith 995
Hypnotism: and the Treatment of Insanity and Allied Conditions
by Suggestion. Dr. Milne Bramwell 1007
Criminal Lunacy in England. Dr. David Nicolson 1017
English Law and the Practice of Lunacy. Dr. T. Outterson Wood and
Dr. Savage 1040

INDEX 1051

ILLUSTRATIONS

PLATES

No.
I. Base of the Brain in the Sporadic Form of Meningococcic ("Posterior-Basic") Meningitis. (Lees and Barlow) . *To face page* 168
II. Ocular Phenomena in the Field of Vision during an Attack of Migraine . . . . " " 563

FIGURES

FIG. PAGE
1, 2. Transverse Sections of Arteries, Contracted and Dilated. (MacWilliam) . 11
3. Cerebral Hemisphere, Lateral Aspect. (Symington) 39
4. Cerebral Hemisphere, Mesial Aspect. (Symington) 39
5. Histological Areas of the Cortex, Lateral Aspect. (Campbell) . . 42
6. Histological Areas of the Cortex, Mesial Aspect. (Campbell) . . 42
7. Motor Areas of the Cortex, Lateral Aspect. (Horsley and Schäfer) . . 52
8. Motor Areas of the Cortex, Mesial Aspect. (Horsley and Schäfer) . . 52
9, 10. Motor Areas of the Chimpanzee. (Grünbaum and Sherrington) . . 54
11. Brain in Meningococcic Meningitis 168
12. Meningococcic Meningitis with Retraction of the Upper Eyelids. (Thursfield) 174
13-16. Meningococcic Meningitis with Head-Retraction and other Signs. (Lees and Barlow) 175, 176, 177
17-19. Syphilitic Meningitis 194, 195, 196
20. Diagram of the Approximate Sites of the Four Word-Centres and their Commissures 387
21. Diagram of the Relative Positions of the Four Word-Centres and their Connexions by Commissures 389
22. Diagram of the Approximate Sites of the Four Word-Centres in the Left Cerebral Hemisphere 425
23. Diagram representing Dejerine's view as to the Mode of Production of Pure Word-Blindness 436
24. Diagram of the Mode of Production of Pure Word-Blindness. (Bastian) . 437
25. Nephroptosis in Neurasthenia 755
26. Photographs of Mongolian and Cretinoid Idiots 879
27. Reflex Cerebral Circuit. (Ramón y Cajal) 949

LIST OF CONTRIBUTORS TO THIS VOLUME

Allbutt, Sir Clifford, K.C.B., M.D., LL.D., D.Sc., F.R.C.P., F.R.S., Regius Professor of Physic in the University of Cambridge ; Fellow of Gonville and Caius College ; Hon. Fellow of the Royal College of Physicians of Ireland, and of the Academy of Medicine of New York.

Barlow, Sir Thomas, Bt., K.C.V.O., LL.D., M.D., D.Sc., F.R.S., Physician Extraordinary to H.M. the King ; President of the Royal College of Physicians, London ; Consulting Physician to University College Hospital, and to the Hospital for Sick Children, Great Ormond Street.

Bastian, H. Charlton, M.D., F.R.S., Consulting Physician to University College Hospital, and to the National Hospital for the Paralysed and Epileptic.

Batten, Frederick E., M.D., F.R.C.P., Physician for Out-patients, the National Hospital for the Paralysed and Epileptic ; Physician, the Hospital for Sick Children, Great Ormond Street.

Beach, Fletcher, M.B., F.R.C.P., Consulting Physician, West End Hospital for Diseases of the Nervous System ; late Medical Superintendent, Darenth Asylum.

Bennett, Sir William, K.C.V.O., F.R.C.S., Consulting Surgeon, St. George's Hospital.

Bradbury, J. B., M.D., F.R.C.P., Downing Professor of Medicine in the University of Cambridge ; Senior Physician, Addenbrooke's Hospital.

Bramwell, Byrom, M.D., F.R.C.P.Ed., Senior Ordinary Physician and Lecturer on Clinical Medicine, Edinburgh Royal Infirmary.

Bramwell, J. Milne, M.B., C.M., formerly Assistant Surgeon to the Perth City and County Infirmary.

Clarke, J. Michell, M.D., F.R.C.P., Physician, Bristol General Hospital ; Professor of Medicine in the University of Bristol.

Clouston, T. S., M.D., F.R.C.P., formerly Physician-Superintendent, Royal Edinburgh Asylum, and Lecturer on Mental Diseases, University of Edinburgh.

Collier, James, M.D., F.R.C.P., Physician, St. George's Hospital ; Physician for Out-patients, the National Hospital for the Paralysed and Epileptic.

Colman, Walter Stacy, M.D., F.R.C.P., Physician to Out-patients, St. Thomas's Hospital ; Physician, the Hospital for Sick Children, Great Ormond Street.

Dent, Clinton T., M.C., F.R.C.S., Senior Surgeon, St. George's Hospital.

Ferrier, Sir David, M.D., LL.D., F.R.S., Consulting Physician to King's College Hospital, and to the National Hospital for the Paralysed and Epileptic.

Goodall, Edwin, M.D., F.R.C.P., Medical Superintendent, Cardiff Mental Hospital.

Gowers, Sir William R., M.D., F.R.S., Consulting Physician to University College Hospital, and to the National Hospital for the Paralysed and Epileptic ; Fellow of University College, London.

Guthrie, Leonard G., M.D., F.R.C.P., Physician to the Hospital for Paralysis and Epilepsy, Maida Vale, and to the Children's Hospital, Paddington Green.

Head, Henry, M.D., F.R.S., Physician, London Hospital; Editor of *Brain*.

Hill, Leonard, M.B., F.R.S., Lecturer on Physiology, London Hospital Medical School.

Hyslop, T. B., M.D., Resident Physician, Bethlem Royal Hospital; Lecturer on Mental Diseases at St. Mary's Hospital, and at the London (Royal Free Hospital) School of Medicine for Women.

Jones, Robert, M.D., F.R.C.P., F.R.C.S., Resident Physician and Superintendent, London County Asylum, Claybury; Lecturer on Mental Diseases, St. Bartholomew's Hospital.

Mackenzie, James, M.D., LL.D., M.R.C.P., Hon. Consulting Medical Officer, Victoria Hospital, Burnley; Physician, West End Hospital for Diseases of the Nervous System.

Mercier, Charles A., M.D., F.R.C.P., Physician for Mental Diseases and Lecturer on Insanity, Charing Cross Hospital.

Nicolson, David, C.B., M.D., Lord Chancellor's Visitor in Lunacy; late Superintendent, Criminal Lunatic Asylum, Broadmoor.

Norman, the late Conolly, F.R.C.P.I., Medical Superintendent, Richmond Asylum, Dublin.

Ormerod, Joseph Arderne, M.D., F.R.C.P., Physician to St. Bartholomew's Hospital, and to the National Hospital for the Paralysed and Epileptic; sometime Fellow of Jesus College, Oxford.

Rayner, Henry, M.D., late Superintendent Male Department, Hanwell Lunatic Asylum, and formerly Lecturer on Psychological Medicine, St. Thomas's Hospital.

Russell, J. S. Risien, M.D., F.R.C.P., Physician to University College Hospital, and to the National Hospital for the Paralysed and Epileptic.

Savage, G. H., M.D., F.R.C.P., Consulting Physician for Mental Diseases, Guy's Hospital; formerly Physician to Bethlem Royal Hospital.

Shuttleworth, G. E., M.D., Hon. Consulting Physician, Royal Albert Asylum for Idiots, Lancaster, and Metropolitan Association for the Feeble-Minded.

Smith, R. Percy, M.D., F.R.C.P., late Resident Physician and Medical Superintendent, Bethlem Royal Hospital; Physician for Mental Diseases and Lecturer on Psychological Medicine, St. Thomas's Hospital.

Taylor, James, M.D., F.R.C.P., Physician, the National Hospital for the Paralysed and Epileptic; Physician, Royal London Ophthalmic Hospital; Consulting Physician, Queen's Hospital for Children.

Tooth, Howard H., C.M.G., M.D., F.R.C.P., Physician to St. Bartholomew's Hospital, and to the National Hospital for the Paralysed and Epileptic; Consulting Physician to the Metropolitan Hospital.

Turner, William Aldren, M.D., F.R.C.P., Physician, King's College Hospital; Physician for Out-patients, the National Hospital for the Paralysed and Epileptic.

Urquhart, A. R., M.D., F.R.C.P.Ed., Physician Superintendent, James Murray's Royal Asylum, Perth.

Wood, T. Outterson, M.D., F.R.C.P.Ed., Consulting Physician, West End Hospital for Diseases of the Nervous System.

In order to avoid frequent interruption of the text, the numbers indicative of items in the lists of "References" are only inserted in cases of emphasis, where two or more references to the same author are in the list, where an author is quoted from a work published under another name, or where an authoritative statement is made without mention of the author's name. In ordinary cases an author's name is sufficient indication of the corresponding item in the list.

DISEASES OF THE NERVOUS SYSTEM

(CONTINUED)

THE EXPERIMENTAL PATHOLOGY OF THE CEREBRAL CIRCULATION

By Leonard Hill, M.B., F.R.S.

The Cerebral Pulsation.—As early as 1750, knowledge of the respiratory nature of the oscillations of the brain volume was established by Schlichting.

In the last forty years the movements of the brain have been accurately recorded by Salathé, François-Franck, Mosso, and many others. Records have been taken from men with cranial defects, and from infants by way of the fontanelle. The tracings shew cardiac and respiratory oscillations. The pulse curve is usually anacrotic; that is to say, a small wave marks the ascending limb of the curve. This anacrotic wave is probably produced by reflection of the primary wave from the wall of the cranium. It has been asserted that the brain cannot pulsate in the closed cranium, on the ground that the brain fills the rigid, unyielding box of the skull in which it lies enclosed. The inaccuracy of such a statement can easily be shewn by the following means:—After the skull of a dog has been trephined and the dura opened, a piece of metal tubing, on the lower end of which a thin rubber membrane is tried, is screwed into the trephine hole. This membrane, after the introduction of the tube, lies in apposition with the brain. To the other end of the metal tube is fastened a piece of glass tubing of fine bore. This glass tube is filled with water containing a bubble of air, to act as an index. In its turn it is attached, by a T-tube, to a pressure bottle and a mercury manometer. Now, in using this simple apparatus, the position of the air-bubble index is marked on the glass tube when the apparatus is held in the horizontal position, and the pressure is nil. The gauge is then screwed into the skull, and the air-index, which at first is forced out, is brought back to its original position by raising the pressure bottle; that is to say, the brain, which is slightly bulged outwards into the trephine hole, is forced into that flattened position which it naturally must assume against the closed cranial wall. The pressure of the cranial contents against the skull-wall is exactly balanced, and the skull becomes once more a closed cavity, and on this procedure the elevation of the manometer gives the normal intracranial tension. The air-index will then exhibit cardiac and respiratory undulations of intracranial pressure.

The diastole and systole of the brain are made possible in two ways:—

(1) By the ebb and flow of the cerebrospinal fluid. The occipito-atlantal and other vertebral ligaments may extend in cerebral diastole, and allow the fluid to escape from the cranial cavity. The pulsation of the fluid can easily be observed by trephining the lamina of the atlas, screwing a tube therein, and connecting this tube with a water manometer. This mechanism is of little importance. Elder has shewn that when a small quantity of Prussian blue is injected into the subarachnoidal space, and the animal is killed a short time afterwards, the blue matter is found not to have shifted its position. Thus there cannot be any marked translation of cerebrospinal fluid synchronous with the cerebral movements.

(2) By expression of the cerebral veins. In the dog it is a simple operation to screw a tube into the torcular Herophili. When this tube is connected with a water manometer the cerebral venous pressure can be recorded. Such a venous manometer exhibits all the cardiac and respiratory oscillations, the latter in a very well marked way.

The brain is an organ which, like any other organ of the body, pulsates with each stroke of the heart; but, owing to its peculiar condition, enclosed as it is in an unyielding box, the cardiac pulse is not entirely spent in distending the arterioles and capillaries, but is transmitted to the venous sinuses. The brain, expanded by the stroke of the cerebral arteries, expresses blood from the cerebral veins into the sinuses. The respiratory oscillations are caused by the variations of pressure in the right side of the heart. In expiration the blood is dammed back into the sinuses; in inspiration the veins are emptied by the suction of the thorax.

The tracings taken from the fontanelle of the normal infant shew that the brain, like the kidney or spleen in an oncometer, presents respiratory oscillations of pressure, the rise generally being inspiratory. Whether the rise is inspiratory or expiratory depends on the relative effect of the inspiratory rise of arterial pressure or expiratory rise of venous pressure, and on the character of the breathing which modifies the period of the respiratory effect on the arterial pressure (T. Lewis).

During forced breathing the brain may expand more during expiration than during inspiration. This is due to the absence of efficient valves in the cranial and vertebral veins, and to the immediate continuity of these veins with the right auricle and with the venae cavae. If the torcular Herophili be opened in the freshly killed animal, and the abdominal and thoracic veins be compressed, venous blood can be driven out of the torcular in a continuous stream. Likewise, in the human subject, fluid can be driven from the femoral vein out of the cerebral sinuses.

Pathologically the movements of the brain can become increased or diminished. Any cause that produces increased tension of the dura may, when the skull is trephined, stop the apparent pulsation of this membrane.

As the pulsation of the brain takes place always in the direction of least resistance, the brain pulsation is but slightly marked in conditions of normal pressure; that is to say, when the heart and the respiration run smoothly. If the skull be trephined during the condition of acute cerebral compression, the pulsation may be visibly increased owing to the slow powerful beats of the heart, and the stertorous breathing.

The pulsation may be imperceptible when the brain is bound down by adhesions in the neighbourhood of the trephine hole; and, again, whenever the cardiac systole is feeble and the respiration shallow.

It is to be concluded, then, that—(i.) The pulsations of the brain exist in the closed cranium. (ii.) These pulsations are of cardiac and respiratory origin. (iii.) The cardiac pulse is transmitted from the cerebral arteries to the cerebral venous sinuses. (iv.) The brain normally swells with the inspiratory rise of arterial blood-pressure, but owing to the open connexion of the venous sinuses with the venae cavae it becomes expanded during expiration when the breathing is deepened. (v.) The cardiac systolic expansion of the brain within the closed cranium is rendered possible by the ebb of the cerebrospinal fluid into the less rigid vertebral canal, and by the expulsion of the blood from the cerebral veins and sinuses. (vi.) Increased tension of the dura mater decreases its exhibition of the cerebral pulse. (vii.) On opening the dura, provided the brain movement be unrestricted by local adhesions, the extent of the pulsation will depend on the magnitude of the cardiac oscillations of arterial pressure, and on the depth of the respiratory movements.

The Cerebrospinal Fluid.—Neither the brain nor the spinal cord possesses true lymphatic vessels; the lymph finds its way out of these organs by means of perivascular spaces in the tunica adventitia of the blood-vessels; these perivascular spaces communicate on the one hand with the cell-spaces, on the other hand with the subarachnoidal space.

The central canal of the cord and the ventricles of the brain are connected with the subarachnoidal space by the foramen of Majendie, and two other foramina in the lateral recesses of the fourth ventricle, by the side of the flocculus cerebelli. The subarachnoidal space is continuous, extending throughout the central nervous system; anatomically, it is separated from the subdural space. The subarachnoidal space is in continuity with the lymph-spaces in the optic, auditory, and other nerves. The anatomical lack of continuity between the subarachnoidal and the subdural spaces is not physiologically important; for it is only by very gradual and most careful injections of coloured gelatin that the continuity of the true cerebral with the spinal subarachnoidal space can be shewn. On opening the dura in the living animal, with the utmost care, the fluid within the cranium, if any, is seen to be in the subdural space. Saline or serum injected subdurally into the cranium at a pressure just above intracranial pressure runs with ease into the subarachnoidal space of the vertebral column.

The plates in most of the anatomical works of to-day, copied from Key and Retzius' monograph, give an erroneous idea of the contents of the cranium in the living animal. Therein we find the arachnoidal cisterns at the base of the brain distended with an artificial injection, and the tentorium cerebelli pushed upwards and out of place. In the living animal the chief content, besides the brain substance, is blood, not cerebrospinal fluid. The living normal brain, with its circulating blood, almost entirely fills the cranium, and the fluid that moistens its surfaces is little more in amount than the synovial fluid in a joint.

The cerebral subarachnoidal space is a potential rather than an actual space, except in those few places where inequalities of the brain surface are rounded off by small collections of fluid beneath this membrane. In the vertebral canal the fluid is relatively more in amount, and by its presence there it wards off compression from the spinal cord during the flexions and torsions of the vertebral column.

The hydrostatic pressure of the cerebrospinal column of fluid during changes of posture has been supposed to be of importance. This is not so, for the pressure in the blood-vessels under the influence of gravity varies in the same sense as the pressure of the cerebrospinal fluid. Moreover, the fluid passes into the veins at any pressure above venous pressure. Thus in the erect posture the lumbar portion of the cord cannot be rendered anaemic by the hydrostatic pressure of the column of cerebrospinal fluid.

My experiments on the behaviour of fluids injected into the craniovertebral cavity have led to results of interest. On trephining the lamina of a vertebra and screwing in a tube, normal saline can be driven into the vertebral canal. If a second trephine hole be made in the parietal region none of the injection into the vertebral canal can be driven out from this hole; the mid-brain is pushed up by the fluid into the isthmus of the tentorium cerebelli and plugs this up, while the great brain likewise moves upwards and plugs the trephine hole in the parietal region. If, on the other hand, the saline be injected into the parietal hole, it can, in most cases, be driven through the cranio-vertebral canal and out of the hole in the vertebral column. The entire central nervous system can thus be irrigated freely. In some cases, when the pressure is low, and nearly always, if the pressure of the injecting fluid be high and applied suddenly, this cannot be done. By the pressure of the fluid the cerebrum is driven into the isthmus of the tentorium cerebelli, and blocks this up so that no fluid can be expelled. If the cerebrum be removed from the cranium of a dog, and the empty cerebral chamber be filled with water, none of the fluid leaks through the foramen magnum. This shews how completely the cerebellar chamber is filled by the brain mass within it.

Saline or serum injected at any pressure above the cerebral venous pressure is found to disappear from the cranio-vertebral cavity; the higher the pressure the more rapid its disappearance. If the saline be coloured with methylene blue, the fluid can be traced passing straight into the cerebral veins and venous sinuses. In so short a time as five to ten minutes the blue colour may be found excreted into the stomach or bladder. In this time the lymphatics of the neck are not coloured, and not until the lapse of an hour's steady injection do the deep cervical lymphatic glands begin to be tinged with colour. It is clear that the rapid absorption of fluid from the cranio-vertebral cavity takes place by means of the veins and not by way of the lymphatics. This statement holds equally true for the pleural and peritoneal cavities. Fluid will not remain for any length of time in the cranio-vertebral cavity at a pressure above the cerebral venous pressure. This is a fact of no little importance in the pathology of cerebral compression.

In the normal healthy state the amount of cerebrospinal fluid is very slight. On opening the occipito-atlantal membrane in the dog but a few cubic centimetres at the most will be obtained. By continuously tapping off the fluid Falkenheim and Naunyn obtained from dogs 36 to 240 cubic centimetres in twenty-four hours, but as soon as the cranio-vertebral cavity is opened the conditions are no longer normal. After such opening, the rate of transudation of the fluid seems to depend directly on the cerebral capillary pressure. The cerebrospinal fluid is normally maintained at the pressure in the cerebral veins. If the fluid be allowed to escape from the cranio-vertebral cavity, the pressure within this cavity then falls to zero, while the cerebral capillary pressure remains unaltered. Consequently transudation from the capillaries becomes continuous, hence the large amount of fluid obtained in some cases of fracture of the skull. The fluid escapes just as any raw surface weeps, as, for example, after an abrasion of the skin. Since high arterial pressure increases transudation, and high venous pressure prevents absorption, these are the most favourable conditions for increasing the outflow of cerebrospinal fluid from a cranial opening. There is probably a secretion of cerebrospinal fluid from the choroid plexuses in the ventricles of the brain, whence it escapes through the foramen of Majendie at the apex of the fourth ventricle into the subarachnoid space at each cardiac systole. Pettit and Girard have demonstrated the secretory functions of the epithelium which covers the choroidal plexus (*vide* Vol. VII. p. 267).

The cerebrospinal fluid should be regarded as the lymph of the brain, its secretion and resorption being explained on the same lines as other lymphatic exudations. In the closed cranio-vertebral cavity the cerebrospinal fluid and the cerebral capillary pressure are one and the same, and there is no question of the fluid being a filtration dependent on capillary pressure. No such filtration-pressure can exist in any of the organs of the body. The cerebrospinal fluid is a secretion dependent on the relative osmotic and surface energy of the tissue-cells and blood, which vary ceaselessly with the chemical processes of living protoplasm. Such a mechanical process as filtration has no part in the wet protoplasmic films formed by capillary wall, blood, tissue-space, and tissue-cell. In infective states the osmotic conditions are altered by the effect of the chemical interaction of toxins and cell protoplasm, and the balance of surface tension and energy of cell and capillary changed by the vaso-dilatation produced by the toxic agents. A marked lymphocytosis occurs, and the percentages of albumin and globulin increase; the capillary pressure is raised locally and the fluid secreted at a higher pressure; and such altered states may lead, if slowly developed, to hydrocephalus, with expansive growth of the skull in the young, to oedema and suppuration and symptoms of brain pressure if the inflammation is acute.

Professor Halliburton and others have found that cerebrospinal fluid, drawn fresh from the subarachnoidal space, possesses a peculiar chemical composition. The presence of a reducing sugar is a characteristic feature. It contains a few small lymphocytes, traces of albumin and glucose, and a little albumose. The fluid does not clot. The specific gravity ranges from 1005 to 1010. The principal constituent is sodium chloride; there are traces of phosphates and carbonates, and of urea. The inorganic salts = about 8 per 1000; the organic matter = about 1 to 5 per 1000. The fluid does not coagulate on boiling, it gives a precipitate with cold nitric acid, and reduces Fehling's solution. The urea may be increased in cases of uraemia, and the sugar in diabetes. The average amount of fluid obtained from the corpse is 120–150 c.c.; it is increased when there is brain atrophy.

If the fluid be drawn off again and again it changes in character, and more nearly approximates to the composition of serum. The same change follows the repeated tappings of the sacs of spina bifida and hydrocephalus.

In a case which I examined with Dr. St. Clair Thomson, cerebrospinal fluid continually dripped from the nose. In all probability the fluid escaped from some perforation in the plate of the ethmoid bone. This fluid, according to the analysis of Professor Halliburton, possessed the characteristic properties of cerebrospinal fluid. When the patient was made to strain, as at stool, the rate of flow of the fluid was doubled. On qualitative analysis it was found by Halliburton that the percentage of total solids in the fluid passed during straining was less than half that passed when the patient remained passive. The same result followed compression of the patient's abdomen. This case confirms the conclusion, arrived at previously, that the secretion, so soon as the cranium is opened, becomes an exudation which depends on the pressure within the cerebral capillaries. Either the act of straining, or passive compression of the abdomen, markedly raises the general venous, and thus the cerebral capillary, pressure.

From the above considerations it may be concluded that:—(i.) The brain, with its circulating blood, almost entirely fills the cranial cavity in the living animal. (ii.) Fluid escapes directly into the cerebral veins from the subarachnoidal or subdural spaces at any pressures above cerebral venous pressure. (iii.) So long as there is cerebrospinal fluid freely moving within the meningeal spaces, the pressure of this fluid cannot be other than that of the cerebral veins. (iv.) The cranio-vertebral cavity can be irrigated with normal saline at a low pressure. This can be effected through one trephine hole without any counter-opening, and is a perfectly harmless operation. (v.) No local pathological increase of cerebral pressure can be transmitted by the cerebrospinal fluid to distant parts, because this fluid can never be retained in the meningeal spaces at a pressure higher than that of the cerebral veins. (vi.) It is thus possible that hydrocephalic distension of the ventricles of the brain may be relieved by establishing a drain into the subdural space. (vii.) The cerebrospinal fluid preserves its peculiar constitution under normal conditions of pressure only. If it be drawn off, its place is taken by a serous exudation. The rate of transudation when the cranio-vertebral cavity is opened depends directly on the difference between cerebral capillary and atmospheric pressures. Under normal conditions the cerebrospinal fluid is a secretion depending on the complex interaction of blood and tissue-cells.

The Principles of the Cerebral Circulation.—At first sight it would appear that the quantity of blood within the cranium must be almost invariable, "for being enclosed in a case of bone, the blood must be continually flowing out of the veins that room may be given to the blood which is entering by the arteries" (Monro). "In such a cavity as the cranium the blood cannot be diminished below a certain quantity, unless something entered in to supply its place, and, in the language of the old philosopher, prevent a vacuum" (Abercrombie).

The only substance which can enter into or forsake the cranium, when the blood quantum varies, is the cerebrospinal fluid. Now the actual amount of this fluid free in the meningeal spaces and ventricles of the brain is of insignificant volume. "It is well known that there is found very little of the cerebrospinal fluid—in fact, in general little more than what is sufficient to moisten the surface of the membranes—in the interior of the cranium in healthy persons up to the middle of life. In these circumstances the quantity of cerebrospinal fluid that could be displaced in the interior of the cranium must be trifling."

A condition under which the cerebrospinal fluid is increased in amount is atrophy of the brain substance. A long continuance of low arterial pressure when the head is elevated above the body may produce the same result, for the pressure of the blood in the cerebral vessels under the influence of gravity may become negative, and the circulation through the brain fail. As the capillary walls and tissue-cells become damaged, serous fluid passes through, and the vessels collapse as the serous fluid increases. In some men who died of cold in a snowstorm Kellie found more than four ounces of serous fluid in the ventricles of the base of the brain.

Under pathological conditions the quantity of blood in the brain, just as in the marrow, both being enclosed in similar rigid cases of bone, can vary by shrinkage of the tissues. Water and tissue substance may pass out from the tissue-cells owing to altered chemical and osmotic states, and the amount of blood in the dilated vessels be thereby greatly increased. In the normal conditions such increase of blood at expense of tissue fluid, or vice versa, must be happening with variations of functional activity, but to a far slighter extent.

The examination of the condition of the cerebral vessels after death is a very deceptive method of obtaining information as to the amount of blood in the brain prior to death. After death conditions alter: serous fluid or gas may pass out of the vessels into the cranium ; by alteration in the position of the body, or pressure on the abdomen and thorax, the cerebral vessels can be injected with venous blood, the arteries emptied, and the cerebrospinal fluid expelled. For example, the ordinary conditions of the circulatory system which are found after death from asphyxia can be entirely removed by the employment of vigorous artificial respiration on the dead body. These are facts too often neglected.

The Monro-Kellie doctrine—that the blood quantum of the brain cannot vary rapidly in the living animal—is easily verified. If the skull be trephined, and the dura opened, a glass window can be screwed into the hole. On then placing the animal in the vertical feet-down position, and inhibiting the heart, the brain does not collapse away from the window, although the arterial pressure falls to zero. On the other hand, if the glass window be faultily placed and allow leakage into the cranial cavity, air will pass within, the blood be expressed, and the brain collapse under atmospheric pressure. Dr. J. S. Bolton has shewn that the actual quantity of blood in the brain, found after death, is influenced little whether the corpse had been suspended head down, or in the standing posture, or laid horizontal for some hours, so long as the skull is unopened.

While the cranium is closed, large alterations of blood-volume are impossible (except by shrinkage or expansion of brain-tissue) ; in the open cranium the atmospheric pressure is brought to bear upon the cerebral capillaries, and the physical conditions of the cerebral circulation are altered. The cerebral capillaries are emptied of blood if air can freely enter the cranial cavity, when the arterial pressure falls below that which is required to elevate the blood from the heart to the brain, and to overcome the resistances due to the viscosity of the blood in the arteries.

I have investigated the circulation in the brain by recording the aortic pressure, the pressure in the superior vena cava, the intracranial pressure, and the cerebral venous pressure, the cranium being, as in the normal condition, a closed cavity. As a control to elucidate certain points, the pressure of the cerebrospinal fluid has been recorded, and the vessels of the pia mater inspected.

The required observations can be carried out thus : The aortic and vena cava pressures are obtained by passing cannulas down the carotid artery and jugular vein respectively. These cannulas are connected with recording manometers. The intracranial pressure is observed by means of my brain pressure-gauge. The cerebral venous pressure can be recorded by screwing a tube into the torcular Herophili, and connecting this with a manometer. Lastly, the pressure of the cerebrospinal fluid can be measured by trephining the lamina of the atlas, screwing a tube into the trephine hole after the dura has been opened, and connecting this with a manometer.

Hürthle has investigated the circulation in the circle of Willis by recording the pressure in the peripheral end of the internal carotid, and I have also used this method.

I failed to obtain any conclusive evidence of the existence of any vasomotor nerves supplying the brain, on stimulation of the vasomotor centre, the central end of the spinal cord after its division in the upper dorsal region, or on stimulation of the stellate ganglion; that is to say, after the whole sympathetic supply to the carotid and vertebral arteries has been excited. It is true the pressure rises in the peripheral end of the internal carotid on stimulation of the cervical sympathetic, and Hürthle takes this as evidence of vaso-constriction. I have, however, failed to find this evidence confirmed by a diminution in brain-volume or cerebral venous pressure. It is possible that this failure is due to the free connexions of the cerebral veins with the sinuses and venous cistern. It is possible, nay probable, that the veins fill as the arteries contract, and so easily and quickly that neither cerebral volume nor cerebral venous pressure shew any alteration.

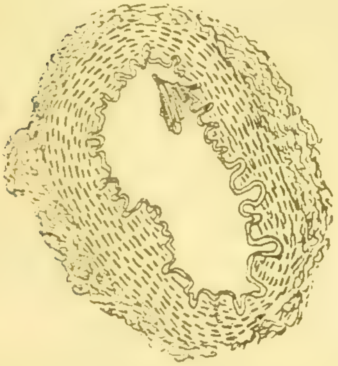

Fig. 1.—Transverse section of contracted middle cerebral artery, from a patient dead of pneumonia, thirty hours after death. Formol, 10 per cent. × 40. (MacWilliam.)

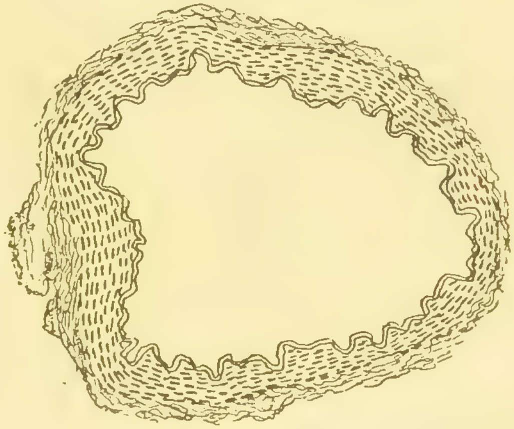

Fig. 2.—Transverse section of middle cerebral artery, from a patient dead of pneumonia, thirty hours after death. Dilated after freezing. Formol, 10 per cent. × 40. (MacWilliam.)

To eliminate such passive compensatory changes Wiggers has studied the artificially perfused brain, and measured the venous outflow from it, using a constant head of pressure to feed the arteries. He says he has obtained an unequivocal diminution of venous outflow on exciting the nerve plexuses in the sheath of the internal carotids.

Biedl and Reiner, measuring the outflow from the cranial branch of the jugular vein in the intact animal, have obtained evidence that the injection of suprarenal extract diminishes the outflow, and the same result has been found on the artificially perfused brain by Ferrier and Brodie and Wiggers. Amyl nitrite, according to Hürthle, Biedl and Reiner, dilates the cerebral vessels. Chloroform is also said to dilate the cerebral vessels (Hürthle, Gärtner and Wagner).

On injection of strophanthin the cerebral vessels were passively dilated by the splanchnic constriction; digitoxin, on the other hand, gave some evidence of active constriction of these vessels, and diminution of brain-volume (Gottlieb and Magnus). Some fairly strong evidence, therefore, has been obtained that the cerebral arteries are constricted by suprarenal extract, etc., like other arteries (with the exception of the pulmonary), and some positive evidence of the existence of vasomotor nerves in the sheaths of the arteries supplying the circle of Willis; but as in almost every experimental condition the cerebral circulation passively follows the changes in the general aortic and vena cava pressures, their power must be comparatively insignificant.

Gulland, W. Hunter, and others have obtained *anatomical* evidence of the existence of nerve-fibres ramifying over the pial vessels. The muscular development and power of post-mortem contraction of such arteries as the basilar and middle cerebral have been portrayed by Professor MacWilliam. The primary function of the muscular wall of the blood-vessels is to maintain tone; the exciting cause of the tonic condition is probably the pressure or quality of the blood. The vasomotor nerves increase or inhibit the excitability of the muscular wall, and thus vary the vascular tone, but the tone soon returns and persists when these nerves are severed. In the splanchnic area the vasomotor influence is greatly developed; in the locomotor organs but slightly. During asphyxia the limbs are passively dilated by the effect of splanchnic constriction, nevertheless the arteries in the limbs are no less muscular than those in the mesentery. In the pulmonary circulation active changes, if any exist, are so masked by passive effects brought about by changes in the vena cava and aortic pressures that the existence of pulmonary vasomotor nerves is denied by Professors Brodie and Dixon. In the cerebral circulation this is also the case.

The circulation in the eye, enclosed as it is in a rigid capsule, is in every respect like that of the brain, and the aqueous humour resembles the cerebrospinal fluid. The aqueous humour is probably secreted by the ciliary processes, just as the cerebrospinal fluid is by the choroidal fringes, and escapes through the spaces of the pectinate ligament into the canal of Schlemm. Sclerosis of this canal, by impeding the circulation, leads to an alteration in the physico-chemical state of the tissues, to increased transudation and tension of the eyeball (glaucoma). The tension, being vascular in origin, depends on the increase of capillary pressure which must follow on the increased swelling of, and transudation from, the tissue of the eye. Room for more fluid in the eye can only be found by narrowing of the veins and conversion of the vascular system into a more rigid system, with increased blood-pressure and rate of blood-flow. By the delicate method of ophthalmoscopic observation, Kahn found that raising the general blood-pressure by suprarenal extract or by dyspnoea causes a passive dilatation of the retinal vessels, which is partly due to obstructed return of the venous blood. When suprarenal extract was injected into the peripheral carotid a short, active constriction preceded the passive dilatation. Stimulation of the cervical sympathetic nerve constricted the retinal vessels in the rabbit, whereas section of the nerve dilated them, but no evidence of such vasomotor action was found by Kahn in the cat and monkey. He failed to find a vasomotor supply for the eye of these animals.

When in any part of the body the arteries contract, the blood-volume in the area diminishes, the veins empty, the whole organ shrinks in size. Since in the closed cranium the brain cannot shrink, the cerebral capillaries must become congested with venous at the expense of arterial blood, if the cerebral arteries became constricted; and this fact may in itself explain the lack of any strong cerebral vasomotor action. There is no need to evoke a cerebral vasomotor mechanism on behalf of the doctrine of cerebral localisation; for, psychologically, it is impossible to maintain that any one portion of the brain can act alone. So long as the mind is in activity, thought skips from memory to memory, while sensations stream from every sense-organ to every sense-centre. The cerebral circulation is regulated by the action of the cardio-inhibitory and vasomotor centres; in what manner will be shewn later.

When the arterial pressure rises, the expansion of cerebral volume can take place to a certain small amount only. For, as soon as all the cerebro-spinal fluid has been driven out of the meningeal spaces, the brain comes everywhere in contact with the rigid wall of the skull. Any further expansion of the arteries and capillaries can only take place by an equivalent compression of the veins, for the semi-fluid brain matter is incompressible. The reservoirs of blood in the veins will therefore be constricted until the cerebral venous pressure again becomes equal to the intracranial pressure; that is, to the pressure of the brain against the veins. Then the whole circulatory system of the brain will have assimilated itself to a scheme of rigid tubes; the velocity of the blood-flow will be increased, whilst the relative distribution of the blood in the arteries, capillaries, and veins will be changed. The doctrine has been enunciated that intracranial pressure varies as the tension of the cerebral arteries, and that, consequently, a high pressure in these arteries must lead to decrease in the amount of blood flowing through the brain. This doctrine is entirely erroneous. The intracranial pressure is found in all physiological conditions to be the same as the cerebral venous pressure. The intracranial pressure is that which remains after the force of the heart has been expended in driving the blood through the capillaries. It is therefore the same as the pressure in the venous sinuses. If the intracranial pressure becomes pathologically greater than the pressure in the venous sinuses, then the cerebral circulation is disordered. By no physiological means can intracranial pressure be maintained at a higher level than cerebral venous pressure; because (*a*) directly the capillaries and veins are obliterated the pressure rises in them to the static pressure, that is, to the mean arterial pressure; (*b*) arterial pressure, transmitted directly through the arteries to the capillaries and veins, must always be greater than arterial pressure minus the elastic tension of the arterial wall transmitted through the brain substance. So long as the high arterial pressure lasts, the expansion of the cerebral vessels reaches its utmost limit, they approach the condition of rigid tubes, and the velocity of the circulation is greatly increased. Thus during the great rise of arterial pressure produced by the injection of absinthe the outflow of blood from the torcular has been seen to increase from two to six times.

Intracranial pressure is purely circulatory in origin, and therefore is variable in quantity. By the term intracranial tension is signified the pressure which the surface of the brain exerts against the walls of the cranium. Cerebral pressure is an expression which has an old pathological connotation, and in dealing with physiological facts it is necessary to avoid its use. Intracranial pressure is the same as cerebral venous pressure, and varies in the same direction, absolutely as vena cava pressure, proportionately as general arterial pressure.

If, in the living animal, the cerebral venous and intracranial pressures be recorded simultaneously with the vena cava and aorta pressures, and if the abdomen be compressed at the same time, the pressures in the brain and the vena cava rise by equal increments when the arterial pressure remains constant; and by greater increments in the brain if the arterial pressure rise also. Whilst the intracranial and cerebral venous pressures vary absolutely as the pressure in the venae cavae, they do not vary absolutely as the general arterial pressure, but only in the same direction, because between arteries and capillaries there lies the unknown resistance in the arterioles due to the viscosity of the blood. In the brain, as in other parts of the body, variations of arterial pressure, read in millimetres of mercury, only produce variations in cerebral venous pressure which can be read in millimetres of water.

The cerebrospinal fluid pressure does not by any means always correspond with the intracranial pressure. For the brain can expand to a far greater degree than the spinal cord, and the brain on expansion, after expressing the small quantity of cerebrospinal fluid from the cranium, comes into contact with the cranial wall. On the other hand, the cerebrospinal fluid not only distends the vertebral ligaments, but leaks away from the subarachnoidal space.

While in normal conditions, with the animal horizontal, the intracranial and cerebral venous pressures generally equal about 100 mm. H_2O, during forced expiratory effort, when the outlets of the venous blood into the thorax are blocked by the high intrathoracic pressure, intracranial or cerebral capillary tension must rise considerably. In the spasms of strychnine poisoning I have seen the intracranial pressure rise to 50 mm. Hg.

On the other hand, in a certain patient, I have observed the intracranial tension to be slightly below zero while he was standing upright. This patient had been trephined for symptoms of insanity. It is thus clear that the functions of the brain can continue when that organ is submitted to very large variations of pressure. In these respects there is nothing which pertains very peculiarly to the cerebral circulation. The circulation within the marrow of the bones must be under exactly the same conditions as that of the brain. The limbs, again, are encased with skin which allows only a certain definite amount of expansion; when this limit is reached any further congestion of blood is prevented, and the tissues of the limb must be submitted to almost arterial pressure. Hence the tension in an inflamed and swollen part. Since the soft parts of the body yield and expand to arterial pressure to a large degree, determination of blood from one part to another can be there brought about by means of the vasomotor mechanism. In the brain, not the blood quantum, but rather the velocity of blood-flow is increased, whenever the arterial pressure is raised by general vaso-constriction.

Since the cerebral circulation is controlled by a comparatively weak (if any) local vasomotor mechanism, and passively follows the changes in the general circulation, it is necessary to determine how the latter maintains the efficiency of the former. This leads us to the discussion of a matter of great importance in cerebral pathology, namely, the effect of the position of the body on the circulation.

In a man, 6 ft. high, the hydrostatic pressure of a column of blood reaching from the vertex to the sole of the feet equals 140 mm. Hg; and from the vertex to the middle of the abdomen about 50 mm. Hg. If in a dog the heart be inhibited, after the skull has been opened, and the animal be placed in the vertical feet-down posture, the brain and heart will empty of blood, while the splanchnic veins and capillaries become distended. In the feet-up posture these conditions will be reversed.

If, on the other hand, the heart be at work, and the animal be placed in the feet-down position, the circulation through the brain ceases whenever the power of the cardiac and respiratory pumps is not sufficient to raise the blood from the splanchnic area, to lift it to the brain, and to overcome the resistance in the cerebral arterioles. In the vertical feet-down position the pressure in the splanchnic and leg areas is increased by the height of the column of blood, which extends from the brain to these vascular areas. This tends to enlarge their capacity; to these parts the flow of blood is aided, while that to the brain is opposed.

Now, in man the column of blood on the venous side is broken into short segments by valves in the veins of the arms, the jugular veins, and the veins of the legs. The longest column of venous blood, unbroken by any valve, is that which extends from the superior longitudinal sinus, by way of the anastomosis with the intravertebral sinuses, to the azygos veins, and thence to the end of the vena cava inferior. Symington and others have thrown doubt on the competency of the jugular valves. The function of the valves is to prevent the over-distension of the lower veins by gravity, and to allow the venous blood to be returned to the heart by the movements of the skeletal muscles. In pathological states leading to incompetency of the valves over-distension is seen to arise and to produce varicose veins.

The capacious abdominal veins are supported under the weight of the column of blood by means of the abdominal wall. The efficiency of this support depends on the tone of the muscular wall and the integrity of the respiratory centre. The veins of the limbs are supported by the muscles on the one side, and the external sheath of the limb—the skin—

on the other side. By such means the distension of the veins and capillaries in the erect posture is largely prevented. The determination of the blood into the lower parts of the body under the influence of gravity is largely prevented by the contraction of the skeletal muscles, by the peristalsis of the viscera, and by the action of the respiratory pump. The respiratory movements aid the venous flow by the alternate compressive action of the abdominal wall and suction action of the thorax. On the arterial side of the circulation the arterioles are held in a state of tone by the vasomotor centre, and limit the output into the veins.

When an animal is suddenly turned from the horizontal to the vertical feet-down position, the peripheral resistance in the great splanchnic area is increased, and the capacity diminished ; thus the circulation through the head is maintained. The blood in all cases takes the pathway of least resistance. The great splanchnic area forms the resistance box of the circulation ; if its channels be constricted the blood takes the pathway through the locomotor organs ; if they be dilated the blood accumulates in the capacious veins of the abdomen. When the splanchnic area is fully dilated the whole of the blood collects within the abdomen, and, if the animal be in the vertical feet-down posture, the influence of gravity may rapidly bring about this result. Splanchnic vasomotor paralysis can, however, be compensated by the action of the respiratory pump. The capacity of the abdominal vessels can be reduced by the compressive action of the respiratory muscles, and the output from the splanchnic arterioles held in check ; while the blood pressed up the vena cava can be aspirated into the right heart by the inspiratory action of the thorax.

If the spinal cord be divided at the level of the first dorsal vertebra, the influence of the bulbar centres on the parts below the section is removed, abdominal and intercostal breathing ceases, the respiration becomes purely diaphragmatic, the tone of the great splanchnic area of the arterioles is lost, and the capacity of the abdominal vessels is greatly increased. The total effect on animal, when lying in the horizontal posture, is a considerable fall of the arterial pressure, coupled with a marked diminution of the respiratory oscillations. If the animal be now placed in the vertical feet-down position the arterial pressure falls more rapidly, and the circulation may come to an end. This is so because the blood passes through the dilated arterioles and collects in the abdominal veins; no mechanism, except the diaphragm, is left for filling the right ventricle, and thus the heart, empty of blood, continues to beat to no purpose. If now the abdominal wall be firmly compressed with the hand, the capacity of the splanchnic area is reduced, the right ventricle filled with blood, and the circulation renewed. The same effect is produced by sinking the animal in a bath. On taking off the hand the heart empties again, the arterial pressure falls, and the circulation ceases. When the animal is once more placed horizontally the influence of gravity is abolished, and the circulation immediately becomes re-established. Experiment shews that the respiratory pump can compensate gravitation when the tone of the splanchnic area is destroyed by section of the splanchnic nerves. The respiratory pump, on the other hand, can be paralysed by the injection of curare. Under these conditions the heart is able to maintain the circulation so long as the capacity of the abdominal vessels is kept under control by the vasomotor nerves.

Whenever, by any means, the thoracic pressure is rendered sufficiently positive to prevent the diastolic filling of the heart the blood stagnates in the abdomen and the circulation ceases. Owing to the influence of gravity this state of affairs is brought about more easily in the vertical feet-down position than in the horizontal posture. Thus, when an animal is in the feet-down position the circulation can be easily stopped by injection of fluid into the pericardium by compressing the thorax, or by forcibly expanding the lungs with air. In the crushes of panic-stricken crowds death takes place from syncope induced by compression of the thorax and cessation of the cerebral circulation. The importance of the respiratory pump as an aid to the circulation is shewn by the symptom of orthopnoea seen in cardiac and pulmonary diseases. The patient does not struggle for air, but struggles by means of his respiratory efforts to maintain a sufficient circulation in the face of cardiac defect, or of increased pulmonary resistance.

On the assumption of the feet-up posture there exists no great vascular field like the splanchnic area to expand under the weight of the column of blood. The face and neck alone become congested, for the brain is confined within the rigid wall of the cranium. In the feet-up posture the arterial pressure in the carotid artery is kept down by a compensatory mechanism. The heart is slowed; the action of the respiratory pump is diminished; the output of the heart is lessened; probably dilatation of the splanchnic arterioles also takes place.

In healthy young men I found that the pressures in the brachial and posterior tibial arteries are the same in the horizontal posture, whereas in the vertical position the one is higher than the other by the height of the column of blood that separates the two points of measurement. The compensatory mechanism acts so that the pressure in the cerebral arteries is kept practically constant, whilst in the leg it varies very greatly on changing from the standing to the inverted posture. The nervous mechanism involved is probably of the simplest kind, for, if the arterial pressure suddenly rise or fall, the bulbar centres are thereby either directly or reflexly excited to decreased or increased activity. A sudden fall of arterial pressure always provokes acceleration of the heart, amplified respiration, and increased vaso-constriction. A sudden rise of pressure, on the other hand, provokes a slow heart, shallow respiration, and vaso-dilatation.

When the compensatory mechanism is abolished by destruction, exhaustion, or inhibition of the bulbar centres, the circulation fails in the vertical feet-down position, the blood passes into the abdomen, and the cerebral circulation ceases. There can be no doubt that the control of this compensatory mechanism is one of the most important and absolutely necessary functions of the group of bulbar centres, a function which must be evolved to its highest point in man.

That the influence of gravity has much to do with the causation of emotional syncope is clear from the success which follows lowering of the patient's head between the knees. Syncope is commonly regarded as being due to a cessation of the action of the heart. It is, however, well known that in cases of syncope the heart does not cease to beat, but continues to pulsate rapidly and very feebly.

The symptoms of syncope are exactly similar to those conditions observed in an animal placed feet downwards with the compensatory mechanism for gravity abolished. By sudden fright in the standing posture the action of the respiratory and vasomotor centres is inhibited, and syncope is induced by the rush of blood to the abdomen and cessation of the cerebral circulation. The patient is restored on being placed in the horizontal or feet-up position. A deep sigh is the first sign of improvement; the right heart is thus filled with blood by the thoracic suction which is accompanied by retraction of the abdomen.

Patients who have lain long in bed, especially when exhausted by disease, lose the power of adapting themselves to changes of posture. Hence the dizziness and danger of syncope which occur when the patient first rises from bed. The condition of shock may be largely, if not entirely, explained by the loss of the compensatory mechanism which maintains the circulation in the face of the influence of gravity. A condition of shock always advances slowly during the course of a prolonged experiment on an animal. The arterial pressure steadily falls; the respiratory oscillations of pressure become less; the heart-beats weaken as the coronary circulation becomes less efficient; the temperature of the animal decreases; the fall of pressure in the feet-down position becomes steadily greater. Such a condition is rapidly intensified if injury be accompanied by severe haemorrhage (*vide* art. "Shock," Vol. III. p. 887).

Among drugs in common use chloroform stands prepotent as a drug which has the power to abolish the compensatory mechanism. Chloroform causes cardiac and vascular dilatation by acting directly on the musculature of the heart and vessels, weakens the respiration, and abolishes the tone of the abdominal and skeletal muscles. Cases are occasionally recorded of men, some of whom lose their memory in the upright posture, and regain it when in recumbency; others can only do mental work when in the horizontal posture; others are suddenly at a loss for memory when attempting to speak in public. These symptoms, and similarly the effects of fear which are so commonly manifested by men before battles, examinations, or public appearances—the vomiting, diarrhoea, and involuntary micturition—may all be associated with temporary inhibition of the compensatory mechanism, and determination of blood to the abdomen.

A useful clinical guide to the condition of the compensatory mechanism in man is afforded by the frequency of the pulse on change of posture. If the heart be markedly accelerated on suddenly passing from the horizontal to the vertical position the mechanism is inefficient. In some cases the change of pulse-rate may be as much as 20 to 40 beats per minute; normally it does not exceed 5 to 10 beats per minute. Immersion in a hot bath at 110° F. for some 10-15 minutes produces such dilatation of the vessels of the skin that the compensatory mechanism fails, and the subject, whose body temperature may be raised to 102-104° F., faints on standing up. After such a bath the pulse may beat 30-40 times more in the vertical than in the horizontal position.

In states of neurasthenia and exhaustion the pressure may be less in the erect posture, and is maintained by a greatly accelerated heart-beat. It is obvious that in such states the maintenance of the horizontal position, and the wearing of an abdominal belt, will aid the circulatory mechanism.

From the above discussion it is now clear that the splanchnic vasomotor mechanism is of overwhelming importance in maintaining the cerebral circulation. We have, in the vasomotor centre, a protective mechanism by which blood can be drawn at need from the abdomen and supplied to the brain. The respiratory centre renders important aid. At the moment that excitation from the outside world demands cerebral response, the splanchnic area is diminished, and more blood is driven through the brain. An anaemia of the brain excites the bulbar centres; these provoke acceleration of the heart, splanchnic constriction, and increased respiration; the cerebral circulation is thereby restored. The vasomotor mechanism of the brain, if it exist, is not powerful; but its blood-supply can be controlled indirectly by the bulbar centres acting on the splanchnic area. These centres are part of the central nervous system; they feel the same needs, and are stimulated by the same centripetal impulses as affect the rest of that system; thus a supply of blood to the brain is maintained which corresponds to its functional activity.

From this section we may conclude that:—

(1) There is some, but not very definite, evidence pointing to the existence of weak cerebral vasomotor nerves.

(2) In almost every experimental condition the cerebral circulation seems passively to follow the changes in the general arterial and venous pressures.

(3) The intracranial pressure, or the pressure of the brain against the cranial wall, is in all physiological conditions the same as the cerebral venous pressure.

(4) The intracranial and cerebral venous pressures vary directly and absolutely with vena cava pressure, but only proportionately with aortic pressure.

(5) The intracranial pressure is purely circulatory in origin, and may vary largely with the changes in circulatory pressure. Normally, in the horizontal posture, it equals about 10 mm. Hg.

(6) The volume of blood in the brain is, in all physiological conditions, but slightly variable. The venous side of the cerebral circulation may, however, be congested at the expense of the arterial side, or the arterial side at the expense of the venous.

(7) In all physiological conditions a rise of arterial pressure accelerates the flow of blood through the brain, and a fall slackens it. A rise of vena cava pressure impedes the cerebral circulation.

(8) The cerebral circulation is controlled by the bulbar centres which act on the general circulatory system. When the brain requires more blood the heart is accelerated, the splanchnic arterioles constricted, and the pressure in the carotid artery raised.

(9) The force of gravity must be regarded as a cardinal factor in dealing with the circulation of the blood.

(10) In the vertical position (head up or down) the effect of gravity is compensated by means of the cardiac and vasomotor mechanisms. The pressure in the carotid artery is thus maintained at about the same level, while that in the legs varies greatly. From the lower part of the body the blood is returned to the heart by means of muscular movements acting on the valved veins, and by the action of the respiratory pump.

(11) When the power of compensation is damaged by loss of muscular tone and paralysis of the vaso-constrictor mechanism, induced by shock or exhausting disease, the influence of gravity becomes of vital importance. In the upright posture the blood then drains into the great abdominal veins, the heart may empty, and the cerebral circulation cease.

(12) The horizontal and feet-up postures at once abolish syncope induced by the feet-down posture, by causing the force of gravity to act in the same sense as the heart, whereby the cerebral circulation is renewed. Firmly bandaging the abdomen has the same effect.

(13) If the heart be weakened by some poison, such as asphyxial blood or chloroform, the sudden dilatation of the right ventricle produced by the assumption of the feet-up position, or by pressure on the abdomen, may throw the heart into paralytic dilatation.

(14) Paralytic dilatation of the heart is relieved by the upright posture, coupled with rhythmic compression of the thorax. The right ventricle is thereby emptied into the abdominal veins.

(15) During changes of posture the heart frequency is normally but little altered (5-10 beats). When the compensatory mechanism is defective, the frequency may be greater, by 30 to 40 beats, in the upright than in the horizontal position. This is a clinical index of defective compensation.

(16) The cerebral circulation is lessened by anything that obstructs the pulmonary circulation. A rise of intrathoracic pressure impedes the flow of blood through the lungs, lowers the arterial pressure, raises the vena cava pressure, and congests the brain with venous blood. Thus syncope can be easily produced by forcible compression of the thorax.

Cerebral Hyperaemia.—On the grounds of experimental research, it has been made evident that no noteworthy increase in the quantity of blood within the cerebral capillaries can take place suddenly. But an increased flow of blood through the brain is produced whenever, by forcible action of the heart or contraction of vascular areas, the general arterial pressure is raised. The relative amount of arterial and venous blood within the brain may then alter—that is to say, the arterial side of the cerebral system may be more, and the venous side less distended than normal. In passive venous congestion of the brain just the opposite condition will hold.

The term cerebral hyperaemia can be used to express that condition in which the flow of arterial blood through the brain is increased per unit of time. From such hyperaemia nothing but good to the organism should arise. By the rise of arterial pressure which takes place in outdoor exercise the brain is swept with a stream of arterialised blood, and exhilaration and mental vigour result. In times of mental labour the same thing happens. The sphygmometer shews that the arterial pressure is raised by mental labour, as during the giving of a public lecture, and throughout the course of an animated argument. This arterial hyperaemia is primarily the sequence and not the cause of functional activity in the brain. Sensory excitement awakes the dormant activities of the cerebral centres, and the circulation is braced up for the struggle. It is necessary to remember that we have no means of analysing the effect of the nerve impulses which stream in upon the centres from the effect of the blood-supply on these centres. During sleep impulses cease to excite and the brain is refreshed by rest; at the same time, the sphygmometer shews that the arterial pressure is lowered, the cerebral veins are congested by the horizontal posture, respiration is far less deep, and the plethysmograph records that the blood is derived from the abdomen and sent in increased volume to the limbs. Thus the brain tissue recovers from fatigue in spite of a diminished cerebral circulation. The expansion of the vessels of the limbs is due probably to two causes: (i.) warmth; (ii.) diminished respiration and absence of muscular movement. Owing to the former the vessels dilate; owing to the latter the blood collects in the veins. As we awaken, the blood forsakes the periphery; by the increased action of the heart, the greater tone of the arterial system, and the important aid which respiration and muscular movements render to the venous circulation, it is driven in a greater volume through the brain. We cannot say that either the wakening or the sleeping state is produced by these circulatory changes. I have observed a patient fall asleep while the arterial pressure remained constant; the fall indicated by the sphygmometer may be as great during a period of rest as during sleep. In sleep, as in chloroform anaesthesia, the brain is congested with venous blood at the expense of arterial blood. It is, however, more probable that this is the result rather than the cause of the functional inactivity of the nervous system. Soon after a short period of hard muscular exercise the sphygmometer shews a fall of arterial pressure. This fall follows the rise of pressure which is found during the period of exercise, and is due probably to the vascular dilatation of the skin and muscles. In spite of this fall of pressure the brain is then often most fitted for mental labour.

Writing of men of genius, Lombroso said that "some, in order to give themselves up to meditation, even put themselves artificially into a state of cerebral semi-congestion. Thus, Schiller plunged his feet into ice. Pitt and Fox prepared their speeches after excessive indulgence in porter. Paisiello composed beneath a mountain of coverlets. Descartes buried his head in a sofa. Bossuet retired into a cold room, with his head enveloped in hot cloths. Cujas worked lying prone on the carpet. It was said of Leibnitz that he 'meditated horizontally,' such being the attitude necessary to enable him to give himself up to the labour of thought. Milton composed with his head leaning over his easy-chair. Thomas and Rossini composed in their beds. Rousseau meditated with his head in the full glare of the sun. Shelley lay on the hearthrug with his head close to the fire." It is possible that some of these men, by means of ice to the feet, or a cold room, increased the flow of arterial blood through the brain, just as many men can think best when exhilarated by rapid exercise, or when walking up and down a room. On the other hand, lying in bed under a mountain of coverlets, or excessive indulgence in porter, lowers the arterial tension. In some cases in which the circulation is feeble and the compensation for gravity defective, active thought may only be possible in the horizontal posture. These oddities of genius cannot be explained thus lightly on purely mechanical grounds; nor must we forget that postural tricks are common enough in ordinary men and in ordinary circumstances.

Arterial hyperaemia is probably always the sequence and not the cause of a primary functional activity in the central nervous system. The arterial pressure is raised by all conditions which produce mental excitement.

In animals arterial hyperaemia of the brain, produced experimentally, gives rise to no symptoms. "Of all regions of cerebral pathology," writes Sir W. Gowers, "that of congestion of the brain is most obscure. We have very little precise knowledge regarding it; and as is often the case, theory has flourished in proportion to the deficiency of fact. It was long thought that the state of the vessels of the brain after death corresponds with their condition during life, and post-mortem distension was accepted as proof that any preceding cerebral symptoms were due to congestion. The fact was unobserved or ignored that a similar condition of the brain is equally common when there are no cerebral symptoms during life, and depends chiefly on the mode of death." It is highly probable that many symptoms have been erroneously ascribed to cerebral hyperaemia which should be placed at the door of cerebral venous congestion, or of anaemia of the brain.

Heightened arterial blood-pressure is probably, in all cases, merely a symptom of that pathological condition by which the other symptoms of cerebral disturbance are produced. It is to be particularly remembered that the vascular condition of the face and head and neck does not necessarily correspond with that of the brain. For example, the flushing, the throbbing, and the headache produced by amyl nitrite are associated with dilatation of the heart and vessels, a fall of arterial pressure, and a diminished cerebral circulation.

In states of exertion, or of mental excitement, the arterial blood is, according to sphygmometer readings, run at high pressure, while the frequency of the heart is notably accelerated. After any severe effort the circulatory mechanism takes time to return to its normal quiet level. The cerebral excitement starts the energetic circulation, but the latter adds fuel to the former, and continues to maintain the brain in activity when the time for effort has ceased and repose should come. In this condition, when sleeplessness and mental irritability torment the exhausted worker, it is well known that diversion of blood to the body by hot baths, hot drinks, and warm bottles, by raising the head high, and by purgation, is a simple and effective form of treatment. Even in these cases it may be the soothing nature of the nerve stimuli, rather than any alteration in the circulation, that induces sleep. Cold, when applied to the head experimentally, produces no effect on the cerebral circulation. The employment of the wet towel by the student who burns the midnight oil must therefore find its explanation in some stimulation to the brain, which is of sensory and not circulatory origin. The cerebral tissue may be actually cooled below the body temperature by the application of an ice-cap. This cooling may produce the required therapeutic effect.

From this section it may be concluded:—(i.) Arterial hyperaemia of the brain does not exist as a pathological state. (ii.) Sphygmometer readings of arterial pressure in man shew that the tension of the cerebral arteries is constantly varied by change of external temperature, exercise, sleep, baths, food, etc. Such variations are purely physiological. (iii.) There is every indication that the quality of the blood which flows through the brain is of far greater importance than the quantity. (iv.) Mental exhaustion probably does not arise during the stage of hyperaemia produced by continued over-effort, but supervenes at that period when the circulatory mechanism begins to fail, and venous congestion accompanied by a deficient supply of oxygen is the fate of the exhausted cerebral centres.

Cerebral Anaemia.—The effects which follow ligature of the common carotid arteries have been known from classical times. The very name of the artery betokens sleep. "Peripatetic mountebanks used," as we are told, "to include a goat among their stage properties, and were said to tie up and relax these arteries in the animal, so that at their pleasure the goat fell down motionless and stupid, or at their bidding leapt up again with great vigour." The first important experiment on this subject was that of Astley Cooper. He occluded both the carotid and vertebral arteries in a rabbit; spasms immediately resulted, and the respiration ceased. In a dog Cooper ligatured the same four arteries. After a preliminary stage of paralysis the animal recovered, and lived to be an excellent house dog. Kussmaul and Tenner tied the left subclavian and innominate arteries. The immediate symptoms were loss of consciousness and voluntary movement. These were followed, in ten to forty-five seconds, by clonic spasms beginning in the muscles of the neck; then occurred dilatation of the pupils, respiratory gasps at longer and longer intervals, and finally cessation of respiration. After complete occlusion of these arteries, for not longer than two to three minutes, the brain, on the ligatures being once more loosened, shewed the power of complete recovery. The sudden re-entry of blood stopped the spasms, and in no case did it cause them. On loosening the ligatures the rabbit often gave a sudden jump forward, then remained for a few moments as if paralysed, and finally recovered completely. Death without spasm occurred in debilitated or over-anaesthetised animals. These spasms are not occasioned by the change of intracranial pressure, for they are produced in no less a degree when the pressure is maintained by an injection of normal saline solution into the carotid artery. The spasms and other symptoms likewise appear when all the cerebral venous outlets are blocked. If one venous outlet be left unobstructed an efficient circulation is maintained, and the animals do not die. The longest period of complete cerebral anaemia after which recovery of function has taken place in animals is about twenty minutes (Stewart, Pike, and Guthrie). Usually the period is much shorter. In one case in which Guthrie transplanted a dog's head on to another dog, stitching together the divided blood-vessels, movements of the eyes, etc., were obtained on re-establishing the circulation after an interruption lasting thirty minutes.

Couty, Markwald, and others have produced anaemia of the brain experimentally by the method of embolism. Either fine powder or melted wax has been used to plug the cerebral arteries. The arrest of the circulation of the whole brain by embolism almost immediately causes cessation of respiration, an enormous rise of arterial pressure, and slowing of the heart-rate. The heart is sometimes completely inhibited. In a few minutes death follows, preceded by acceleration of the heart and fall of pressure. Embolic anaemia, of the cerebral hemispheres alone, causes slowing of the pulse without any rise of arterial pressure. The animal is sleepy and unable to walk, while the limbs remain in abnormal positions. There is no response to stimulation of light or sound. If the mid-brain be cut off from its blood-supply opisthotonos occurs. If the basilar artery be embolised by injections into the vertebral arteries, the greatest pressor effects occur, and after some spasms from failure of respiration death quickly results.

The effects which follow ligation of the four cerebral arteries vary greatly in different animals; to a less extent in different individuals. In dogs a free anastomosis is set up by way of a branch from either superior intercostal artery. This passes in with the brachial plexus and joins the anterior spinal artery. These arteries, after the ligature of the vertebrals and carotids has been carried out, become almost as large as the vertebrals, and maintain an efficient circulation. Thus, in dogs death never ensues. On the other hand, almost all rabbits and 40 per cent of cats die, either at once or in a few hours, after the quadruple ligature has been simultaneously applied. Monkeys likewise die in the course of a few hours. Most monkeys survive the simultaneous ligation of both carotids. They succumb to ligation of the two carotids and one vertebral. If the ligatures be applied in monkeys, not simultaneously but successively, at intervals of a few days, no untoward symptoms arise. When a record of cerebral venous pressure is taken and the four cerebral arteries are ligatured in the dog, the venous pressure is seen at first to fall distinctly, and then gradually to recover itself. The pressure in the circle of Willis (peripheral end of carotid) falls likewise. It rises subsequently as the anastomotic pathways become open, and in some cases even to a higher level than normal. This is owing to the pressor effect produced by bulbar anaemia.

The symptoms of acute cerebral anaemia are the same whether produced by ligature or by embolism of the arteries. If the cerebrum be rendered bloodless, loss of consciousness and general motor paralysis result. Epileptiform spasms may occur in strong subjects. The pupils dilate and nystagmus often occurs. Rigidity and reflex hyper-excitability are marked symptoms.

In monkeys, if two carotids and one vertebral be ligatured, the pupil is dilated on the side opposite to the obstructed vertebral. When the hind-brain is suddenly rendered anaemic, there ensue general spasm of a tonic character associated with rigidity, vasomotor spasm producing a high blood-pressure, respiratory spasm, and a slow heart caused by spasm of the vagus centre. The state of spasm is followed by paralysis, as is shewn by the failure of respiration, fall of arterial pressure, and acceleration of the heart. In exhausted or deeply anaesthetised subjects spasm does not appear, and the stage of paralysis is rapidly reached. Generally the primary cause of death is failure of respiration. If this be compensated by artificial respiration the heart continues to beat, and then the vasomotor paralysis which ensues may be a secondary cause of death. This can be compensated by compression of the abdomen and by placing the animal in the vertical feet-up position. In rare cases vasomotor paralysis may be the primary cause of death.

Cheyne-Stokes respiration and Traube-Hering blood-pressure curves are commonly obtained in conditions of anaemia of the spinal bulb. The injection of morphine, a solution of magnesium sulphate, and the administration of chloroform frequently produce these curious phenomena in dogs, especially after ligation of the cerebral arteries. In hibernating animals they are constantly present; group respiration may frequently be seen in sleeping children. The phenomena may occur when the arterial pressure is high. Such is the case in dogs poisoned with morphine. In some cases Traube curves may be peripheral and not central in origin. This periodicity of action is a sign of lowered nutrition in the bulbar centres. Forced breathing, especially through a tube, causes Cheyne-Stokes respiration, by interfering with the circulation.

Cheyne-Stokes respiration may be abolished by inhalation either of oxygen, or 5 per cent of carbon dioxide. The tension of carbon dioxide in the blood is the natural regulator of breathing. Want of oxygen causes increased breathing, and this washes out the carbon dioxide and then the respiratory centre fails to discharge until the carbon dioxide tension rises again. Breathing oxygen prevents the want of oxygen, whereas inhalation of 5 per cent carbon dioxide keeps the centre excited continually (Pembrey).

Obliteration of the cerebral arteries in man produces the same symptoms as in animals. If both common carotids be suddenly compressed the pupils widen, respiration deepens, dizziness and loss of consciousness follow. Epileptiform spasms frequently occur. I have produced clonic spasms in myself by compression of one carotid artery, thus confirming the experience of Schiff. The first effect of the compression was a tingling sensation in the eye on the same side; then followed a march of a sensation of pins and needles down the opposite side of the body: this began in the fingers, spread up the arm, then down the leg. Finally, clonic spasms of the flexors of the forearm occurred, accompanied by a feeling of vertigo and alarm. Consciousness of the clonus was aroused by the sensation of the hand striking the arm on the chair. In dogs, with the four cerebral arteries tied, I find the excitability of the cortex markedly increased.

Cases of epileptic fit have been recorded which have been preceded by sudden failure of the pulse, and Dr. A. E. Russell has put forward the view that the fundamental factor underlying both the ordinary faint and the epileptic fit is cerebral anaemia.

The effects of compression of one carotid vary in different men; no doubt in relation to the freedom of the anastomosis in the circle of Willis, and to the excitability of the cerebral cortex. The spasms, when produced, are in every respect the same as Jacksonian epilepsy, or the epileptic fits produced by tetanisation of the sensorimotor area. On occlusion of one carotid artery in the monkey compensation by the circle of Willis is not immediately complete. Horsley and Spencer found that, on ligaturing one carotid, the pia mater pales on the side of the ligature, and the cortex becomes far less excitable. The first effect of such anaemia is to raise the excitability of the cortex (Orchansky). In an hour the circulation is fully restored. From the results of physiological experiment we can be assured that ligature of both carotids, if done slowly, and at due intervals of time, can be carried out without fatal results. If the artery were so gradually occluded by a screw-clamp as to allow time for expansion of anastomotic pathways, no ill effects, such as hemiplegia, should ever result. There is a pathological case on record of gradual obliteration of all four cerebral arteries; efficient anastomosis was established in this case, and no cerebral symptoms were evoked during life.

Sudden and simultaneous ligature of both carotids must be regarded as a highly dangerous operation. Sudden occlusion even of one carotid has in some cases been followed by hemiplegia.

In certain cases, by the simultaneous ligation of the four arteries in the dog, or of three arteries in the monkey, a condition of idiocy has been established. To produce this is not easy, as the cerebral circulation must be reduced to such a point that the animal hangs between life and death. Most dogs recover from the ligation of the two carotids and two vertebrals, after but slight and temporary signs of paralysis. In some cases one superior intercostal artery has been tied also, and yet the animals have completely recovered. Most monkeys, on the other hand, die in the course of twenty hours from the ligation of two carotids and one vertebral. In one case the animal lived for five days in a condition of complete idiocy, paralysis, and rigidity. When in another monkey one carotid and one vertebral were tied, and the other carotid ligatured after an interval of three days, no marked symptoms ensued. Thus a very few days are sufficient to establish the expansion of anastomotic pathways.

A dog rendered idiotic by the method of ligation exactly resembles the dogs from which Goltz removed the cerebrum. He sleeps and wanders round the room alternately; there is a continual tendency to circus movements; the tactile sense is greatly paralysed; movement is defective; his legs slip outwards and give way under him. He stumbles blindly into every object, but can smell and hear. An enraged cat, tobacco smoke, lighted matches, pass equally unnoticed before his face. He persistently tries to get through impossible objects, and finally falls asleep in front of them, his body and limbs being placed in unnatural positions. He eats reflexly when food is placed in his mouth. The optic discs present congested veins and small anaemic arteries.[1] In four or five days' time all these symptoms disappear, and the animal recovers completely.

On histological examination of the brains of these dogs, Dr. Mott found that within twenty-four hours of the operation, and during the stage of idiocy, the nuclei of the cortical cells became swollen, and the cytoplasm of the cell stained diffusely by Nissl's methylene blue method. The stichochrome granules disappeared from the cells, to reappear once more when the animal recovered. In the monkey the pathological changes have gone further, and the symptoms have been more profound. One monkey became completely rigid in the extended position, anaesthetic, and paralysed. It remained in the same condition for five days and was then killed. The brain was softened. Microscopically, many of the cortical cells were found greatly swollen and devoid of stichochrome granules; the perivascular spaces were distended with exudation, and contained numerous phagocytes. It is noteworthy that by Golgi's method the cells and their dendrites appeared unaltered. It is extraordinary that this monkey, although dead to all other forms of stimulation, when a cat entered the room twice made a hoarse guttural sound of alarm and attempts at flight movements.

Cerebral Anaemia of Slow Onset.—When general anaemia of the brain comes on gradually, symptoms of exalted excitability do not appear. In man, headache, hyperaesthesia, tinnitus, and vertigo are common symptoms, associated with mental dulness and drowsiness. In some cases great mental irritability and insomnia may appear. The patient loses the power of decisive action, may suffer from melancholia and be filled with unreasoning fears. Dimness of sight or double vision and deafness may be complained of. Muscular power is weakened, memory is deficient, power of application and energy of action are lost. Continued anaemia of the brain may result from heart failure, from loss of vasomotor tone, from exhausting diarrhoea, or from defects in the quality of the blood.

---

[1] In these conditions the author finds the cortex to be *hyper-excitable*. The sensory synapses and not the motor pathways are paralysed.

Whatsoever tends to obstruct the general venous circulation causes at the same time cerebral venous congestion; and this, by obstructing the flow of arterial blood, leads to anaemia of the brain. Likewise whatsoever tends to lower arterial blood-pressure lessens the flow in the cerebral arteries.

In infants collapsed from choleraic diarrhoea the depression of the fontanelle shews that the blood-pressure is insufficient to overcome the resistance in the cerebral arteries. In these cases pallor of the face is well marked. Somnolence may pass into coma, then the pupils dilate, and become insensible to light; and death results from the gradual paralysis of the bulbar centres.

Chronic anaemia of the brain may result in permanent damage. In children the development of the brain may be arrested, and imbecility be the consequence. Long-continued general loss of vascular tone or degeneration of cerebral arteries, and consequent failure of efficient cerebral circulation, may be a fruitful source of cerebral degeneration and of lunacy. Valvular disease of the heart may lead to the same end. Persistent mechanical congestion of the veins, arising from pulmonary disease and from cough, must reduce the cerebral circulation.

The fact that the brain can continue its functions with a greatly reduced blood-supply is shewn, not only by the experiments on the effect of ligation of the cerebral arteries, but by the lack of any marked cerebral symptoms in advanced cases of pernicious anaemia. I have demonstrated by determination of the gases in cerebral arterial and venous blood that the brain is not a seat of active combustion. The combustion of the muscles, whether at rest or in the state of epileptic spasm, is vastly greater than that of the brain.

Cerebral Venous Congestion.—The symptoms of passive congestion are most marked when the congestion is sudden in onset. Mechanical congestion is caused by the head-down position, by anything which impedes the flow of blood in the jugular veins, such as tight clothing round the neck, and by any cause which hinders the entry of venous blood into the thorax or heart.

Experimentally it has been found that a marked rise of cerebral venous pressure occurs when a band is tightened round the neck of a dog so as to exclude the trachea. Even flexion of the neck or contraction of the neck muscles will raise the cerebral venous pressure.

The garrotter produces loss of consciousness in his victim by suddenly occluding both the carotid arteries and the jugular veins. Cough or holding of the breath during great muscular efforts are means by which cerebral congestion is produced in ordinary life. Sir W. Gowers quotes the case of a man who exhibited clonic spasms at the climax of long coughing fits. Congestive headache is often produced by coughing. Forced breathing has a like effect. Since the cerebral capillary pressure is increased by any rise of intrathoracic pressure, such as occurs when the breath is held during an effort, we might expect to find arterial degeneration in men who have followed laborious trades. Hard toil, however, does not appear to be a sufficient cause in itself; the wall of the vessel must be predisposed, by some toxic agency, to degenerate.

It is easy to understand why cerebral haemorrhage should most frequently occur when the veins are congested by straining at stool, muscular effort, violent cough, stooping to button boots, or during copulation; or again during sleep when the body is in the horizontal posture. It must be remembered that cerebral capillary pressure varies absolutely with general venous, but only proportionately with arterial pressure.

Persistent mechanical congestion of the brain causes somnolence, mental inactivity, vertigo, and other symptoms which are characteristic of cerebral anaemia. Anaemia of the brain produces the same effect, whether the flow of arterial blood be reduced by a fall of arterial pressure or by a rise of venous pressure. In both cases the brain is congested with venous at the expense of arterial blood.

Cerebral venous congestion is produced during epileptic convulsions. In the status epilepticus this congestion must be greatly prolonged, and perhaps leads to those signs of meningitis which are sometimes found after death. It is important to remember that, owing to the impeded circulation and back pressure, the heart becomes engorged in diastole during the fits. The lungs become congested for the same reason. Diastolic engorgement leads to a deficient coronary circulation, for the blood circulates in the coronary arteries in inverse ratio to the pressure within the cavities of the heart; hence, perhaps, the fatty change in the heart which, according to Dr. Mott, is generally found in these cases (*vide* Vol. I. p. 577).

Given an explosive unstable condition of the nerve-cells, it is quite conceivable that epileptic fits may be sometimes discharged by changes in the circulation.

In those young children in whom convulsions are excited by paroxysms of coughing, or exhausting diarrhoea, venous congestion of and want of oxygen in the brain are probably the immediate cause of the nerve storm.

To sum up then:—

1. *The symptoms of acute cerebral anaemia may be produced in man by*:—(i.) Embolism or thrombosis of cerebral arteries. (ii.) Meningeal or cerebral haemorrhage; in these cases the anaemia of the brain may be local or general. (iii.) Failure of the heart's action. (iv.) Obstruction of the respiration, the symptoms of asphyxia being those of acute cerebral anaemia. (v.) The introduction into the system of some substance by which the blood is rendered useless for tissue respiration. (vi.) Great loss of blood. (vii.) Determination of blood to the abdomen

by sudden vasomotor paralysis. (viii.) Complete obstruction of the cerebral venous outflow. (ix.) Compression of the cerebral arteries.

We have no evidence that cerebral anaemia can be ascribed to spasm of the cerebral arteries.

2. *The usual symptoms of acute cerebral anaemia* are in sequence :—(*a*) Loss of consciousness; (*b*) epileptic spasm; (*c*) dilated pupils and nystagmus; (*d*) respiratory spasm; (*e*) slow heart; (*f*) rise of arterial pressure; (*g*) fall of arterial pressure, acceleration of the heart, cessation of respiration. The cortical and bulbar centres are first excited and then paralysed. The fatal symptoms arise when the bulbar circulation ceases.

3. If an animal be in a state of collapse or deep anaesthesia, or if the anaemia be slow in onset, the excitatory symptoms fail to appear.

4. In death from acute cerebral anaemia respiratory paralysis usually precedes vasomotor paralysis.

5. Cheyne-Stokes respiration and Traube-Hering curves are common in states of partial anaemia of the bulbar centres.

6. Ligation of both carotids and both vertebrals does not entirely cut off the brain from blood. The anastomoses with the anterior spinal artery still remain open.

7. A condition of idiocy, accompanied by motor paralysis and anaesthesia, can be induced by ligation of the cerebral arteries. In this condition the cortical cells lose their stichochrome granules, and stain with methylene blue diffusely. The nuclei become swollen. If the cerebral circulation, by the opening of anastomotic pathways, become once more efficient, the condition of idiocy passes off as the cells regain their normal standard.

8. The cerebral arteries can be tied with safety if sufficient time be allowed to elapse between the successive applications of the ligatures. In ligaturing the carotid arteries in man the vessels should be obliterated slowly by means of a screw-clamp, so as to allow time for the anastomotic pathways to be opened up.

9. Chronic cerebral anaemia may be caused by (*a*) loss of vasomotor tone and a feeble heart; (*b*) venous congestion arising from valvular disease of the heart; (*c*) impeded pulmonary circulation; (*d*) pressure on the cerebral veins.

10. A moderate degree of anaemia augments the excitability of the cortex. Severe anaemia reduces the cortical excitability, while it very greatly increases that of the bulbar centres. Total anaemia of the cerebrum produces almost instantaneous loss of consciousness, and abolition of cortical excitability.

The Experimental Effects of Compression of the Brain, or Expression of the Cerebral Blood.—Boerhaave tells of a man at Paris who, at times, would beg money in a piece of his own skull, which had been struck off by an accident, his brain being in part covered only with dura mater. "Upon gently pressing the dura mater he suddenly perceived, as it were, a thousand sparks before his eyes, and upon pressing a little more forcibly his eyes lost all their sight; by pressing the hand still stronger on the dura mater he fell down in a deep sleep, which was attended with all the symptoms of a slight apoplexy merely by this pressure with the hand, which was no sooner removed but he as gradually recovered from the symptoms as they were brought on, the apoplectic symptoms first vanishing, then the lethargy, and lastly the blindness; all his senses recovering their former perfection." La Peyronie has left records of an equally interesting patient. By injecting the sac of an abscess over the region of the corpus callosum the man could be thrown into stupor or awakened at will. By pressing on the sac of a meningocele children can be put to sleep and the respiration slowed.

Every experimenter on the subject of compression of the brain has produced in animals the same series of symptoms:—(i.) Clonic spasms and circus movements when a high pressure is suddenly applied. (ii.) Constriction of the pupils, followed by dilatation: the compressed side is affected first; nystagmus may occur. (iii.) Stupor and coma. (iv.) Slow heart and slow deep snoring respiration, followed, in the later stage, by respiratory gasps and acceleration of the heart. (v.) Vomiting, emptying of the bladder and rectum.

Compression can be experimentally produced by:—(*a*) The injection of a simple fluid, such as serum or saline, into the subdural space through a tube which has been screwed into the skull. (*b*) By the production of haemorrhage in the cranium: the carotid artery can be connected with a tube which has been screwed into the skull, and the blood can then be permitted to flow at arterial pressure into the cranial cavity. (*c*) By the local introduction of a foreign body of given limited volume: to effect this, an india-rubber bag can be inserted into the subdural space, and by means of a graduated syringe this bag can be distended to a definite volume.

An important distinction must be drawn between compression by a foreign body of limited volume and compression by the continuous injection of fluid at a constant pressure. In the latter case, if the fluid spread with ease to all parts of the cranial cavity, the pressure throughout this cavity will rise to that of the injection, and all the veins and capillaries become compressed; finally, when the injection-pressure is made equal to the arterial pressure they will be obliterated. Thus, when the pressure of the injection-fluid is raised gradually, fatal symptoms do not originate until the injection- and consequently the intracranial pressure reach almost to the height of the arterial pressure. If, on the other hand, the injection-fluid should not spread easily to all parts of the cranio-vertebral cavity the distribution of pressure will be various. Fluid can leak away from the cranial cavity with great ease. If there be considerable resistance to the spread of the injected fluid, and this fluid leak from the cranial cavity, it is clear that the pressure will be greater at the seat of injection than at more distant parts. If the fluid do not spread at all, but form a local collection at the seat of injection, the condition then becomes one of local compression; that is to say, the blood will be driven from the part of the brain which is compressed until the local cerebral vascular pressure rises to that of the injected fluid.

When compression is applied locally, the brain does not transmit pressure equally in all directions. This conclusion has been reached by measurements of intracranial pressure in the cerebral, cerebellar, and vertebral chambers respectively. The brain can by no means be regarded as a bag of fluid enclosed in a rigid box; it is rather a viscous, inert mass of the consistency not of a bag of water but of a lump of putty. The brain substance itself is indeed incompressible, but the blood-vessels form an expressible sponge-work, lying chiefly on the outside of the brain mass. Each cerebral hemisphere lies in a separate chamber, partially protected from any increase of pressure in the opposite hemisphere by the strong falciform ligament. The cerebellum and spinal bulb lie in a chamber protected from cerebral pressure by the tentorium cerebelli; the narrow isthmus tentorii cerebelli is accurately filled by the peduncles of the great brain. The cerebellar chamber is connected with the vertebral canal by the narrow foramen magnum. The orifice of this foramen is blocked by the cerebellum and the spinal bulb. When fluid is rapidly injected under high pressure into the subdural space in the parietal region, the great brain is driven down and blocks the isthmus tentorii cerebelli. At the same time the cerebellum and bulb descend and completely block the foramen magnum.

When fluid is injected at low pressures the result is different, for the fluid, following the course of the cerebral vessels, can escape into the vertebral canal at each cerebral pulsation. It is necessary to bear in mind that, in all cases of simple injections, the fluid rapidly escapes from the cranio-vertebral cavity by way of the veins. Thus, to maintain pressure within the cavity, the injection must be continuous. The cause of the major symptoms of apoplexy is the arrest of blood-flow in the bulbar centres. To produce the anaemia of these centres any pressure, above the capillary pressure, applied locally to the centres is sufficient. The resistance in the bulbar capillaries need only be increased until that point is reached when the blood will flow through other pathways in the remaining parts of the brain offering less resistance. Now the bulb is so situated that the pressure of fluid injected into the meningeal space cannot be locally applied to the bulbar centres. For example, if fluid be injected through the occipito-atlantal membrane, it will not only spread to all parts of the vertebral canal, but cause the base of the brain to float up. Thus the pressure in the cranio-vertebral cavity rises everywhere to much the same point, and the injection-pressure must be raised almost to the arterial pressure before the major symptoms of apoplexy arise. Local compression can, however, be applied directly to the bulbar centres by the introduction of a foreign body into the fourth ventricle; it can be applied indirectly through the descent of the brain mass when a local pressure of sufficient height is applied to the cerebrum or cerebellum. By translation of the brain mass the bulb can be pressed against the bony margin of the foramen magnum, and the capillaries of the bulbar centres obliterated.

In the case of a haemorrhage into the cranial cavity blood can form a local collection in the brain substance, in the ventricles of the brain, between the dura and the bony wall of the skull, or in the subdural space. In some cases, on the other hand, the blood can spread like an injection-fluid to all parts of the cranio-vertebral cavity. In this last case the amount of haemorrhage, and consequent expression of blood from the brain, can reach very large limits before the bulbar centres are rendered completely anaemic. As blood clots within the cranium, and forms a foreign body, it cannot leak away through the veins in the same way as simple injection-fluid, such as normal saline or serum. If the haemorrhage and consequent clot be large, no remission of the symptoms occurs after the cessation of the haemorrhage; but the remission can be attained immediately by freely opening the skull, and clearing out the clot. It is very important to note that the mere opening of a small trephine hole is not sufficient to relieve compression; the clot still remains within, and the brain matter presses up against the trephine hole, and closes it up as a valve. To remove the compression entirely, the clot must either be cleared out, or the hole made large enough to allow a compensatory expansion of the volume of the brain.

It cannot be too forcibly urged that it is not the rise of intracranial pressure but the cerebral anaemia which produces the symptoms of apoplexy. The blood-clot as a foreign body occupies a certain volume of the cranial cavity, and the cerebral blood-volume is decreased to a corresponding amount. In the case of the insertion of a foreign body the pressure is raised to arterial pressure in that part of the brain where the capillaries and veins are entirely obliterated; in the neighbouring part of the brain, where the vessels are compressed but not obliterated, the pressure will be less; and in the most distant parts, which lie in some other chamber of the cranio-vertebral cavity, the pressure may remain normal. Since the major symptoms of apoplexy will not arise until the bulbar capillaries are obliterated, it follows that fatal results will ensue from a small haemorrhage into the cerebellar chamber which in the cerebral chamber would produce but slight symptoms. A very small local haemorrhage in the neighbourhood of the bulbar centres will suffice to obliterate the bulbar capillaries and produce death. To produce death by bulbar anaemia, in the dog, a foreign body must be introduced within the cerebral chamber = 4 to 6 c.c. in volume; in the cerebellar chamber = 1 to 2 c.c.; and in the fourth ventricle = 0·5 to 1 c.c.

Since at any pressure above cerebral venous pressure the cerebrospinal fluid rapidly leaks away from the cranial cavity, this fluid can be neglected in dealing with the pathology of compression. It is erroneous to teach that increased cerebral pressure is transmitted to all parts of the cranio-vertebral cavity by the cerebrospinal fluid. It is equally erroneous to teach that a foreign body within the cranium exerts pressure. Intracranial pressure is always circulatory in origin;

the capillaries and veins are compressed, and the passage of blood blocked by the presence of the foreign body. The pressure in the compressed area is raised to arterial pressure so soon as the blood can no longer find an exit through the arterioles. When a local area of capillaries is thus blocked by a foreign body the blood takes the pathway of least resistance through the other vascular areas of the brain. In and around the compressed area, if it be extensive, there lie a large number of arterioles in which the mean arterial pressure is attained, for the outlet from these vessels is blocked. The arterial pressure transmitted from this area through the brain substance will tend to produce compression of other capillary and venous areas. At the same time, exudation of fluid at arterial pressure may take place from the blocked vessels and produce oedema of the brain, which in its turn will tend to compress neighbouring capillary areas. Compensation can be brought about by the transference of tissue-fluid from the brain into the blood and by phagocytes removing tissue substance.

When a blood-clot is removed by the surgeon inflammatory oedema of the brain may arise secondarily. The increase of vascular tension in the inflamed area of dilated arterioles will tend to compress neighbouring cerebral areas in which the capillary tension is less. This inflammatory oedema is the origin of hernia cerebri; the hernia is nature's method of relieving the cerebral compression, and indicates the line which surgical treatment should follow.

The growth of a tumour within the cranium may produce compression of the cerebral capillaries. Owing to their greater width the pressure in the capillaries of a glioma is higher than the cerebral capillary pressure. The cranial contents cannot be increased, and if the quantitative ratio of cell-tissue to blood-vessel be altered it must be at the expense of the blood-volume. Room for the new growth can be found only by compensatory expression of blood, or by atrophy. If the tumour grow slowly, brain atrophy may take place as fast as the new growth increases; thus intracranial tension may not be raised. Tumour can produce acute attacks of compression by exciting inflammation with haemorrhages, oedema of the brain substance, thrombosis of the blood-vessels, and thickening of the meninges. If inflammatory products collect in the cranial cavity the blood-volume must be proportionally diminished or there must occur a corresponding shrinkage of brain substance. Whenever an inflamed area becomes hyperaemic a corresponding area must become anaemic or the tissue substance in the inflamed area must shrink. There is some evidence to shew that this shrinkage takes place. In sections of the bone-marrow of animals infected with the pneumococcus the blood-vessels appear much congested and swollen, while the amount of marrow substance appears correspondingly diminished. A transference of water and actual tissue substance from the tissues into blood-vessels is probably a usual phenomenon of inflammation.

A local collection of fluid in the ventricles, at a pressure higher than cerebral capillary pressure, must inevitably lead to cerebral anaemia and atrophy. Symptoms of compression rapidly appear in the adult, in the child, atrophy of brain substance and expansion of the cranial cavity. A large number of cases of hydrocephalus have now been recorded which shew that the cause of the mischief is the blocking of the aqueduct or the veins of Galen by inflammatory exudations or tumours. The primary cause is altered nutrition followed by increased transudation of cerebrospinal fluid. It is not the increased capillary pressure, but the deficient blood-flow leading to chemical and osmotic changes, which causes the transudation. Ventricular hydrocephalus should be relieved by draining the cavity into the general meningeal space. Dr. G. A. Sutherland and Sir Watson Cheyne have demonstrated the truth of this conclusion: in a case of acute hydrocephalus Sir W. Cheyne inserted a catgut drain leading from the lateral ventricle to the subdural space, and closed the cranial opening; under this treatment the head rapidly diminished in size and the symptoms disappeared.

From the final section of this article the following conclusions may be drawn: —(i.) The brain does not transmit pressure equally in all directions. (ii.) In regard to compression by foreign bodies there is a large amount of pressure discontinuity between the cerebral and cerebellar chambers. There is complete pressure discontinuity between the cranial and vertebral cavities. (iii.) The cerebrospinal fluid cannot transmit a local rise of pressure to other parts of the brain. (iv.) Any pathological increase of cerebral pressure is circulatory in origin: a foreign body obliterates veins and capillaries, and thus at the seat of obliteration the cerebral pressure is raised from capillary to arterial pressure. (v.) It is not mechanical pressure but the cessation of blood-flow which produces the symptoms of compression. (vi.) The major symptoms of compression are produced by anaemia of the spinal bulb, and are exactly comparable to the symptoms of acute cerebral anaemia otherwise produced. (vii.) A far smaller foreign body kills in the bulbar region, and in the cerebellar chamber, than in the cerebral chamber. (viii.) A trephine hole does not necessarily relieve compression. The foreign body must be removed, or the opening be large enough to allow an equivalent expansion of the cranial contents. (ix.) A secondary increase of compression can be brought about by congestive or inflammatory oedema. Compensation for a foreign body is established by transference of tissue fluid or substance into the blood-stream and their removal from the cranial cavity, that is, by a corresponding atrophy of the cerebral substance.

LEONARD HILL.

REFERENCES

Literature: Critical and historical analyses of the experimental researches on the cerebral circulation will be found in von BERGMANN's Monograph on Brain Surgery, *Deutsche Chirurgie*, 1880, xxx. 274; in RICHET's *Dictionnaire de physiologie*, Paris, 1897, tome ii. 745; and in *The Physiology and Pathology of the Cerebral Circulation*, Leonard Hill, London, 1896.—KEY and RETZIUS's Monograph (*Studien in der Anat. des Nerven-Systems*, Stockholm, 1875) contains a complete analysis of researches on the cerebrospinal fluid and the meningeal spaces from the time of Galen downwards.—

Beevor, C. E. "On the Distribution of the different Arteries supplying the Human Brain," *Phil. Trans.*, 1908, B. cc. 1. **Cerebrospinal Fluid**: 1. Adamkiewicz. *Sitz. d. k. Akad. d. Wiss.*, Wien, 1883, lxxxviii. Abt. iii. 11-98, 311-355.—2. Bochefontaine. *Compt. rend. Soc. biol.*, Paris, 1880, 65; *Arch. de physiol.*, Paris, 1879, 791.—3. Falkenheim und Naunyn. *Arch. f. exper. Path. u. Pharm.*, 1887, xxii. 261.—4. Key und Retzius. *Stud. in der Anat. des Nerven-Systems*, Stockholm, 1875.—5. Hill, Leonard. *The Phys. and Path. of the Cerebral Circulation*, London, 1896.—6. Reiner und Schnitzler. *Centralbl. f. Physiol.*, Leipzig u. Wien, 1894, viii. 684.—7. Schwalbe. *Arch. f. mikroscop. Anat.*, 1870, vi. 1, 261. **Cerebral Circulation**: 8. Althan. *Der Kreislauf in der Schädel-Rückgratshöhle*, Dorpat, 1871.—9. Bayliss and L. Hill. *Journ. Physiol.*, London, 1895, xviii. 334.—10. von Bergmann. *Deutsche Chirurgie*, 1880, xxx. 274.—11. Biedl und Reiner. *Arch. f. d. Ges. d. Physiol.*, Bonn, 1900, lxxix. 158.—12. Browning. *Circulation in the Central Nervous System*, Philadelphia, 1897.—13. Cappie. *The Intracranial Circulation*, Edinburgh, 1890.—14. Cavazzani, E. *Arch. ital. de biol.*, Turin, 1891, xvi. 23; 1893, xix. 214; *Centralbl. f. Physiol.*, Leipzig u. Wien, 1892, vi. 393, 533.—15. Cramer. *Exper. Untersuch. ü. d. Blutdruck im Gehirn*, Dorpat, 1873.—16. Dean. *Journ. Path. and Bacteriol.*, London, 1892, i. 26.—17. Elder. *Brit. Med. Journ.*, 1897, ii. 1414.—18. Falkenheim und Naunyn. *Arch. f. exper. Path. u. Pharm.*, Leipzig, 1887, xxii. 261.—18*a*. Ferrier and Brodie. *Brit. Med. Journ.*, 1902, ii. 1340.—19. François-Franck. *Journ. de l'anat. et physiol.*, Paris, 1877, 267.—20. Fredericq. *Trav. du lab.*, 1886, i. 9.—21. Gärtner und Wagner. *Wien. med. Wchnschr.*, 1887, xxxvii. 602, 639.—22. Geigel. *Virchows Arch.*, Berlin, 1890, cxix. 93; cxxi. 432; 1891, cxxiii. 27; cxxv. 92.—23. Girard, J., et Pettit. *Compt. rend. Soc. biol.*, Paris, 1902, liv. 698.—24. Gottlieb und Magnus. *Arch. f. exper. Path. u. Pharmak.*, 1902, 48, 262.—25. Grashey. "Exper. Beiträge zur Lehre v. d. Blut-Circulation in der Schädel-Rückgratshöhle," *Festschr. für L. A. Buchner*, München, 1892.—26. Hill, Leonard. *Proc. Roy. Soc.*, 1894, lv. 52; *The Phys. and Path. of the Cerebral Circulation*, London, 1896.—27. Hill and Macleod. "Supposed Existence of Cerebral Vasomotor Nerves," *Journ. Physiol.*, London, 1900-1, xxvi. 394.—28. Hunter, W. *Ibid.*, 1900-1, xxvi. 465.—29. Hürthle. *Arch. f. d. Ges. d. Physiol.*, Bonn, 1889, xliv. 561.—30. Jensen. *Ibid.*, 1904, ciii. 171.—31. Kahn. *Centralbl. f. Physiol.*, Leipz. u. Wien, 1904, xviii. 153.—32. Knoll. *Sitz. d. kais. Akad. d. Wiss.* (Math. nat. Cl.), Wien, 1886, xciii. Abt. iii. 217.—33. Levy. *Proc. Roy. Soc. Lond.*, 1894, lv. 275.—34. Lewy. *Virchows Arch.*, 1890, cxxii. 146.—35. MacWilliam. *Brit. Med. Journ.*, 1908, ii. 1477.—36. Mosso. *Über den Kreislauf des Blutes im menschlichen Gehirn*, Leipzig, 1881.—37. Naunyn und Schreiber. *Arch. f. exper. Path. u. Pharmak.*, Leipz., 1881, xiv. 1.—38. Pick. *Ibid.*, 1899, xlii. 399.—39. Ramón y Cajal. *Arch. f. Phys. u. Anat.*, 1895, Anat. Abt. 375.—40. Reiner und Schnitzler. *Centralbl. f. Physiol.*, Leipzig u. Wien, 1894, viii. 684.—41. Riegel und Jolly. *Virchows Arch.*, 1871, lii. 218.—42. Roy and Sherrington. *Journ. Physiol.*, London, 1890, xi. 85.—43. Salathé. *Trav. du labor. de Marey*, 1876, ii. 345.—44. Schulten. *Arch. f. Ophthal.*, Leipzig, 1884, xxx. Abt. iii. 89.—45. Shimamura. *Neurol. Centralbl.*, 1894, xiii. 685, 769.—46. Wertheimer. *Arch. de physiol.*, 1895, 5. s., vii. 735.—47. Wiechowski. *Arch. f. exper. Path. u. Pharmak.*, Leipz., 1902, xlviii. 376; 1905, lii. 389.—48. Wiggers, C. J. *Am. Journ. Physiol.*, 1908, xxi. 454. **Cerebral Anaemia**: 49. Bastgen. *Puls-Frequenz u. Hirndruck bei capillärer Embolie der Hirngefässe.* Würzburg, 1879.—50. Cooper, Astley. *Guy's Hosp. Rep.*, London, 1836, i. 457.—51. Corin. *Bull. Acad. roy. des sc. de Belg.*, Bruxelles, 3. sér., xiv.—52. Couty. *Compt. rend. Soc. biol.*, Paris, 1876, xxviii. 296.—53. Ferrari. *Wien. med. Jahrb.*, 1888, 81.—54. Hill, Leonard. *Physiol. and Path. of the Cerebral Circulation*, London, 1896.—55. Horsley and Spencer. *Brit. Med. Journ.*, 1889, i. 457.—56. Kussmaul und Tenner. *Molesch. Untersuch.*, 1857, iii. 1.—57. Markwald. *Ztschr. f. Biol.*, München, 1890, xxvi. 259.—58. Orchansky. *Arch. f. Physiol.*, Berlin, 1883, 297.—59. Panum. *Ztschr. f. klin. Med.*, vii.—60. Pitt, Pembrey, and Allen. "Observations on Cheyne-Stokes Respiration," *Med.-Chir. Trans.*, London, 1907, xc. 49.—61. Russell, A. E. "Pathology of Epilepsy," *Proc. Roy. Soc. Med.*, 1908, i. (Med. Sect.), 72.—62. Schiff. *Lehrbuch d. Physiol.*, 1858-9, 108.—63. Spanbock. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1888, viii. 283. **Cerebral Compression**: 64. Adamkiewicz. *Sitz. d. k. Akad. d. Wiss.*, Wien, 1883, lxxxviii. Abt. iii. 11, 231.—65. Bergmann. *Deutsche Chirurgie*, 1880, xxx. 274.—66. Cybulski. *O Ucisku Mozgu*, Krakow, 1891; *Centralbl. f. Physiol.*, Leipzig u.

Wien, 1890-1, iv. 834.—67. DEAN. *Journ. Path. and Bacteriol.*, Edin. and Lond., 1892, i. 26.—68. DURET. *Arch. de physiol.*, Paris, 1874; *Étud. expér. sur les traumatismes cérébraux*, Paris, 1878.—69. HILL, LEONARD. *Phys. and Path. of the Cerebral Circulation*, London, 1896.—70. HORSLEY and SPENCER. *Trans. Roy. Soc. London*, 1892.—71. LEYDEN. *Virchows Arch.*, Berlin, 1866, xxxvii. 519.—72. NAUNYN und SCHREIBER. *Arch. f. exper. Path. u. Pharmak.*, Leipzig, 1881, xiv. 1.—73. PAGENSTECHER. *Exp. Stud. über Gehirndruck*, Heidelberg, 1871.—74. REINER und SCHNITZLER. *Wien. klin. Wchnschr.*, 1895, 371.—75. SCHULTEN. *Arch. f. Ophthalm.*, Leipzig, 1884, xxx. 1.

L. H.

THE REGIONAL DIAGNOSIS OF CEREBRAL DISEASE[1]

By Sir DAVID FERRIER, M.D., F.R.S.

Introductory.—The regional diagnosis of cerebral disease is, in some instances, comparatively easy; in others exceedingly difficult, or not infrequently altogether impossible. The case is simplest when a stationary lesion, such as softening, invades centres, tracts, or structures, the function of which has been determined with some accuracy, whether by anatomical, physiological, or clinical research, singly or conjointly. The case is most difficult when the lesion is of such a character as to exercise a widespread disturbance of the whole intracranial structures; or when it is seated in a region the exact function of which has not been accurately determined by any method of investigation; or, again, when it may be compensated by the corresponding region on the opposite side, or by other parts in the same hemisphere.

The data which enable us, so far as it is at present possible, to arrive at a correct regional diagnosis are seldom anatomical, physiological, or clinical alone, but rather a combination of all three; and in the following pages, therefore, reference will be made to whatsoever method of investigation seems best calculated to throw light on these questions.

Beyond the fact, familiar since the time of Aretaeus, that in hemiplegia the lesion is in the opposite hemisphere, comparatively little had been positively established by clinical investigation alone until recent years. Under conditions apparently similar there was so much diversity in symptomatology, that clinical research had not succeeded in establishing much beyond a few empirical generalisations—such as that lesion of Broca's region is the cause of certain forms of aphasia; and that irritation of the Rolandic area is the cause of unilateral convulsions, appropriately named "Jacksonian epilepsy"—generalisations extremely valuable in a diagnostic point of view, but unintelligible according to previous conceptions of the constitution of the organ of mind.

The chaotic darkness of clinical medicine was first illumined, and a new era inaugurated, by the experimental methods first begun by Fritsch and Hitzig, and since assiduously pursued by many investigators. These observers, apart from what they have positively established, and irrespective of those individual differences and discrepancies which not unnaturally arise in the investigation of vital problems of great complexity, have so stimulated thoroughness in clinical investigation and accuracy in pathological records, microscopical and macroscopical, that they have revolutionised cerebral physiology and pathology. It is, therefore, only in the observations of the last thirty odd years that the materials are to be found on which we may base reasonable conclusions or speculations as to the probable nature and extent of intracranial lesions or disturbances of function. The observations of previous generations derive a dignity and importance more as examples of practical rules or of laws otherwise arrived at, than as contributions to establish these laws. It is unnecessary here to describe the methods which have proved so fertile in the hands of Fritsch and Hitzig and their followers, as these are now matters of general knowledge. Suffice it to say that they combine both the excitation and destruction methods; that is, excitation by electrical stimulation, and destruction by methods calculated, as far as possible, to obviate primary or secondary implication of other structures, near or remote. Excitation and abolition, or impairment, of function practically embrace all the phenomena which can be induced by cerebral lesions; all lesions may therefore be classified into two great groups, *irritative* and *destructive.* These two classes of lesions, however, are not separable by hard and fast lines; for a lesion which is irritative in the first instance may in time prove destructive, and cause annihilation of the function which formerly it excited; and, similarly, lesions, essentially destructive from the first, may be the centre of a zone of irritation, so that irritative phenomena, of greater or less range, are superadded to the paralytic symptoms. An effect intermediate between that of irritation and destruction must also be taken into account, namely, the perturbation, inhibition, or temporary cessation of the functions of parts more or less remote from the seat of actual lesion. This is a dynamical influence, termed by v. Monakow *diaschisis,* most frequently seen in connexion with sudden lesions, and probably due to the rupture of the solidarity which normally exists between the several intracranial centres; but whether it should be regarded as being irritative or destructive, is not quite clear. As, however, the ultimate effect is the abolition or impairment of function, it may rightly be classed among the destructive lesions and designated indirect, in contradistinction to those which act directly on the parts in which they are primarily situated. It is the lesions of this last class which create so much difficulty in the accurate regional diagnosis of cerebral lesions, and which also largely explain the discrepancies which abound in the reports of experimental lesions in the brains of the lower animals; for the estimation of the duration and range of these inhibitory phenomena is by no means easy to calculate. It is necessary, therefore, to bear these considerations carefully in mind in attempting, among the phenomena of cerebral disease, to separate the essential from the accidental, and to refer to their respective causes the various symptoms met with in connexion with any given lesion.

---

[1] In the preparation of this article I gratefully acknowledge much valuable help from Dr. W. Aldren Turner, Dr. Ashley Mackintosh, Dr. Purves Stewart, and Dr. S. A. K. Wilson.

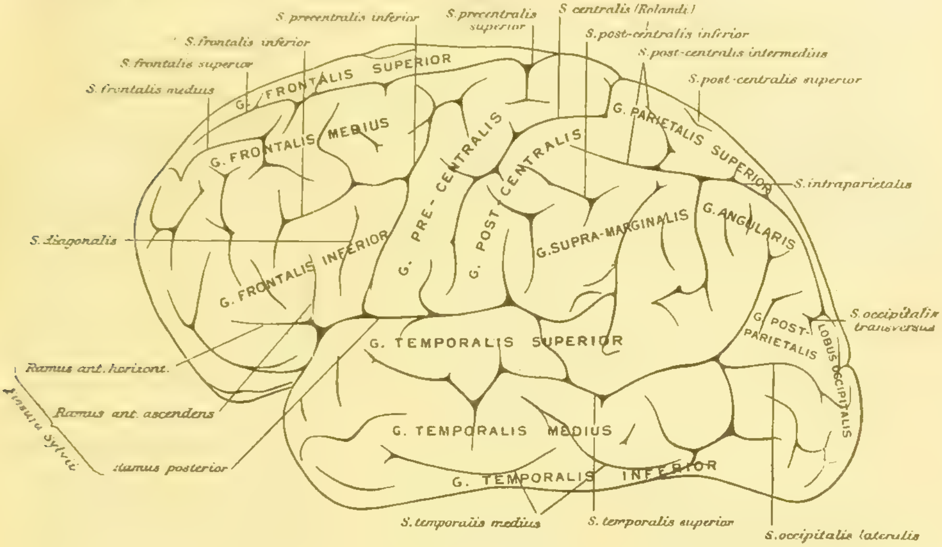

FIG. 3.—Cerebral hemisphere—lateral aspect. (After Symington.)

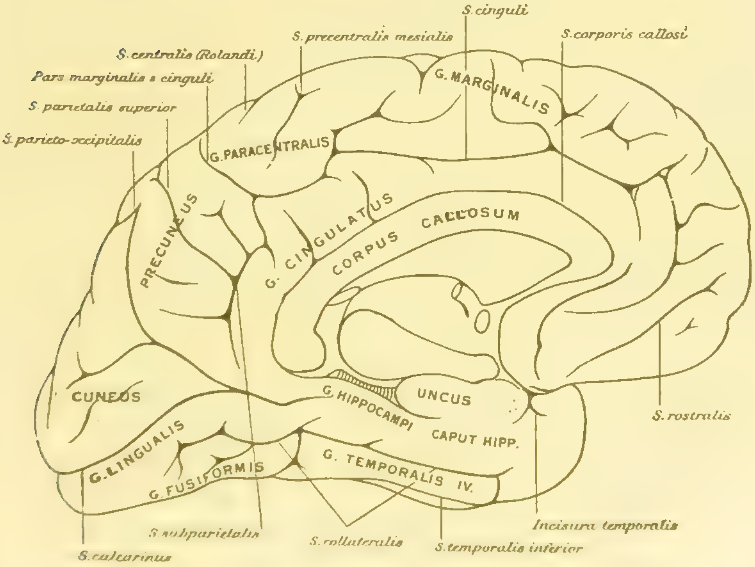

FIG. 4.—Cerebral hemisphere—mesial aspect. (After Symington.)

Destructive lesions are exemplified in their simplest form in atrophic degenerations; necrobiotic softenings, conditioned by non-irritative vascular occlusion; and slight aseptic trauma.

Irritative lesions are exemplified in inflammatory processes, neoplasms, and infective foci; whilst *indirect or inhibitory lesions* are exemplified in pathological processes of a sudden character, such as extensive injury, haemorrhagic extravasation, and the pressure and other remote effects of intracranial tumours.

For the purposes of this article the morbid anatomy of intracranial lesions is unimportant and will not be considered. What we have to consider *quâ* regional diagnosis, is whether the lesion be essentially irritative or destructive in character; and whether it be such as is calculated to exercise a disturbing influence on regions anatomically remote from the part actually invaded. A knowledge is also assumed of the general features of cerebral anatomy, and of the convolutional arrangement of the cortex, as well as of the vascular supply.

LESIONS OF THE FRONTAL LOBE.—The frontal lobe, as here defined, is not coextensive with the frontal lobe of anatomists, but is restricted to that portion of the cerebral hemisphere which lies anterior to the precentral sulcus and its ideal prolongation upwards to the longitudinal fissure (Fig. 3). This line cuts off the base of the superior frontal convolution which functionally should be included in the Rolandic area. Physiological experiment further indicates that the frontal lobe as so defined must be divided into a *prefrontal* and a *postfrontal* area. The division between the two is indicated in the brain of the macaque monkey by a shallow transverse sulcus, the homology of which in man is somewhat doubtful. Histological investigations, more particularly those of Campbell and Brodmann, have opened up a suggestive line of research in the direction of correlation between structure and function, though for the present mere hypothesis is far in advance of established truth. Campbell divides the cortex in front of the precentral sulcus into three divisions (Fig. 5)—(1) The "intermediate precentral area," which covers part of the precentral gyrus, the base of the superior and middle frontal gyri, and a considerable portion of the inferior frontal gyrus, and part of the frontal operculum. It resembles the precentral cortex, but is devoid of Betz-cells. (2) The "frontal area," which covers the anterior half of the superior frontal, nearly the whole of the middle frontal, and the anterior part of the inferior frontal gyrus. Mesially it occupies a small portion of the orbital gyrus and orbital surface. It differs from the intermediate precentral cortex in the almost complete absence of fibres of large calibre, and diminution in size of the large pyramidal cells in the external and internal layers. (3) The "prefrontal area," which covers the most anterior portion of the middle frontal gyrus, the anterior portion of the marginal gyrus on the mesial surface, and a large portion of the orbital surface. It is poor both in fibres and in cells, and the cortex is shallow. The frontal lobe—including the prefrontal and postfrontal areas — forms, according to Flechsig, *an association area*, in which are correlated impressions from the various sensory or projection areas of the cortex. The frontal lobe, however, has its own projection system in the form of the *fronto-pontine* tract (Flechsig), which, as Dr. Turner and I have shewn, degenerates downwards after lesions both of the prefrontal and frontal area in the most mesially situated portion of the internal capsule, as seen in frontal section, and in the corresponding situation in the crus cerebri. We could not trace it beyond the pons. Flechsig is of opinion that it connects the frontal lobe with the opposite cerebellar hemisphere indirectly through the nucleus pontis.

Similar degeneration was described by Brissaud as the result of lesions of the anterior limb of the internal capsule in man. As the degeneration could not be traced into the anterior pyramid, he concludes that the tract of the foot of the crus connects the frontal region with the motor nuclei of the medulla oblongata. Bechterew and Witkowsky have observed atrophy of the cells of the substantia nigra after old lesion of the anterior part of the internal capsule with descending degeneration in the crus. Dr. Turner and I confirmed this in our experiments on the prefrontal lobe. The frontal lobe is considered by Bechterew to be connected with the occipital lobe by a large tract of fibres, termed the fasciculus longitudinalis superior. Removal of the prefrontal lobe, however, does not, according to our observations, occasion any degeneration traceable into the occipital lobe. The frontal lobe is also connected by projection fibres with the anterior and median nuclei of the optic thalamus which degenerate after removal of the frontal lobe (von Monakow, Rutishauser, Dejerine).

Electrical irritation of the posterior half, or postfrontal area, causes movements of the head and eyes to the opposite side ; whilst irritation of the prefrontal area is uniformly negative, or is occasionally accompanied by ocular movements only. The area for movements of the eyes is placed by Sherrington and Grünbaum more anteriorly in the chimpanzee (see Fig. 9) than in the macaque (Fig. 7). It would appear, however, from Russell's experiments that the lateral, or conjugate, deviation of the eyes to the opposite side becomes modified to upward or other movements, when the external rectus of the opposite eye is divided—a fact which is taken to indicate that other ocular movements are represented in this area, but are usually concealed by the stronger and more easily excited lateral movements.

When the postfrontal region in monkeys is destroyed, a temporary deviation of the head and eyes to the side of the lesion always occurs. This, however, is only transient, even when the destruction has been almost, if not absolutely, complete. In two experiments which I have described (15) the postfrontal area was destroyed in both hemispheres. The animals were unable to turn the head and eyes to either side for a day after the operation. At first they were unable to look around when sounds were made in close proximity to the ear, or, if they did, they moved the trunk and head *en masse.*

Removal of the prefrontal region alone causes no discoverable physiological symptoms, either sensory or motor. I found, however, in several instances, that, after the symptoms which followed destruction of the postfrontal area had entirely disappeared, the subsequent destruction of the prefrontal area induced paralysis of the head and eyes of exactly the same nature as before. Thus, in one case, after cauterisation of the whole

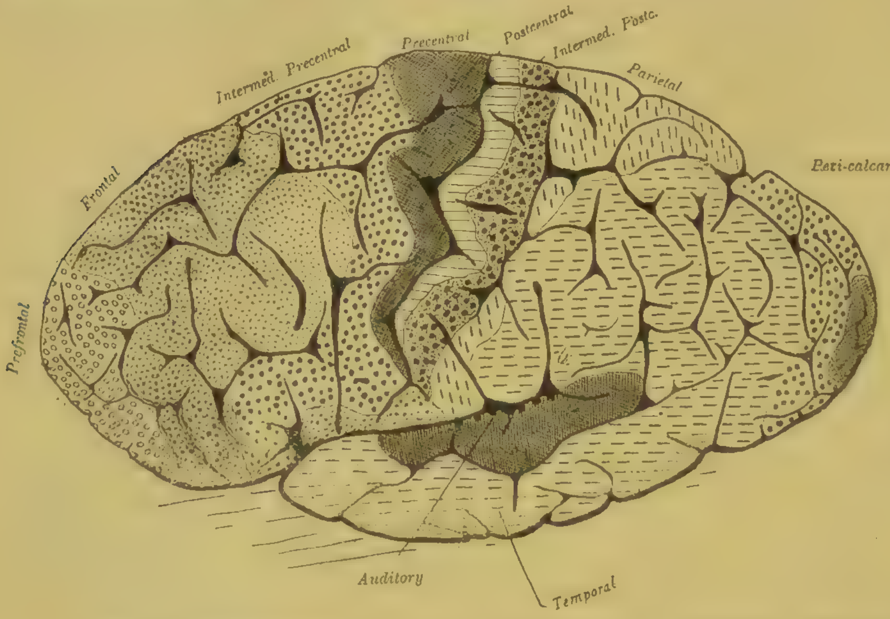

FIG. 5.—Histological areas of the cortex—lateral aspect. (After Campbell, modified.)

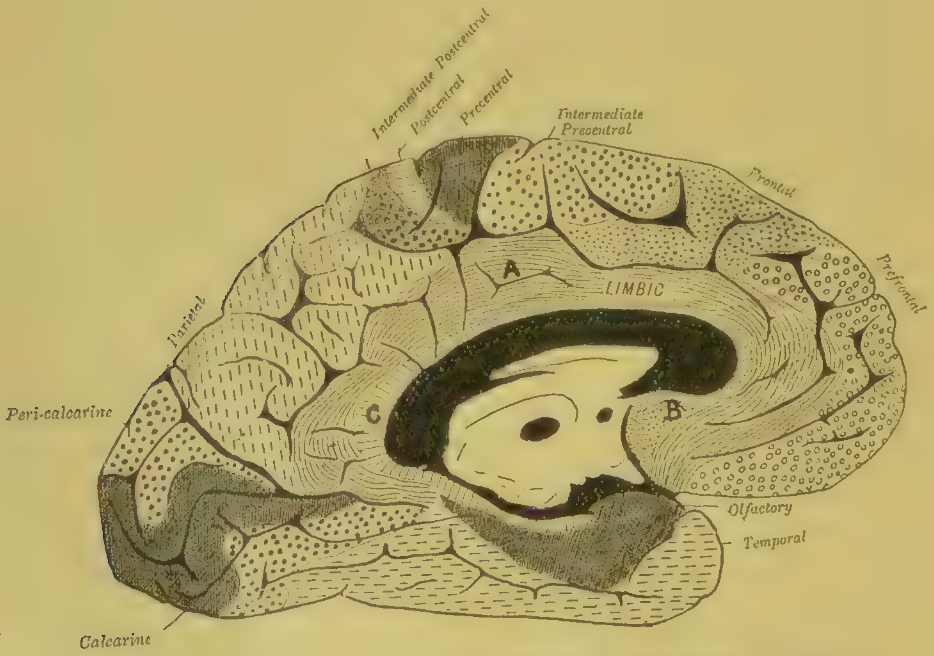

FIG. 6.—Histological areas of the cortex—mesial aspect. (After Campbell, modified.)

of the excitable area on its convex as well as mesial aspect, the animal, which at first exhibited marked conjugate deviation of the head and eyes to the side of the lesion, recovered within three days to such an extent that the defects were no longer perceptible. Extirpation of the prefrontal region anterior to the former lesion, a month later, reproduced the conjugate deviation of the head and eyes to the side of the lesion. The conjugate deviation of the eyes continued for some time after the distortion of the head had been recovered from, but within three days this defect also had disappeared.

It is difficult to remove both frontal lobes thoroughly without injuring the head of the nucleus caudatus, or causing lesions incompatible with long survival. However, in one experiment Turner and I succeeded in removing the whole of both frontal lobes after an interval of more than a month between the two operations. The removal of each lobe was followed by temporary conjugate deviation of the head and eyes; but, notwithstanding the apparent total removal of the frontal lobe on both sides, there was no permanent inability to move the head and eyes freely in all directions. It would appear, therefore, from this experiment that, though the frontal lobes are evidently in relation with the movements of the head and eyes, complete destruction of these regions does not give rise to permanent paralysis; and still more striking is the fact that in this same animal, after an interval of five weeks, destruction of the angular gyri, in which, as we shall see hereafter, some are inclined to place the centres for the ocular movements, did not cause ptosis, or paralyse the ocular movements. The animal, however, speedily succumbed to the shock of the operation, so that our period of observation was unfortunately short. When the lesion is accurately confined to the frontal lobe, and does not extend backwards to the motor region proper or cause general disturbance of the hemisphere, temporary conjugate deviation of the head and eyes to the side of lesion is the only discoverable physiological symptom.

Hitzig, however, after destruction of the frontal lobe, observed, along with impairment of the movements of the limbs, affection of vision of the opposite eye, and was inclined to think that this is not accidental, but that the frontal lobe is related to vision. In the last-mentioned experiment, we observed similar effects after complete extirpation of the left frontal lobe, together with a portion of the base of the superior frontal, anterior part of the gyrus fornicatus, and a part of the head of the nucleus caudatus. The lesion was therefore not strictly confined to the frontal lobe. In addition to the conjugate deviation of the head and eyes there occurred a paretic condition of the right arm and leg (continuing longer in the leg), and also a slight and temporary impairment of tactile sensibility, as well as distinct right hemianopsy. Bianchi has obtained similar results in monkeys. These symptoms, except the weakness in the right leg, entirely disappeared in the course of a month; and, as will be shewn by other experiments, cannot be attributed to lesion of the frontal lobe, as such.

The recorded cases in man of injury and disease strictly confined to the frontal lobes accord with the negative character of experimental lesions in monkeys, so far as relates to the sensory and motor faculties. The majority may also be described as latent, that is, not revealed by any strictly localising symptoms. Of 73 cases of lesion of the frontal lobes, unilateral and bilateral, collected from various sources, 24 were traumatic, caused, that is, by fractures, gunshot wounds, or perforating instruments; 4 were due to haemorrhage; 12 to abscess, mostly secondary; in 1 the abscess was secondary to mastoid disease; 25 were due to tumours; and 1 was a case of atrophy. The remainder were tabulated as "softening."

Haemorrhage.—With the exception of the inferior and posterior part of the middle frontal convolution, the frontal lobe, both on its convex and mesial aspect, is supplied by the anterior cerebral artery. This artery does not appear to be particularly liable to rupture or occlusion; hence haemorrhages, apart from trauma, and embolic or thrombotic softening are comparatively rare.

Traumatic Lesions.—Many cases are on record of recovery, without any obvious impairment of sensation or motion, after traumatic injuries of a most extensive character. One of the most remarkable is the famous American crowbar case. The subject of the lesion was a young man aged twenty-five. While engaged in stamping a blasting charge in a rock with a pointed iron bar, 3 ft. 7 in. in length, 1¼ in diameter, and weighing 13½ lbs., the charge suddenly exploded. The bar, propelled with its point first, entered at the left angle of the patient's jaw, and emerged anterior to the coronal suture. The patient was only momentarily stunned, and recovered without any defect of sensation or motion, except ptosis and loss of vision in the left eye, due to local injury of the optic and oculomotor nerves. Examination of the topographical relations of the brain and skull clearly proves that the track of the instrument was anterior to the Rolandic area, and could only have injured the excitable postfrontal area, unilateral lesions of which, as we know from experiments, cause transient symptoms only. Whether they occurred at all in this patient has not been noted.

Another similar and not less remarkable case came under my observation. It was that of a young man, aged twenty-six, subject to epileptic fits, who, apparently in a dazed condition, and without purpose, discharged a pistol through the roof of his mouth. The bullet lodged under the frontal bone, which it fractured and partially raised. The fractured bone was elevated and the bullet extracted by Dr. Walker of Peterborough. The position of the opening was half an inch to the left of the middle line, and an inch anterior to the coronal suture. The patient entirely recovered, with the exception of ptosis and weakness of the superior rectus in the left eye, and diplopia for objects situated above the horizontal line. When I saw the patient, within three months of the injury, there was no trace of any affection of motion or sensation, general or special. Many other cases of unilateral traumatic lesion are on record; but it is not necessary to refer to them in detail, as they merely illustrate the same negative character of such lesions.

Of bilateral lesions one of the most interesting is that reported by Davidson. This was the case of a labourer who received a blow on the side of the head with an iron hook, which fractured the frontal bone and injured the brain as far back as the coronal suture. The injured portion of the brain included, on the right side, the greater part of the superior and middle frontal convolutions; and, on the left, a considerable amount of the middle of the superior frontal convolution. With the exception of an occasional spasmodic extension of the right arm, there were no other symptoms in the domain of motion or sensation.

Abscesses in the frontal lobe are, for the most part, consecutive to injuries or perforating wounds, or to diseases of the frontal, ethmoid, or orbital bones; but a case has been reported by Prentiss of abscess in the left anterior lobe secondary to abscess in the right petrous bone and lateral sinus. In most of these cases there was entire absence of sensory or motor symptoms, and in many there was nothing noteworthy, or recorded, in reference to the mental condition. In some, however, to be alluded to subsequently, the mental condition was such as to attract attention.

Pitres reports a case of injury to the left frontal region which gave rise to headache, vomiting, and death two months afterwards, without having caused any obvious affection of intelligence, or paralysis of motion or sensation. After death an abscess was found in the centre of the left frontal lobe. Sir W. Macewen reports a case of a blow on the forehead by a stone; suppuration occurred in the wound, followed by headache, drowsiness, vomiting, and occasional rigors, and double optic neuritis. Apart from these general symptoms there was no affection of motion or sensation. The patient was trephined, and an abscess was evacuated in the left frontal lobe.

Tumours form a large proportion of the cases of disease of the frontal lobe, either in the substance of the lobe itself, or growing from the dura mater, and compressing and destroying the subjacent cortex. Apart from the general symptoms of cerebral tumour, namely, headache, vomiting, optic neuritis, and epileptiform convulsions, in many instances there has been nothing to indicate with certainty the seat of the disease. Hun reports a case of one and a half years' duration, with symptoms merely of general weakness, in which death suddenly occurred in an attack of coma. A tumour the size of a small hen's egg was found in the lower part of the frontal lobe. Dr. Hale White reports a case of a woman who for one month had suffered from headache, vomiting, and occasional loss of sight. Optic neuritis existed, but there were no special symptoms. The patient was found one day dead in bed. The post-mortem examination revealed a glioma in the frontal lobe. Dr. Edwin Bramwell has recorded a case of large glioma of the right frontal lobe. Though repeatedly examined by competent observers, no localising symptom was discovered till just before death, which was sudden. There was no anosmia, and no discernible mental change except some irritability of temper. For four and a half years she had been subject to general epileptic seizures. Many similar cases might be mentioned.

In some cases the tumour has been indicated not by any symptoms referable to the frontal lobe, as such, but by affection of the nerves in the anterior fossa. Thus, Schlager, quoted by Lépine, reports a case of a woman who for some years before her death suffered from vertiginous sensations and epileptiform attacks, preceded by an olfactory aura. There was no other affection of motion or sensation. She died comatose. A tumour, the size of a pigeon's egg, was found in the substance of the left frontal lobe, and had completely destroyed the left olfactory tract. In other cases there have been observed indications of paresis or paralysis of the ocular nerves, fulness or protrusion of the eyeball, and failure of vision in the eye on the side of lesion, not to be accounted for by the general optic neuritis. These observations are true also of abscesses.

Griffith and Sheldon have reported a case of tumour occupying the anterior fossae, and thrusting itself between the frontal lobes, chiefly destroying the orbital surface and exerting pressure upon the olfactory tracts and optic nerves. The symptoms in this case, in addition to those of cerebral tumour in general, were impairment of memory, loss of smell on the right side, and paresis of the left abducens. Before death both discs became atrophied, with almost total loss of vision. There were no motor or sensory symptoms, and, except for some attacks of maniacal excitement, the mind was quite clear and coherent until shortly before death. The occurrence of anosmia on the side of lesion is not infrequent, and may be of important localising value.

In another group of cases there has been superadded to the general symptoms of cerebral tumour some affection of motion and sensation on the opposite side of the body. This is true not only of tumours, but also of abscesses, and of inflammatory softening extending backwards from the primary lesion of the frontal lobes. These symptoms are undoubtedly due to pressure on, or direct implication of, the Rolandic zone or the hemisphere in general, and often afford valuable diagnostic indications of the seat of the disease, which in its earlier stages may not have declared itself by any definite symptoms.

Of a similar character are the symptoms of cerebellar disease first described by Bruns, which have been frequently noted since he called attention to them. Bruns described 4 cases of tumour of the frontal lobe in which, in addition to some degree of paresis or paralysis of a hemiplegic or monoplegic character on the opposite side, there were also disturbances of equilibrium and co-ordination. In one case (sarcoma affecting both frontal lobes and involving the genu of the corpus callosum) there was instability of equilibrium with a tendency to retropulsion; and in another (haematoma indenting the left frontal lobe) the patient could not stand without support, and had slight paralysis of the right hand and face. A case of frontal lobe tumour with purely cerebellar symptoms has been recently under my own care. And of 24 cases of frontal tumour occurring in the National Hospital within the last ten years, analysed for me by Dr. S. A. K. Wilson, 4 out of 11 of right frontal tumour and 1 out of 13 of left frontal tumour exhibited cerebellar symptoms. One case diagnosed as left cerebellar proved on post-mortem examination to be left frontal. This combination of symptoms, as Bruns indicates, is in all probability due to pressure exerted backwards on the cerebellum in the line of thrust, and is peculiar to tumours of the frontal region. Dr. Grainger Stewart has frequently observed in tumours of the prefrontal or postfrontal area a fine vibratory tremor of the homolateral arm, and less markedly of the leg, on holding the limb outstretched—together with loss or speedy exhaustion of the contralateral superficial reflexes, especially the abdominal and epigastric. These symptoms, however, were not present in Dr. E. Bramwell's case, specially examined on these points.

The symptoms hitherto described are in accordance with the negative effects, *qua* motion and sensation, which have been uniformly found in monkeys after extirpation of the frontal lobes proper; and the occurrence of spasmodic or paralytic affections of the limbs is also in harmony with the effects of lesions primarily or secondarily involving the motor centres of the Rolandic area. It is a question, however, whether the symptoms of injury and disease of the frontal lobes in man entirely bear out those which have been described as the result of irritation and destruction of these regions in monkeys and other animals. It has already been stated that irritation of the postfrontal area uniformly causes conjugate movement of the eyes and head to the opposite side, and that extirpation of this region is uniformly followed by conjugate deviation of the head and eyes to the side of lesion. This, however, is only of temporary duration, even when the postfrontal centres have been entirely destroyed; and it would be in accordance, therefore, with the results of experimental physiology that such phenomena should not occur in man except in connexion with irritative or sudden destructive lesions of the area in question.

Irritation would cause conjugate movement of the eyes to the opposite side, whilst sudden destruction would cause deviation to the side of lesion, owing to the non-antagonised action of the centres in the opposite hemisphere. Slowly progressive lesions, however, need not reveal themselves by conjugate deviation, as compensation would be effected as rapidly as the process of destruction. Chouppe has reported a case of tuberculous meningitis in which, in addition to the pain in the head, vomiting, and so forth, the most remarkable symptom was rotation of the head and eyes to the right. This could be overcome with moderate effort, but the head and eyes returned to their abnormal position when left to themselves. There were no symptoms of paralysis or contracture. After death a superficial lesion of the size of a franc piece was found on the superior part of the middle frontal convolution in the left hemisphere. This may be regarded as in all probability an irritative lesion of the oculomotor area. In Gouget's case there was conjugate deviation of the head and eyes to the side of lesion; but as it was accompanied by general hemiplegia, it does not admit of being referred to limited lesion of the left frontal lobe. In Lannelongue's case the conjugate deviation of the head and eyes to the left might perhaps be attributed to the lesion at the base of the left second frontal convolution.

Apart from the cases which I have quoted, however, I do not find any very satisfactory evidence of conjugate movement of the head and eyes having been observed in connexion with lesions implicating the central regions homologous with the ocular centres in the monkey.

I have elsewhere (13) described the *mental symptoms* observable in monkeys after extirpation of the frontal lobes. These, however, only occur when the lesion is bilateral, and some experimenters (Horsley and Schäfer) have failed to corroborate them. They may easily escape superficial observation, and it is only by careful comparison of the animals before and after operation that they are capable of being detected, and even then it is difficult to state their nature precisely. The animals do not appear to have lost any of their mental faculties entirely. They, however, exhibit a curious listless apathy or purposeless restlessness, and certainly lose the intelligent keenness and cunning which they possessed before the operation. I confirmed my earlier observations in a more recent experiment (1894) with Dr. Turner. The whole of the frontal lobes, including the oculomotor centres, were entirely extirpated on both sides. Though the animal recovered perfectly, with the exception of slight weakness in the right leg, and enjoyed excellent physical health, the mental alteration, of the character just described, was so striking that there could be no question of its reality. Bianchi has also observed noteworthy psychical changes in monkeys after removal of both frontal lobes, which he describes as a disaggregation of the personality, and changes in character. Franz also by special experimental tests has demonstrated a psychical degradation which he attributes to defect of associational processes of which the frontal lobes are the principal seat.

It has already been stated that in a large proportion of the cases of disease of the frontal lobe in man no mental symptoms have been noted; but in others they have been so marked as to have deserved special mention in the clinical records. Many of the cases have, however, been tumours, and it is therefore a question how far these symptoms should be attributed to lesion of the frontal lobes, as such, apart from general disturbance of the brain. But mental symptoms, of the character above described, have also been observed in connexion with lesions of a purely stationary character, not calculated to affect the brain as a whole. A case has been recorded by Baraduc of atrophy of the frontal convolutions in both hemispheres, due to obliteration of the arterial blood-supply. The patient, who was an inmate of the Hospice de Ménages for six years, exhibited no paralysis of sensation or motion. He was, however, in a state of dementia, marching about restlessly the whole day, picking up objects from the ground, never speaking, and quite oblivious of all the wants of nature. In Davidson's case, referred to on p. 45, the only symptoms of the cerebral lesion, apart from occasional spasm of the right arm, were of a psychical nature. Though the patient seemed to understand all that was said to him, and did what he was told, "every action he performed left the impression on the mind of the observer that it was purely automatic or machine-like." Marked mental deficiency has also been frequently observed in connexion with arrested development or atrophy of the frontal lobes. Thus, in the case of a girl aged fifteen reported by Cruveilhier, who had remained in a state of idiocy from birth, the anterior two-thirds of the frontal lobes were completely deficient. Dr. J. S. Bolton maintains that the prefrontal region—specially the layer of pyramidal cells—is under-developed in all grades of mental deficiency, and undergoes primary atrophy in proportion to the advance of dementia.

I have recorded a case of a tumour of the left frontal lobe in a man aged forty, who, when he came under observation, in addition to the general phenomena of cerebral tumour, exhibited symptoms essentially mental. These consisted chiefly in great failure of memory, inability to concentrate his attention, general mental hebetude, and a continual tendency to fall asleep.

A successful operation for tumour in this region was performed by Durante. The symptoms on which Durante based his diagnosis of tumour of the frontal lobe were loss of memory and change of disposition, anosmia, and some downward and outward displacement of the left eye. The patient recovered perfectly after removal of the tumour. The symptoms in a case recorded by M'Burney and Starr were headache, vomiting, and optic neuritis, severe and constant pain in the frontal region, increased by pressure or percussion, and marked mental dulness and difficulty in concentration of attention, together with slight hemiplegic weakness on the right side. A case with similar symptoms, dependent on an infiltrating glioma the size of a hen's egg in the left frontal lobe, is related by Knapp. Of 24 cases of tumour of the frontal lobe in the National Hospital (Wilson's collection), comprising 11 in the right and 13 in the left, 8 of the 11 and 11 of the 13, *i.e.* 19 in all, exhibited marked mental deterioration. In Starr's collection of 23 cases of frontal tumours one-half shewed mental disturbance. Other important collections and analyses have been made by Williamson, Welt, Phelps, Müller, and Schuster. According to Schuster, tumours of the frontal lobes are associated with psychical disturbances three times as often as those situated in other parts of the brain.

The mental symptoms met with in cases of frontal tumour are variable and cannot always be discriminated from (if they are not in many cases altogether due to) those caused by increase of intracranial pressure, irrespective of locality. In many there has been a condition of mental dulness, inattention, loss of memory, and lethargy. In others there has been a peculiar change of character termed *Moria* (Jastrowitz) or *Witzelsucht* (Oppenheim), *i.e.* a tendency to make silly jests or puns :—a jocularity which contrasts markedly with the patient's miserable condition otherwise. Since attention has been called to this particular symptom it has been noted by many recent observers, and according to Schuster it has been recorded in 15 out of 147 cases of frontal tumour, *i.e.* in about 10 per cent. It was present in some of the cases in the National Hospital above referred to. The symptom, however, has been observed in cases of tumours not specially invading the frontal lobes, and its true nature and relation to impairment of the higher intellectual faculties require further elucidation.

An examination of the cases mentioned above, as well as of many others which are to be found in medical records, shews that the regional diagnosis of lesions of the frontal lobes, independently of external indications, such as fracture and the like, may be a matter of doubt if not of absolute impossibility. It is abundantly evident that extensive lesions may exist in one or even both frontal lobes without revealing themselves in any impairment of motion or sensation, general or special; or even in mental symptoms of such character as to have attracted special attention.

In connexion also with lesions of the frontal lobes reference may be made to the condition termed *motor apraxia* or aphasia of the limbs (*vide* p. 447).

Summary.—(i.) Lesions of the frontal lobe may therefore be said to be not infrequently latent.

(ii.) On the other hand, in some cases, especially if the lesion is bilateral, and even in the case of lesions which are not calculated to cause pressure or disturbance of the brain in general, there may be mental symptoms of which the chief characteristics are failure of memory, hebetude, apathetic indifference or tendency to sleep, vague restlessness, and inability to concentrate attention, or a silly jocularity.

(iii.) The regional diagnosis of lesion of the frontal lobe is rendered more probable if, in addition to the psychical symptoms, there occur convulsive or paralytic symptoms, monoplegic or hemiplegic, on the opposite side of the body. These symptoms are indicative of extension of the lesion backwards into the Rolandic area.

(iv.) The diagnosis of lesions of the frontal lobe may be made with still greater certainty if, in addition to the symptoms enumerated under paragraphs ii. and iii., there are signs of pressure in the anterior fossa; consisting in protrusion or displacement of the eyeball, with perhaps unilateral loss of vision, anosmia, and paralysis of one or other of the oculomotor nerves.

(v.) The diagnosis is confirmed if, in addition to some, or a combination of several, of the above-mentioned symptoms, there is pain on deep pressure on the frontal bone. This local pain may, however, be entirely absent if the lesion be subcortical, and is especially to be found in cases of tumour causing tension or irritation of the dura mater.

REFERENCES

1. Baraduc. *Progrès méd.*, Paris, 1876, iv. 598.—2. Bechterew. *Leitungsbahnen*, 1894, 157.—3. Bianchi. "Functions of the Frontal Lobes," *Brain*, 1895, xviii. 497.—3*a*. Bolton, J. S. *Phil. Trans.*, Lond., 1900, B. cxciii. 165.—4. Bramwell,

E. *Rev. Neurol. and Psychiat.*, Edin., 1908, vi. 577.—5. BRISSAUD. *Recherches anatomo-pathol. et physiol.*, Paris, 1880.—6. BRODMANN. *Journ. Psychol. u. Neurol.*, 1903 *et seq.*—7. BRUNS. *Deutsche med. Wchnschr.*, 1892, xviii. 138.—8. BURR. *Philadelphia Neurol. Soc. Trans.*, ii. 18.—9. CAMPBELL. *Histological Studies on the Localisation of Cerebral Function*, 1905.—10. CHOUPPE. *Bull. Soc. anat.*, 1871, 2. s., xvi. 380.—11. DAVIDSON. *Lancet*, 1877, i. 342—12. DURANTE. *Trans. Ninth Intern. Med. Congress*, 1887, i. 570.—13. FERRIER. *Functions of the Brain*, 2nd edition, 1886.—14. *Idem. Phil. Trans.*, 1884, clxxix. 479.—15. *Idem. Cerebral Localisation*, 1890.—16. *Idem. Lancet*, 1892, i. 1240.—17. FLECHSIG. *Die Localisation der geistigen Vorgänge*, 1896.—18. FRANZ. "The Frontal Lobes," *Arch. of Psychol.*, March 1907.—19. GOUGET. *Bull. Soc. anat.*, 5, 6, 349.—20. GRAINGER STEWART. *Lancet*, 1906, ii. 1209.—21. GRIFFITH and SHELDON. *Journ. Ment. Sc.*, 1890, xxxvi. 223.—22. HARTMANN. *Monatsschr. f. Psychiat.*, Berlin, 1907, xxi. 97-248.—23. HITZIG. *Arch. f. Psychiat.*, 1887, xv.—24. HORSLEY and SCHÄFER. *Phil. Trans.*, 1888, B. clxxix. 1.—25. HUN. *Amer. Journ. Med. Sc.*, 1887, xciii. 140.—26. JACKSON, HUGHLINGS. *Med. Times and Gaz.*, June 23, 659.—27. JASTROWITZ. *Deutsche med. Wchnschr.*, 1888, xiv. 125.—28. KNAPP. *Intracranial Growths*, 1891, 48, Case 4.—29. LANNELONGUE et MAUCLAIRE. *Bull. Soc. anat.*, 1892, 5. s., vi. 185.—30. LÉPINE. *Localisations dans les maladies cérébrales*, 1875, 97.—31. LIEPMANN. *Monatsschr. f. Psychiat.*, Berlin, 1900, viii. 15, 102, 182 *et passim.*—32. LIEPMANN und MAAS. *Berlin. klin. Wchnschr.*, June 1907.—33. M'BURNEY and STARR. *New York Acad. of Medicine*, 1893.—34. MACEWEN. *Pyogenic Diseases*, 183.—35. von MONAKOW. *Gehirn-Pathologie*, 1905.—36. MORRISON. *Philadelphia Neur. Soc. Trans.*, ii. 5.—37. MÜLLER. *Allg. Ztschr. f. Psychiat.*, 1902, lix. 830.—38. OPPENHEIM. *Path. d. Gehirngeschwülste*, 1890.—39. PHELPS. *Amer. Journ. Med. Sc.*, Phila., 1902, cxxiii. 563, 751, and 1906, cxxxi. 457.—40. PITRES. *Lésions du centre ovale*, Paris, 1877, —41. PRENTISS. *Amer. Journ. Med. Sc.*, 1882, lxxxiii. 453.—42. RUSSELL, RISIEN. *Journ. Physiol.*, 1894, xvii. 1.—43. SCHUSTER. *Psych. Störungen bei Hirntumoren*, 1902.—44. STARR. *Amer. Journ. Med. Sc.*, 1884, lxxxvii. 366.—45. THOMPSON, GILMAN. "Tumours of the Frontal Lobe," *Med. News*, Phila., 1890, 586.—46. TREVES. *Lancet*, 1878, i. 344, 378.—47. VAN VLEUTEN. *Allg. Ztschr. f. Psychiat.*, 1907, lxiv. 203.—48. WELT. *Deutsches Arch. f. klin. Med.*, 1888, xlii. 339.—49. WHITE, HALE. *Brit. Med. Journ.*, 1885, i. 131.—50. WILLIAMSON. *Brain*, London, 1896, xix.—51. WILSON, S. A. K. "Study of Apraxia," *Brain*, London, 1908, xxxi. 164, 344.

DISEASE OF THE ROLANDIC AREA.—The Rolandic area includes the convolutions which bound the fissure of Rolando; namely, the ascending frontal or precentral convolution and the base of the superior frontal, the ascending parietal and its broadened base, the postero-parietal or superior parietal lobule, together with the corresponding portion of the marginal convolution which forms the mesial aspect of the hemisphere. In this region are situated specialised areas, stimulation of which by the electric current gives rise to certain definite movements of the opposite side of the body; namely, of the trunk, limbs, facial, lingual, and laryngeal muscles; and destruction of which causes hemiplegia, if the entire region be destroyed; or dissociated paralyses or monoplegias, if individual areas only are destroyed; the paralysis or monoplegia in this case corresponding to the movements which are excited by the electric current. The excitable areas, according to the results obtained by myself, Schäfer, Horsley, and Beevor in the lower monkeys (macaques), are briefly as follows (Fig. 7 and Fig. 8).

At the upper extremity of the central convolutions, and extending over the margin of the hemisphere into the posterior part of the marginal convolution—paracentral lobule,—electrical stimulation gives rise to

LLEGE
PITAL

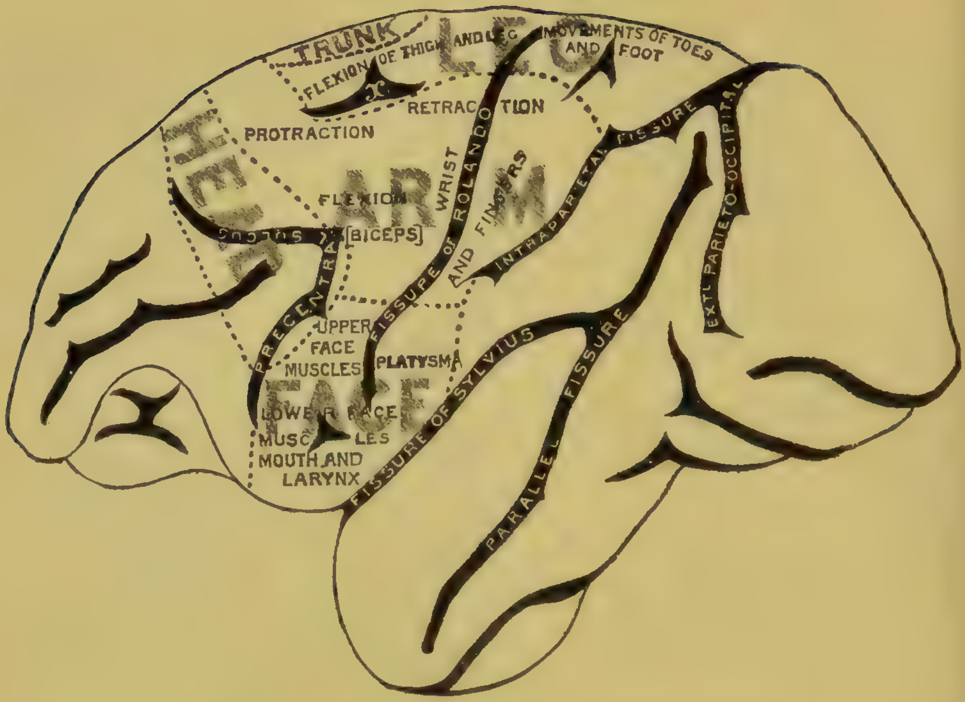

FIG. 7.—Motor areas—lateral aspect. (Horsley and Schäfer.)

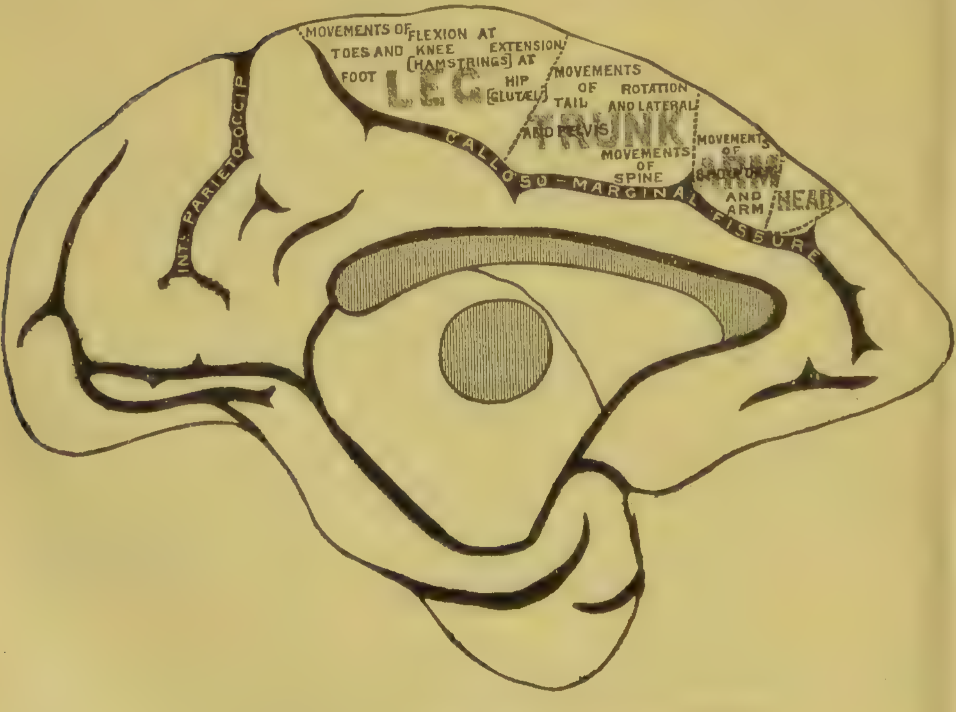
MOVEMENTS OF TOES AND FOOT
FLEXION AT KNEE (HAMSTRINGS)
EXTENSION AT HIP (GLUTÆI)
LEG
MOVEMENTS OF TAIL AND PELVIS
ROTATION AND LATERAL MOVEMENTS OF SPINE
TRUNK
MOVEMENTS OF SHOULDER AND ARM
ARM
HEAD
CALLOSO-MARGINAL FISSURE
INT. PARIETO-OCCIP

FIG. 8.—Motor areas—mesial aspect. (Horsley and Schäfer.)

movements of the *lower extremity*. The movements so excited vary with the position of the electrodes in this area. Behind the fissure of Rolando they are chiefly, or exclusively, of the foot or toes; in front of the fissure of Rolando they are combined with movements (flexion) of the leg and thigh. With minimal stimulation (Horsley and Beevor) it is possible to note differentiated movements of the lower limb with still greater minuteness, and, in particular, the great toe may be separately thrown into action by stimulation at the uppermost extremity of the fissure of Rolando.

Below the leg area, and partly in front of it, and occupying the middle third, or rather middle two-fourths, of the central convolutions, there is an area, stimulation of which causes movements of the *upper extremity*. In this area one may distinguish, more or less completely, protraction and retraction of the upper arm, flexion and supination of the forearm, and movements of the wrist, thumb, and fingers. The proximal movements are more particularly represented in the upper part of this area; the distal movements, namely, those of the fingers and thumb, in its lower part. By minimal stimulation of the lower extremity of the intraparietal sulcus the thumb alone may be thrown into action.

Below the arm area, and occupying the lower third of the central convolutions, electrical stimulation causes movements of the *face*, *mouth*, *tongue*, *and larynx*. In the upper part of this area stimulation in front of the fissure of Rolando causes movements of the upper facial muscles, and behind this fissure, of the platysma myoides. In the lower portion, stimulation causes movements of the mouth and tongue—protrusion of the tongue being generally caused by stimulation anterior, and retraction by stimulation posterior to the fissure of Rolando. In the anterior division, also, of the lower half of this area stimulation gives rise to phonatory closure of the vocal cords (Horsley and Semon), and occasionally, as I have found in some animals, to actual phonation.

The Rolandic region, as has been stated, extends over the margin of the hemisphere into the marginal convolution, on the mesial aspect of the hemisphere; and stimulation of this convolution from before backwards gives rise to movements of the arm and shoulder-girdle, trunk and pelvis, and lastly of the leg, foot, and toes (Horsley and Schäfer). Movements excited by stimulation of this region, however, are less capable of being exactly differentiated one from another. Though there is a similarity in position of the various centres in the lower and anthropoid apes, the experiments of Beevor and Horsley on the orang, and of Profs. Grünbaum and Sherrington on the chimpanzee, shew that the differentiation is much greater, and individual movements of the limbs can be isolated to a much greater degree than in the macaque. In the orang, however, Beevor and Horsley found the excitable area less extensive than in the macaque, the ascending parietal or postcentral gyrus giving much fewer results, and the superior parietal lobule practically inexcitable. The more recent experiments of Profs. Grünbaum and Sherrington on the chimpanzee by

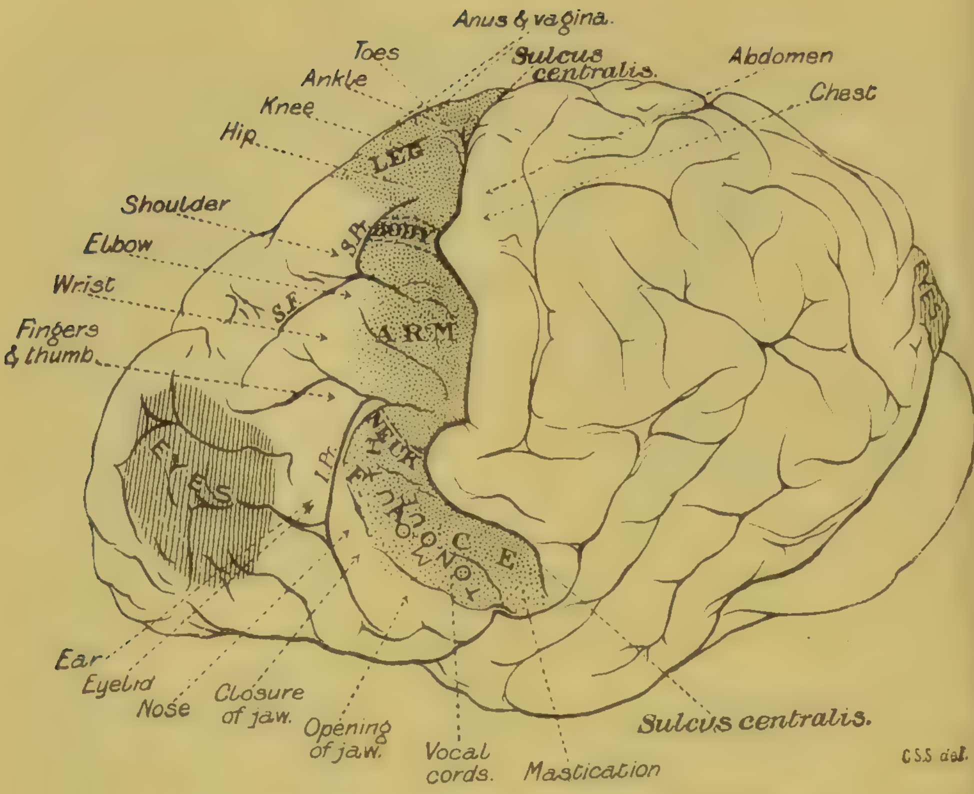

FIG. 9.—Motor areas of the chimpanzee. (Grünbaum and Sherrington.)

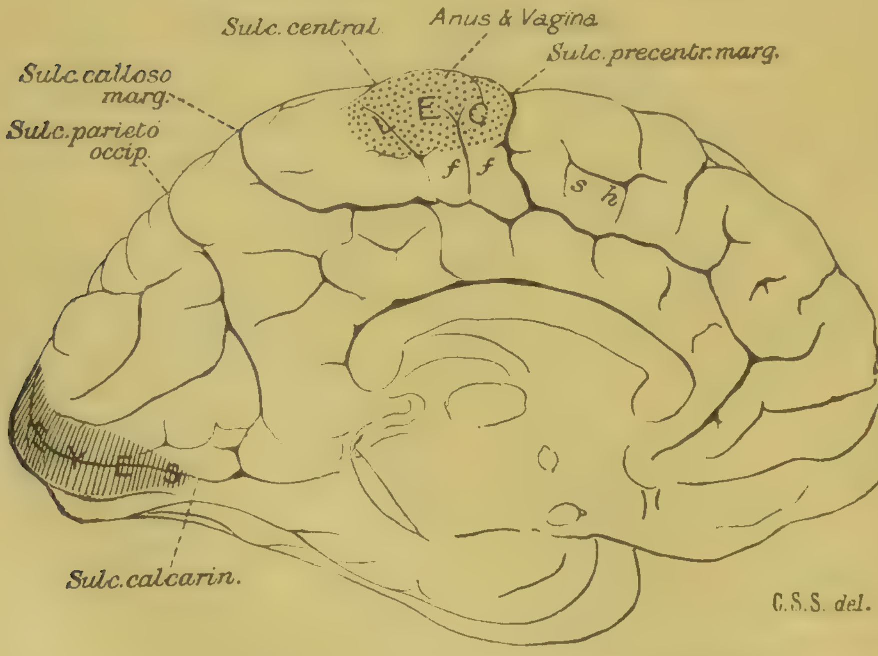

FIG. 10.—Motor areas of the chimpanzee. (Grünbaum and Sherrington.)

means of the unipolar method of stimulation restrict the excitable area to the precentral, to the exclusion of the postcentral gyrus. Their results otherwise differ in several respects from those of Horsley and Schäfer, particularly as to the extent of the excitable area on the mesial surface (see Figs. 9 and 10). Whether the excitation of movements of the fingers and toes from the postcentral gyrus and superior parietal lobule respectively is the result, as has been suggested, of diffusion into, or hyper-excitability of, the real motor centres within or across the central fissure, or whether there is no exact parallelism between the anatomical boundaries of the excitable and non-excitable areas in the lower and higher apes is a question which may be argued, but the weight of evidence is in favour of restricting the motor cortex in man to the region in front of the fissure of Rolando. Several observations have been made in man as to the effects of electrical stimulation of the Rolandic area—by Mills, Keen, Lloyd and Deaver, Nancrede, Ransom, Dana, Horsley and Beevor, Lloyd, etc. Mills and Keen obtained movements from the precentral convolution; Nancrede and Lloyd and Deaver from the postcentral convolution; but Lloyd, in his most recent contribution to this subject, found on using the unipolar method that only the precentral gyrus gave movements on stimulation, stimulation of the postcentral being entirely negative. Beevor and Horsley found also only the precentral convolution excitable in a case of operation for the relief of focal epilepsy.

It is also of considerable importance that there are notable differences in structure between the precentral and postcentral gyrus, as has been pointed out by Cajal, Campbell, and Brodmann. According to Dr. Campbell, the cortex of the precentral gyrus is characterised by a great wealth of nerve-fibres, and the same area corresponds approximately to the distribution of the giant-cells of Betz and Bevan Lewis. The postcentral cortex is not so rich in fibres as the precentral; it contains an inter-radiary plexus of fibres of large calibre; it has no Betz-cells, but a pronounced layer of stellate cells, and differs from the rest of the parietal region in containing pyramidal cells of larger dimensions. The giant or Betz-cells would seem to be the centres of origin of the pyramidal tracts, as they disappear—by "reaction at a distance"—on section of the pyramidal tracts of the spinal cord (Holmes and May), and in amyotrophic lateral sclerosis (A. W. Campbell).

It is stated also by Profs. Grünbaum and Sherrington that there is no secondary descending degeneration in the pyramidal tracts after lesions strictly confined to the postcentral cortex in the chimpanzee. In the macaque, however, Mellus has described secondary degeneration from lesion of the thumb centre at the lower extremity of the postcentral gyrus.

The results of the excitation method shew that whether the individual segments of a limb are separately localised, or are represented more or less throughout a common area, the areas as a whole are completely differentiated from each other. Thus, no movements of the leg can be excited from irritation of the facial centre, nor of the face from the leg centre.

It has been established, by experiments on monkeys, that destruction of the areas, excitation of which produces definite movements, causes paralysis of the same movements on the opposite side of the body, which varies in extent and completeness with the destruction of the respective centres. When the destruction is complete, the paralysis is permanent, and is followed by degeneration of the pyramidal tracts of the spinal cord and contracture of the paralysed limbs (25). Some movements, however, are so bilaterally represented in each cerebral hemisphere that even complete extirpation of the centres in the one hemisphere causes little or no obvious impairment of these movements; and, in order that paralysis should result, it is necessary that the centres on both sides should be destroyed. Of this character are, first, those of the trunk, hip, and shoulder-girdle, which have their centres in each marginal convolution; hence in order that these movements may be paralysed it is necessary that the marginal convolution should be destroyed in both hemispheres; secondly, the movements of the mouth and tongue; and, thirdly, those of the larynx.

Unilateral extirpation of the oro-lingual or of the laryngeal centres, at the lower extremity of the Rolandic area, does not appreciably impair the movements of articulation or of phonation; whereas they become completely paralysed when the centres are destroyed in both hemispheres. It would appear that such movements as may not be primarily represented bilaterally in each hemisphere are secondarily associated, according to Broadbent's hypothesis, by commissural fibres between the respective bulbar and spinal nuclei. That the bilateral representation of movements in the cerebral hemispheres is founded not merely on functional but on structural relations, is proved by the fact that secondary degeneration in the pyramidal tracts occurs on both sides in accordance with the degree of representation in each hemisphere. Thus, according to the researches of Prof. Sherrington, if the lesions affect the centres for the limbs only, the pyramidal degeneration is almost entirely on the opposite side; whereas it is decidedly bilateral if the lesion invade the marginal convolution: and this is still more pronounced if the lesion invade the laryngeal centre.

These facts shew that, even in the case of the limbs, each hemisphere represents both sides of the body, mainly the opposite, but also to some extent the same side; and this is in accordance with the observations of Brown-Séquard, Pitres, and Friedländer—that lesions which cause hemiplegia of the opposite side cause also some diminution in the dynamic energy of the limbs on the same side.

The bilateral relations of each hemisphere, so well marked in monkeys and man, are still more pronounced in some of the lower animals; and it is this bilateral representation which accounts for a large amount of recovery, even when the motor centres of one hemisphere have been entirely destroyed. The recovery extends more particularly to those movements which are more or less habitually associated with those of the other side, and least of all to those movements which are most independent and most highly specialised. Hence, in cortical motor paralysis the arm is more paralysed than the leg, and the distal movements of the limb more than the proximal.

Cortical motor paralysis is essentially paralysis of voluntary movement, or that which implies conscious discrimination; and is consistent with the retention of co-ordinated movements of the same muscular groups of considerable complexity of the automatic or instinctive order. The degree in which these are retained, after extirpation of the motor cortex, varies much in different orders of animals; they are highest in fishes and birds, and lowest in monkey and man.

Lesions invading the Rolandic area are divisible into two great groups; first, *irritative* lesions, and, secondly, *destructive* lesions. Practically, however, they cannot be separated from each other by hard and fast lines, for not infrequently those lesions, which ultimately prove destructive of the centres which they invade, are primarily, or at some time in their course, associated with irritative phenomena.

Irritative Lesions.—Irritative lesions are so called because they tend to induce a sudden or explosive discharge similar to that which may be induced artificially by long-continued or repeated electrical stimulation of the cortex. The discharge is apt to leave a state of exhaustion, so that a paralytic condition, more or less enduring, may be observed to follow the epileptiform convulsion. This may be a post-epileptiform hemiplegia or a post-epileptiform monoplegia, according as the convulsion has been general or limited. As the pathology of unilateral epileptiform convulsions was first clearly indicated by Dr. Hughlings Jackson, the affection has been appropriately termed "Jacksonian epilepsy."

Jacksonian epilepsy may be taken in all cases to indicate the existence of some irritative lesion of the cortical motor centres. The mere occurrence of a unilateral convulsion, or of an epileptic attack in which the convulsions are exhibited mainly on one side, does not, however, prove, apart from other indications, that the lesion is organic; or, if organic, that it is situated in the cortical motor zone. I have found experimentally, and it is also in accordance with the facts of human pathology, that irritation primarily situated outside the motor zone may result in an attack of unilateral convulsions. But in these cases there is no doubt that the convulsions are proximately due to irritation secondarily set up in the cortical matter of the Rolandic area; since convulsions of the type known as Jacksonian epilepsy—that is, tonic followed by clonic spasms on the opposite side of the body—never occur on stimulation of the medullary fibres alone, and cannot be produced if the cortical motor centres are destroyed on both sides. Irritation of the subjacent medullary fibres causes tonic contractions only, and the duration of the effect is strictly proportional to that of the stimulus which is applied.

In the great majority of instances the lesions which cause Jacksonian epilepsy are situated either in the cortex itself or immediately subjacent. There is reason to believe, however, that an irritable or explosive condition of the cortex manifesting itself in unilateral spasms, monoplegic or hemiplegic, may be set up by lesions primarily situated in the periphery. Thus, Mills has recorded a case of epilepsy of the Jacksonian type in connexion with a neuroma of the right hand, the result of injury. The spasms affected the right side, beginning with the fingers, and extending to the arm, face, and leg without loss of consciousness. Convulsions of a similar peripheral origin have been produced by Brown-Séquard by lesions of the sciatic nerve in guinea-pigs. It is also worthy of note that unilateral convulsions may be caused on the same side by irritation of the dura mater. This I have frequently seen in the course of my experiments.

The morbid anatomy of Jacksonian epilepsy is variable, but, from the point of view of regional diagnosis, the exact character of the lesions is unimportant. Generally, it may be said, they are such as are calculated to produce an inflammatory or hyperaemic condition of the cortical centres. They include exostoses, depressed fractures, spicula of bone and cicatrices, resulting from traumatic lesions, meningeal irritation, tuberculous and other tumours, and especially syphilitic lesions of the cortex. These last are such a frequent and potent cause of Jacksonian epilepsy that syphilitic epilepsy and Jacksonian epilepsy are sometimes used interchangeably, though the one is by no means coextensive with the other.

As has been remarked before, the disease, which begins as an irritative lesion, generally tends to destroy the region in which it is situated, and thus leads to paralysis with secondary degeneration; irritative phenomena being prone to occur so long as the vitality of the grey matter is not entirely exhausted. It is this tendency which enables us to arrive at the regional diagnosis of the seat of the lesion in a more precise manner than if the phenomena were of the purely irritative order; for the determination of the seat of an irritative lesion is more uncertain than that of a destructive lesion, owing to the difficulty of determining the exact extent of the zone through which a vital irritation may diffuse itself.

If, however, the convulsive discharge be associated with, or followed by, paralytic weakness in the face, arm, or leg, the position of the lesion can be more accurately determined, according to the principles which will be laid down more fully in the next section.

Jacksonian epilepsy in its early stages, and often throughout its whole course, is limited to the face, arm, or leg; or to some particular muscular group on the opposite side of the body. With this monospasm or protospasm there is often a tendency to generalisation, and the convulsions usually follow a definite order. If the spasms begin in the face, they next attack the arm, and then the leg; if they begin in the hand, they ascend the arm, then attack the face, and ultimately spread to the leg; if they begin in the leg, they next affect the arm, and lastly the face. This order is rarely inverted, and it never happens that a spasm, beginning in the face, attacks the leg before affecting the arm. The explanation of the sequence of the spasms in Jacksonian epilepsy is afforded by the position in the Rolandic area of the respective centres of the face, arm, and leg. In order that irritation starting from the facial area should ascend to the leg area, it is necessary that it should diffuse itself through the arm area which separates them from each other. Usually, when the convulsions, starting in a given point, have become unilateral, consciousness is lost; and in this case the convulsions generally extend to the other side, and become bilateral.

Three types of Jacksonian epilepsy may be conceived, according as the spasms begin in the face, arm, or leg.

First, Facial Monospasm or Protospasm.—In this case the spasms usually begin in the angle of the mouth, spread thence to the head and neck, causing rotation and conjugate deviation to the opposite side, and thence tend to spread to the arm and become general. Under such conditions we may assume the existence of an irritative lesion of the facial centre situated at the lower extremity of the Rolandic area. It is not necessary here to refer in detail to the evidence in support of this proposition. The chief facts bearing on this subject may be found on reference to Charcot and Pitres, Grasset, de Boyer, and my *Localisation of Cerebral Disease*.

In an interesting case reported by Dr. Byrom Bramwell the fits began with spasm in the right platysma, and were frequently confined to this muscle. The lesion in this case was a small spiculum of bone projecting from the inner table of the skull, the result of a cranial injury received some years previously, which impinged on the cortex at the inferior extremity of the ascending parietal convolution. The situation of the lesion exactly corresponded to the position of the centre for the platysma, as defined by electrical irritation of the brain of the macaque.

Secondly, Brachial Monospasm or Protospasm.—Of cases of spasms, dependent on cortical lesion, beginning in the hand and arm, or limited to it, there are now many on record. As a rule they begin in the fingers, and more especially in the thumb and index-finger; but this is not necessarily so, as much depends upon the exact position of the lesion in the arm area.

As has already been noted, the proximal movements of the arm are represented in the upper part of the arm area, and the distal in the lower. In some recorded cases no exact description is given of the march of the spasms; but in others, in which they specially began in the fingers or hand, the lesion has been found either in the ascending parietal convolution, or in close proximity to it.

One of the most interesting cases of this kind has been reported by Dreschfeld. The patient suffered from repeated attacks of convulsions limited to the left arm, beginning with closure of the fist, flexion of the wrist and pronation of the forearm, together with spasms of the left platysma. The dura mater was found adherent to the cortex on the right side over the greater part of the ascending parietal convolution and supramarginal lobule. In a case under the care of Dr. Hughlings Jackson, reported by Sir V. Horsley (41), the patient was subject to spasms beginning in the left thumb and forefinger, and frequently followed by temporary paralysis of the left arm. The spasms depended upon the existence of a small tumour situated at the junction of the lower and middle thirds of the posterior central convolution in the right hemisphere. The lesion was accurately diagnosed from the symptoms, exposed, and removed. In those cases in which the spasms have begun in the upper arm and descended to the hand, the lesion has been found at the upper part of the arm area. A typical case of this kind has been recorded by Dr. Hughlings Jackson.

In these and similar facts we have reason to believe that the exact situation of the lesion in the arm area may be determined by careful observation of the mode in which the attacks set in; and that, in accordance with electrical stimulation, the spasms will begin in the proximal or distal part of the limb, according as the lesion is situated in the upper or lower division of the arm area.

Thirdly, Crural Monospasm or Protospasm.—In the third group of cases the fits begin in the lower extremity, and may start in the foot and ascend to the thigh, or in the muscles of the thigh and descend to the foot. In such cases the lesion is found at the upper extremity of the Rolandic fissure, as in a case reported by Dr. Hughlings Jackson. In another reported by the same author the fits began in the right leg, and were frequently limited to it, followed in time by paralysis of this limb and other signs of extension to the hemisphere in general. A tumour was found at the upper extremity of the ascending frontal and base of the superior and middle frontal convolutions. Many similar instances might be mentioned. For further details the reader is referred to my Goulstonian and Croonian lectures.

The monospasms or protospasms of Jacksonian epilepsy are not infrequently preceded, or accompanied, by a subjective sensation or sensory aura in the parts invaded by the spasm. It may be limited to these, or the sensory discharge may be more extensive. Thus, in a case reported by Ransom, the fits began with tingling in the left thumb, followed by spasms which spread up the arm to the face. Occasionally the tingling sensation occurred alone, without spasm; and in addition, at a later date, there was also a visual aura in the left eye. The lesion in this case was an adhesion of the pia mater to the cortex at the junction of the middle with the lower third of the Rolandic area of the right hemisphere.

Confirmatory evidence of the regional diagnosis founded on the symptomatology may in many cases be obtained by deep pressure on the skull over the suspected seat of the lesion. Tenderness or distinct pain may thus be elicited, even though the patient may not have complained spontaneously of pain in this region. This is particularly true of syphilitic and irritative lesions in general. In the case of lesions, however, which do not actually reach the cortex, this symptom may be entirely absent.

Summary.—(i.) Irritative lesions of the Rolandic area are indicated by epileptiform convulsions on the opposite side of the body, which may be limited to the arm, leg, or face; or, beginning in this way, may become general, with or without loss of consciousness.

(ii.) There need not be any discoverable organic lesion; but if, in addition to the muscular spasms, there is paralysis more or less enduring, and tending to become permanent, we may diagnose an organic lesion.

(iii.) The nature of the lesion, apart from external indications, must be determined by the characters peculiar to each, such as tumour, syphilis, and the like; and its exact seat by its limitation or mode of onset in the face, arm, or leg respectively.

(iv.) If in the face, the lesion is situated in the lower third of the Rolandic area, or in close proximity to it. If in the arm, in the middle third; if in the leg, at the upper third of the Rolandic area: and it may be more exactly defined in these respective areas according to the muscles primarily thrown into spasm.

(v.) Confirmatory of the evidence derived from the mode of onset of the convulsions is the fact that pain may frequently be elicited by deep pressure over the corresponding cranio-topographical area.

From these indications the seat of the disease may as a rule be accurately defined with a view to excision of an epileptogenic focus.

Destructive Lesions.—A. *Affections of Motion.*—Destructive lesions of the Rolandic area are the counterpart of irritative lesions, and manifest themselves in paralysis of voluntary motion, limited or general, according as the lesion invades special areas or the whole Rolandic region. This is true of all lesions which positively destroy the cortical grey matter. The apparent exceptions to this rule have been cases of superficial lesion, or tumours growing in or on the cortex, which, it is well known, may in the course of their growth gradually push aside, without actually destroying, the true nerve structures in which they have their seat. Cases of this kind have been reported by Samt, Magnan, Bramwell (12), Bennett, and others. Or the lesions may have caused a certain amount of destruction, but either the symptoms during life have not been carefully observed, or the lesions have occurred before the brain had reached its full development, thus allowing of a greater degree of compensation by the motor centres of the other hemisphere, or by the subjacent ganglia. In this group may be classed the case recorded by Cunningham, and cases of porencephaly, or infantile hemiplegia, in many of which the paralysis is less in degree and extent than is usually found as the result of similar lesions in adults.

Hemiplegia of the cortical type, due to destruction of the whole Rolandic area, does not differ essentially from hemiplegia due to destruction of the anterior part of the posterior limb of the internal capsule (see p. 103). There is the same relative affection of the different movements, those being most paralysed which are most volitional, and least associated with those of the other side of the body. The facial paralysis is most marked in the lower facial region, and less so, but still evident on careful examination, in the upper facial region. The movements of the leg are less paralysed than those of the arm, and the proximal movements of the arm less than those of the wrist and fingers; whilst those of the trunk, articulation, and phonation are not materially affected. *Voluntary* respiratory movement is less marked on the paralysed side, but *natural* or reflex respiratory action is on the contrary rather increased (Hughlings Jackson).

If the destruction of the cortical substance be complete, the paralysis will be of permanent duration, and followed sooner or later by secondary contracture of the paralysed limbs, with descending sclerosis of the pyramidal tracts. If the lesion is not such as to destroy the grey matter completely, the hemiplegia may be transitory, as is not infrequently seen in the course of general paralysis, or of superficial meningo-encephalitis. A complete hemiplegia, however, of considerable duration does not necessarily indicate total destruction of the whole motor area, for the lesion may have anatomical limits, whereas the paralysis on the opposite side is general. In such cases we have to deal with merely dynamic disturbances, extending beyond the actually demonstrable lesion; and in the course of time the parts which have not been destroyed may regain their functions, and the paralysis become restricted.

Though the rule is that the paralysis occurs on the opposite side of the body, there is no doubt that in some rare instances the paralysis has been direct, that is, on the same side as the lesion. These cases, however, are by no means so numerous as Brown-Séquard has represented, for a large proportion may without doubt be set down to faulty records. But that they may occur has been proved conclusively by a case, carefully observed and recorded by Charcot and Pitres (15), of left hemiplegia with aphasia occurring suddenly without loss of consciousness, and without affection of sensation. The paralysis and aphasia existed a whole year. After death the right hemisphere was found intact. In the left hemisphere a large area of softening was found extending from the ascending limb of the Sylvian fissure into the lower part of the ascending frontal, the corresponding portion of the ascending parietal, and the base of the second frontal convolutions. The lesion was limited below by the horizontal limb of the fissure of Sylvius; and sections through the softened region shewed that the lesion involved only the cortex and subjacent medullary fibres, and did not invade the internal capsule or basal ganglia. Secondary degeneration was traced into the left crus and the left pyramid. In this case there was no decussation of the anterior pyramidal tracts in the usual situation. Under such conditions a direct paralysis is easily understood and accounted for.

Complete hemiplegia, due to a destructive lesion of the Rolandic area, does not, as has been said above, differ essentially from that caused by lesion of the corresponding part of the internal capsule. This statement, however, requires qualification in respect to the left hemisphere. In this case, in the vast majority of instances, the paralysis of the limbs and face is associated with aphasia—an association which at once distinguishes cortical from capsular right hemiplegia.

Hemiplegia of the ordinary kind, however, is not the most common form of cortical paralysis, for only rarely can the whole motor cortex be simultaneously destroyed by any pathological or traumatic lesion. Necrotic softening, from vascular occlusion of the middle cerebral artery, still leaves intact the upper part of the Rolandic area supplied by the anterior cerebral; and a simultaneous blocking of these two channels, though conceivable, is practically unknown. Hence in the completest form of cortical hemiplegia from vascular occlusion the lower extremity is comparatively little affected. More often complete hemiplegia of cortical origin is a succession of monoplegias, due to extension of a lesion beginning in some definite area; and we may diagnose the primary seat of the lesion by a consideration of the movements which were first paralysed and the mode in which others were successively abolished.

Crural Monoplegia.—If the paralysis is limited to the leg, or begins in it, the primary seat of the lesion is the paracentral lobule or upper extremity of the central convolutions adjoining the longitudinal fissure. In this form of monoplegia—crural monoplegia—of which there are now many cases on record, traumatic and otherwise, the lesion has been found in the region indicated. This region appears to be a favourite seat of circumscribed tuberculous disease. (On this, *vide* case reported by Souques and Charcot; a similar one by Ballet; and another by myself (28).)

The frequency of tuberculous disease in this region is attributed by Souques and Charcot to the relative richness of its circulation, as it derives blood from both the Sylvian and anterior cerebral arteries. This also is the region more particularly involved in birth palsy, or congenital spastic paraplegia due to traumatic meningeal haemorrhage (M'Nutt). Crural paralysis is very frequently associated with a greater or less degree of paralysis of the arm also, constituting the mixed type, *brachio-crural monoplegia.* In at least ten cases of crural monoplegia, which I have found on record, the lesion occupied the paracentral lobule, and the adjoining convex aspect of the hemisphere at the upper third of the Rolandic zone. And in many of the combined cases the progress of the disease from above downwards was distinctly traced by the course of a paralysis beginning in the leg and gradually extending to the arm.

Brachial Monoplegia.—Paralysis limited to the upper extremity or to some of the movements of this limb, constituting brachial monoplegia, is of very common occurrence. The lesions, with some variation in their extent, have been found to implicate the middle two-fourths of the precentral gyrus, and the evidence is in favour of the lower portion being especially related to the finer movements of the hand and wrist. It is necessary, however, in this connexion to remember that a lesion primarily situated outside the motor area and gradually invading it, such as a tumour, is apt first of all to cause weakness of the wrist and fingers—an illustration of the general principle that the finer or more specialised movements are the first to give way before an invading or destroying lesion. Many of the cases of brachial monoplegia on record have been the result of excision of a supposed focus of Jacksonian epilepsy; some of these cases will be referred to in connexion with another problem (p. 68).

Brachial monoplegia is frequently associated with paralysis of the face of the ordinary cerebral type, constituting the combined form, *brachio-facial monoplegia*; an association specially apt to occur from occlusion of the Sylvian artery, and as a rule associated with aphasia when the lesion is in the left hemisphere. This associated paralysis is owing to the implication of the facial with the brachial centres; and in at least twenty cases of this kind, which I have investigated, the lesion was found to invade the lower half of the Rolandic zone. These cases are so common that it is unnecessary to refer to them in detail. If paralysis of this form on the right side is not associated with aphasia the probability is that the patient is left-handed; or that the lesion is not cortical, as in a case reported by Dieulafoy.

Facial Monoplegia.—Facial paralysis of the cerebral type—facial monoplegia—that is, paralysis limited to the lower facial region, uncomplicated with paralysis of the arm, is not a very common occurrence. Usually it is associated with some affection of the tongue and muscles of articulation. One of the earliest cases of cortical facial paralysis on record has been reported by Hitzig. This was the case of a soldier who, after a bullet wound on the right side of the head, began to have spasm in the left side of the face and tongue, and to a slight extent also of the left hand. After death an abscess was found at the lower extremity of the right ascending frontal convolution, between the precentral sulcus and fissure of Rolando. In an interesting case of left facial spasm of the Jacksonian type, under my own care, operated upon by Sir V. Horsley, excision of the lower extremity of the right ascending frontal convolution caused weakness of the left side of the face and defective articulation.

The lowermost extremity of the ascending frontal convolution and base of the third frontal is the region in which stimulation causes movements of the tongue, of the muscles of articulation, and also of the larynx. Physiological experiments shew that these centres have bilateral relations, stimulation causing movements of both sides. Hence it is that unilateral lesion causes only slight or almost imperceptible weakness on the one side. This fact explains also why the lesion which causes motor aphasia does not imply paralysis of articulation or phonation.

It is not necessary, in this section, to discuss the pathology of aphasia further than to state that the aphasia here referred to is Broca's or motor aphasia; that is, the inability to express ideas in articulate speech, while ideation in general, and the comprehension of speech spoken and written, remain unimpaired. Usually the lesion causing aphasia is vascular obstruction of the left middle cerebral artery; and it has been supposed by some that the greater frequency of aphasia with right than with left hemiplegia is due to the greater frequency of vascular occlusion in the left hemisphere, as compared with the right. I have elsewhere (25) shewn that this surmise is completely disposed of by the reported cases of aphasia from traumatic lesions at the lower extremity of the Rolandic zone. A case reported by Simon has almost the character of a precise experimental lesion. A healthy man was seen to fall from his horse, and shortly endeavour to remount. A doctor who was of the party found him quite aphasic, and able only to make signs and gestures. He ultimately could utter certain words, *e.g.* "Wasser," which he used for all objects. He gradually developed slight paralysis of the limbs, and died comatose three weeks after the accident. After death a depressed fracture nearly circular in form was seen, and corresponding in situation with this a piece of bone was found embedded in the left third frontal convolution. This and the neighbouring parts were softened and contained numerous extravasations. For references to other instances see Sidney Jones, Proust and Terillon, MacCormac, Macewen.

As already stated, in cases of motor aphasia there is no paralysis of the muscles of articulation, but at most a slight weakness of the right angle of the mouth and slight deviation of the tongue to the right. If, however, the lesion is bilateral, not merely is there aphasia, but also paralysis of the muscles of the tongue and articulation. The best instance of this kind on record is that reported by Sir T. Barlow.

A boy aged ten, the subject of aortic disease, of which he ultimately died, was seized with right hemiplegia (chiefly brachio-facial) and aphasia. From this he recovered at the end of a month. Three months later he was seized with left brachio-facial monoplegia. With this attack there occurred aphasia also, together with paralysis of the movements of the mouth and tongue. He was unable to shew his teeth, or to protrude his tongue; and there appeared, in the words of Sir T. Barlow, "to be loss of voluntary motor power over the muscles concerned in deglutition and articulation; reflex deglutition, however, was unimpaired. Mastication was difficult, but not entirely paralysed, and the patient was able to phonate. There was no affection of sensation in the paralysed parts, either in the skin or mucous membranes, and the muscles reacted normally to the faradic current." The condition lasted till death. After death a patch of softening was found in each hemisphere, occupying the lower end of the ascending frontal, and the hinder ends of the middle and inferior frontal convolutions.

The condition existing in this case is in many respects like that met with in bulbar paralysis, though there are some essential points of difference which will be discussed on p. 106, in connexion with a similar group of symptoms termed "pseudo-bulbar" paralysis, to distinguish it from that due to nuclear lesion of the medulla oblongata.

As has been mentioned, stimulation of the lower extremity of the ascending frontal convolution causes phonatory movements of the vocal cords. The effect here is distinctly bilateral; it is not to be expected, therefore, that unilateral lesion should cause any appreciable affection of the movements of the vocal cords. It seems somewhat strange, however, that in Barlow's case phonation was still possible, notwithstanding the apparent total destruction of the region in which the laryngeal movements are centralised. If the centres in question were entirely destroyed, we must assume that, as in the case of the oculomotor centres, compensation may be effected by the basal ganglia. Some cases have, however, been reported in which unilateral cortical lesion in the region indicated was associated with paralysis of the opposite vocal cord. In one of these, by Seguin, there was, apparently in connexion with lesion of the posterior part of the third frontal convolution on the right side, some affection of phonation; and in another, by Garel and Dor, there was paralysis of the left vocal cord, apparently in connexion with a lesion of the right hemisphere at the base of the third frontal convolution. The cases, however, were not verified by laryngoscopic examination, nor was the possibility of a bulbar or peripheral nerve lesion disproved; and it is exceedingly improbable, in view of the facts of Barlow's case, together with those of experimental physiology, that paralysis of the vocal cord can be produced by a unilateral cortical lesion. Moreover, Sir F. Semon has shewn, by laryngoscopic examination of cases of right hemiplegia and aphasia within a few hours of the attack, that both vocal cords move in a perfectly normal manner.

Whilst, therefore, it is probable that the laryngeal centre is situated at the lower extremity of the ascending frontal convolution, there is no satisfactory evidence on record that complete paralysis of one or both cords has been caused by cortical lesion.

The same may be said of the muscles of mastication. Movements of the same muscles are also concerned in the act of articulation, and these are excited bilaterally by stimulation of the cortex. Paralysis of these muscles cannot, therefore, be expected from anything less than a bilateral lesion in corresponding parts. In Sir T. Barlow's case, however, though the movements of mastication were excessively feeble, they were not entirely paralysed.

Lépine, Langer, and Picot (quoted by Charcot and Pitres) have observed trismus in connexion with cortical lesions; but the pathology of this symptom, with its bearing on the position of the masticatory centre, has not been established. A case has been reported, by Hirt, in which masticatory paralysis was apparently due to cortical lesion; but the case was a complicated one, for, in addition to multiple cortical lesions, there was degeneration in the spinal cord also.

We may feel assured that every movement under the control of the will is represented unilaterally or bilaterally in the motor area; but the clinical evidence at present available does not enable us to indicate the centres of any others beyond those already discussed.

B. *Affections of Sensation.*—Cortical paralysis due to destructive lesion of the motor area is essentially a motor paralysis, and need not be accompanied by loss of sensation, superficial or deep.

The patient whose limbs are entirely paralysed from cortical disease may feel and localise the slightest touch, appreciate pain and temperature, and correctly indicate every movement communicated to his paralysed limbs. Thus, in a case of aphasia and right hemiplegia under my care, the patient, whose right arm, a month after the onset, was still absolutely paralysed, felt and localised the slightest touch on the paralysed limb, and correctly indicated with the left hand every position passively communicated to the right. After death necrotic softening, due to blocking of the Sylvian artery, was found in the Rolandic area of the left hemisphere. In another case recently under my care of Jacksonian epilepsy, and gradually increasing hemiplegia of the right side, a tumour was removed which was found invading the upper half of the precentral gyrus. The operation was followed by complete hemiplegia, permanent in the foot but diminishing in the hand. Within a few days of the operation the patient, though completely hemiplegic as to motion, could feel and accurately localise a touch on his paralysed limbs, and could indicate with his left limbs the position and movements passively communicated to his right, and also distinguish the form of objects placed under his paralysed fingers, such as a coin, a match-box, a pencil-case. There was no astereognosis. But though, as the above cases prove, paralysis of cortical origin may occur without the slightest affection of sensation, it is undoubtedly true that affections of sensation of a greater or less extent are met with in connexion with lesions apparently confined to the cortex of the Rolandic zone. And it is a question on which there is still considerable difference of opinion, whether sensation is not also represented, along with motion, in what is usually designated the motor area of the cortex.

An analysis of 284 recorded cases of lesion of the Rolandic zone, which I made in 1890 (*Croonian Lectures on Cerebral Localisation*), shewed that in 100 the state of the sensibility was not mentioned; in 121 it was noted to be intact, and in many of these every variety of sensibility was expressly stated to have been carefully investigated. In 63 some impairment of sensibility was noted; in 28 of these, however, the lesion was not strictly confined to the Rolandic area, but implicated the adjacent lobes, especially the parietal. Among the remaining 35, conditions existed, in the majority at any rate, which were calculated to implicate centres or tracts demonstrably connected with the conveyance or perception of sensory impressions. Similar conclusions were arrived at by Charcot in the work (14) on which he was engaged with Pitres just before his death:—"In our opinion the anaesthesias which sometimes accompany motor paralysis of cortical origin are most frequently functional, and are analogous to, if not identical with, hysterical anaesthesia. The discussion of the reasons which appear to us to justify this opinion would take us far beyond the limits which we have set ourselves in this chapter. We content ourselves, therefore, with stating that, from an examination of the facts of clinical pathology, the anaesthesias which sometimes accompany paralysis of cortical origin are unessential and accidental phenomena, and do not depend directly on lesions of the Rolandic zone, and play no pathogenic part in the development of paralytic symptoms."

Nothnagel, writing in 1879, arrived at the conclusion that "up to the present there is no case in which, with a purely superficial or purely cortical lesion, persistent anaesthesia has been observed. In all cases the medullary substance has also been affected, and it is not improbable that in such cases the sensory tracts of the internal capsule may have been injured directly or indirectly. For the diagnosis, therefore, of superficial lesions, disturbances of sensation have no importance."

On the other hand, many writers—Petrina, Exner, Luciani and Seppilli, Starr (73), Dana (17), and others—maintain that the facts of disease prove that the sensory centres coincide anatomically with the motor centres in the Rolandic zone. And as this view is largely entertained by clinical observers, it appears necessary to consider somewhat in detail the facts on which it is based. As already stated, there is no doubt that in many cases of lesion of the Rolandic zone, anaesthesia has been observed coinciding to a greater or less extent with the motor paralysis. The question, however, is whether the affection of sensation is a direct result of the cortical lesion, or only an indirect or dynamic influence on sensory centres anatomically distinct from the motor centres proper. In this relation tumours, and all such lesions as are known to cause indefinite indirect effects, are of no value as evidence, and may be summarily excluded. Since attention has been specially directed to this point, many cases of excision of portions of the motor cortex for the cure of focal or Jacksonian epilepsy have been put on record, and it is of importance to consider their bearing on the question. In the first edition of this work (1899, Vol. VII. pp. 298-301), a number of cases were detailed (v. Bergmann, Keen, Lloyd and Deaver, Hale White, Bidwell and Sherrington, Diller and Buchanan); analysis of which shews that there are some variations in the condition of sensation after cortical lesions of the Rolandic area. In the majority of pure cortical lesions there was not the slightest affection of sensibility in any of its forms from the first, though there was marked motor paralysis; whilst in some the slight affection of sensation which was at first observable ultimately disappeared. This was true of Beevor and Ballance's case, in which, notwithstanding the permanent paralysis of the hand and foot, sensibility completely returned. In Starr's case the persistence of defective tactile and painful sensibility at the tips of the fingers might be ascribed to the rigid flexion of the fingers; and in others, such as Albertoni and Brigatti's—as the lesions were for the removal of tumours extending deeply into the brain substance—there was at least the possibility of injury of the sensory tracts.

It is evident from the recorded cases that, at any rate, relatively small portions of the convexity of the Rolandic zone may be removed without causing the slightest impairment of tactile or cutaneous sensibility in any of its forms, though the lesion may be sufficient to produce more or less extensive paralysis. It is probable, therefore, that such affections of sensation as may occur from destruction of a larger area are of a merely dynamical character. If there were any necessary relation between the sensory defect and the motor paralysis the two should coexist and be equally enduring, but this is not the case; for the defect of sensibility, at first observable, gradually lessens and ultimately disappears.

The order in which sensibility normally returns in cerebral hemianaesthesia explains the apparent limitation of the sensory defect to the limb most paralysed. The affection of tactile and general sensibility follows the same rule as the distribution of muscular paralysis ; that is, it is most marked and endures longest in the parts most volitional. Hence when cerebral hemianaesthesia is beginning it shews itself first in the fingers, and disappears last from this part. A certain degree of sensory defect may therefore be observed in the hand, when the face, trunk, and leg have entirely recovered. The remnant, therefore, of a general hemianaesthesia, when associated with partial hemiplegia or brachial monoplegia, may bring about the coexistence of brachial monoplegia with brachial mono-anaesthesia, apparently from the one cortical lesion, but in reality an effect of complex causation.

Most of those who hold that the motor and sensory centres coincide in the Rolandic area are obliged to admit that they are not coextensive, and to assume that the sensory centre of any given part occupies relatively a much larger area than its motor centre. Hence, for instance, in order to cause anaesthesia of the face, the lesion must be such as to cause also motor paralysis of the arm or leg or more ; that is, that the sensory centre of the face embraces also the motor centres of the arm and leg. Such a hypothesis on the part of those who otherwise advocate a localisation of function in the cerebral cortex appears to me rather absurd.

That clinical and experimental lesions (Schiff, Munk, Tripier, Luciani and Seppilli, Mott, etc.) of the Rolandic area of considerable extent should temporarily cause some impairment of sensation on the paralysed side, may be admitted without the necessity of assuming that the sensory centres are anatomically coincident with the motor.

So far as sensation can be tested in the lower animals, neither Prof. Schäfer nor I have been able to detect any impairment, but, on the contrary, clear evidence of the retention of tactile sensibility on removal of the individual motor centres ; and Prof. Schäfer has removed the whole motor cortex in successive operations, causing complete motor paralysis without impairment of tactile sensibility. Extensive lesions, however, of the motor centres may, as Dr. Turner and I have found, temporarily cause not only impairment of tactile sensibility over the whole of the opposite side of the body, but also homonymous hemianopsy.

In several instances we destroyed the Rolandic area, wholly or partially, in such a way as to remove the cortex in the most thorough manner, both on the convexity of the convolutions and in the depths of the sulci. This was followed in one case by total hemiplegia of the opposite side, great impairment of tactile sensibility, and hemianopsy. The destruction of the Rolandic area did not extend into the facial centre, but the anaesthesia affected the face equally with the parts that were paralysed. In the course of a few days, however, while the motor paralysis continued, the anaesthesia and hemianopsy gradually diminished, the last remnant of anaesthesia being the inability to localise correctly a small ivory clip exerting slight pressure on the skin of the fingers or toes.

In another experiment, in which the upper third of the Rolandic area was entirely destroyed in the left hemisphere, the lesion extending down to the calloso-marginal fissure on the mesial aspect, there was almost total paralysis of the right leg, and to a considerable extent also of the right arm. Together with this motor paralysis there was loss of tactile sensibility, without analgesia, over the whole of the right side—ear, face, arm, and leg. Within three days, however, this had almost entirely disappeared, so that the animal, blindfolded, was evidently aware of drops of water allowed to fall on its toes, as it put its hand at once on the spot.

The same arguments, therefore, which are employed to establish the localisation of the sensory centres in the motor area might be employed in favour of the localisation of the visual centre in the same region. And in a case of removal of a whole frontal lobe previously referred to (p. 43) the same combination of symptoms resulted. We know, however, from other data, that the visual defect can only be of dynamic origin, and the same can be argued for the tactile sensory impairment.

In several instances, especially those of excision of a portion of the Rolandic area, it has been stated that in addition to some impairment of tactile sensibility there has been loss or impairment of the sense of position or movement. On the other hand, cases have been adduced, by Nothnagel, Luciani and Seppilli, Seligmüller and others, of loss of the muscular sense without motor paralysis, more especially in connexion with lesions of the parietal lobe. Redlich, however, shews that the great majority of these have been of such a nature — namely, abscesses or tumours, and so forth—as to exercise an indirect effect upon other parts; and the cases which he himself mentions as having more the character of stationary lesions have all, as he states, been of an extensive character, involving sometimes the upper, sometimes the lower parietal lobe, as well as the angular gyrus and the temporal convolutions or the occipital lobe; and in one case, at least, the loss of the muscular sense was complicated also by hemianopsy. In two of the cases the Rolandic zone was not affected. Redlich states that in 132 cases of hemiplegia, examined by him in the Vienna Hospital, he found 28 associated with more or less enduring impairment of the muscular and stereognostic sense—that is, the faculty of distinguishing form. In all his cases there was some impairment of tactile sensibility also. The impairment of the muscular sense did not, however, run quite parallel to the impairment of tactile sensibility. As a rule the muscular sense was more impaired than the mere perception of tactile impressions; but as regards both the defect was more marked in the limb most paralysed, and more especially in the distal portions of this limb. His observations are in harmony with what has previously been stated, namely, that impairment of tactile sensibility disappears last from the parts which take longest to recover from paralysis after an ordinary attack of hemiplegia. In the great majority of instances in which there was impairment of muscular sense in the hand, tactile sensibility was also impaired over the whole of the corresponding side, as may be assumed from the fact that it was generally impaired in the face. This also is in harmony with my observations, that the impairment of tactile sensibility is often well marked in parts which are not paralysed as to motion. Since special attention has been given to the investigation of the appreciation of form in hemiplegia, Verger has found astereognosis in more than 50 per cent of recent cases, and Walton and Paul in 33 per cent of old-standing ones.

During the past few years increasing attention has been given to the investigation of sensory disturbances in connexion with pathological conditions of the nerves and nerve-centres, and this has been largely due to the important work of Dr. Head on the functions of the peripheral nerves, and on the paths of conduction of the different forms of afferent impulses within the spinal cord. Many of the phenomena of sensation and sensory conduction are such as cannot be satisfactorily tested on the lower animals, and evidence in regard to them must depend on the personal testimony of the individual. It has become necessary, therefore, to re-examine in the light of more modern instances the question of the relation of lesions of the Rolandic area to sensory disturbances, particularly those which lead to astereognosis or inability to judge of the form of objects. Astereognosis has been observed in cases in which there has not been any appreciable impairment of tactile sensibility or even localisation. It is essentially associated with defects in the perception of position and movement and of what Dr. Head terms tactile discrimination or spatial sense, namely, the perception of the space between the points of a pair of compasses. The afferent impressions subserving the sense of movement and the spatial sense are conveyed, as Dr. Head has shewn, apart from all other forms of sensibility, in the posterior columns of the spinal cord on the same side—all the others crossing sooner or later to the opposite side. Reference has already been made to the investigations of Cajal, Campbell, and Brodmann, which indicate noteworthy differences in structure between the precentral and postcentral gyri. And the mesial fillet, which is the direct continuation of the posterior columns of the spinal cord (after their passage through the gracile and cuneate nuclei, and decussation in the inter-olivary layer), ascends to the ventrilateral nucleus of the optic thalamus, and thence, according to the researches of v. Monakow, Tschermak, etc., more particularly to the postcentral gyrus. There is thus a strong body of evidence in favour of the view that the precentral gyrus is the true motor zone, and the postcentral gyrus the kinaesthetic zone of Bastian. If this is so, it would not be difficult to understand that lesions of the Rolandic area might sometimes be associated with motor paralysis only, and at other times with paralysis of motion and also astereognosis, according as the lesions affected the precentral gyrus alone or the two together. The close proximity of the two gyri and community of vascular supply make their conjoint lesion by the processes of disease the rule rather than the exception; and even apparently restricted experimental lesions of the one are not free from the risk of temporary or permanent disturbance of the closely related centre. This seems to me the true explanation of Sir V. Horsley's case (41*a*). Cushing has made important observations on two epileptics whose attacks were ushered in by a sensory aura in the right hand. In both, while in a conscious state, unipolar faradic stimulation of the precentral convolution elicited characteristic motor responses, these being attended by no sensation other than that which accompanies similar movements on stimulation of a peripheral nerve. On the other hand, stimulation of the postcentral convolution gave definite sensory impressions which were likened in the one case to a sensation of numbness, and in the other to definite tactual impulses. These observations bear out the view that the postcentral convolution only is sensory in function. There are comparatively few cases on record of accurately limited purely cortical lesions of the postcentral gyrus; Starr and M'Cosh have reported a case of loss of the muscular sense, apart from tactile anaesthesia in connexion with lesion of the postcentral gyrus. After trephining for a cranio-cerebral injury of the upper part of the left ascending parietal convolution a varicose mass of veins on the cortex was tied and the subjacent tissue probed with a hypodermic needle. The result of this was an ataxic condition of the right arm, and inability to indicate the position passively communicated to it. Stereognosis was not at this time investigated, but it was probably lost. Dr. Purves Stewart relates a case of superficial softening of the middle third of the left postcentral gyrus which began with paroxysms of pain in the right hand, followed six years later by occasional tingling in the index and middle fingers of the right hand. Under observation during some months the patient developed clumsiness of the right hand and astereognosis, with slight weakness of the right side of the face. The right hand gradually became paralysed, and there was also some motor aphasia. An operation was performed and the diseased patch removed. At the time of the operation an opacity of the pia-arachnoid was seen on the posterior extremity of the second and third frontal gyri—otherwise all the rest of the cortex was normal. A week after the operation there was complete astereognosis of the right hand, while tactile sensibility was unimpaired all over the right side. There was total paralysis of the right thumb and fingers and weakness of the wrist, deficient dorsi-flexion of the right foot, and marked weakness of the right angle of the mouth. Five months later there was still weakness of the right lower face, but all the movements of the upper extremity could be carried out. There was not the slightest tactile anaesthesia of the face or limbs, but there was defective joint sense in the digits, and complete astereognosis. The patient died from an intercurrent ailment six months after the operation, and it was found that in addition to the lesion of the postcentral gyrus there was a subcortical band of softening crossing below the precentral gyrus. The importance of this case is the occurrence of astereognosis without affection of tactile sensibility from a lesion essentially of the postcentral gyrus. Dr. S. A. K. Wilson quotes a case of bullet-wound (bullet extracted) of the right upper parietal lobule, in which there was astereognosis without loss of tactile sensibility in the left hand, with occasional erroneous estimation of passive movements of the fingers, particularly of flexor movements. There was no motor paralysis whatsoever, and there was no alteration of deep-pressure pain. Walton and Paul have recorded several instances of astereognosis with preservation or comparatively slight affection of other forms of sensibility in connexion with lesions posterior to the fissure of Rolando. In one (Case 3) of Jacksonian epilepsy, beginning in and followed by paralysis of the left leg, there was loss of spatial and posture sense, with astereognosis of the foot. The patient could not distinguish between a coin and a pencil placed transversely under the foot. Pain, temperature, and contact senses were normal, and there was no loss of sensation in the hand. An operation disclosed a haemorrhagic focus just behind the upper end of the fissure of Rolando on the right side.

Diller has reported a case of traumatic cranio-cerebral lesion of the left parietal region crossing the fissure of Rolando downwards and backwards about its middle. This was followed by left hemiplegia, which completely cleared up five months after the accident. When examined nearly nineteen years afterwards, the patient (who had become tabetic in the interval) exhibited complete astereognosis of the right hand, with loss of the sense of position and marked defect of spatial sense. Contact, pain, and temperature senses were unimpaired. The left hand was perfectly normal. Diller mentions another case which, though without anatomical data, was evidently a specific cortical lesion of the left hemisphere. In this patient there was also complete astereognosis of the right hand, all the other forms of sensibility, except the sense of position and movement and the spatial sense, being unimpaired.

Similar cases of astereognosis from cortical lesions presumably postcentral have been referred to by von Monakow, Mills and Weisenburg, etc.; but they are not all free from complications, and add nothing to what is exemplified by those detailed above.

The lesson taught by these and similar cases seems to be that in old-standing cases of lesion of the Rolandic zone, when all the forms of sensory impairment, which may be ascribed to diaschisis or dynamic disturbances of sensory centres elsewhere situated, have disappeared, there may still remain astereognosis or inability to appreciate the form of objects; and that this defect is in all probability causally related to lesion of the postcentral gyrus and its expansion in the postero-parietal lobule.

Whether there is a differentiation in the kinaesthetic area corresponding to the differentiation of the motor centres is not as yet definitely established. It is not improbable, as has been supposed, that they may face each other on opposite sides of the fissure of Rolando, but hitherto astereognosis has been for the most part observed only in the hand in whatever situation the lesion has been situated in the postcentral region. In the case, however, above quoted from Walton and Paul, astereognosis was specially observed in the foot in relation with the lesion at the upper extremity of the postcentral gyrus. This is in favour of the view that the motor and kinaesthetic centres are situated anteroposteriorly to each other. But many more observations are necessary before this can be regarded as more than merely probable. It is obvious that if astereognosis and other forms of sensory impairment can occur independently of motor paralysis, the view of Dejerine and Long that the cortical motor and sensory projection-fibres are inextricably intermingled is untenable.

Some of the cases of loss of tactile and muscular sensibility, apparently in connexion with cortical lesions of the Rolandic area, have been without doubt cases of hysterical hemianaesthesia. What appears to me an exemplary case of this kind has been adduced by Sir W. Gowers. It is a case reported by Demange, in which there was extensive cortical softening over the whole of one hemisphere. Associated with this was general and special anaesthesia on the opposite side of the body of the usual hysterical type. A similar instance is mentioned by Nothnagel. This was the case of a young man aged twenty-eight, who became suddenly aphasic. At the same time he became completely anaesthetic on the right side, and to a large extent on the left. This condition, however, lasted for a few days only, but the aphasia continued. A lesion was found in the left hemisphere invading Broca's convolution and its neighbourhood. I have recently had under my care a typical instance of hysterical anaesthesia associated with right hemiplegia and epileptiform convulsions of traumatic origin. The patient had been subjected to several trephining operations. Many of his epileptiform attacks have been undoubtedly only of the functional type.

Summary.—(i.) Destructive lesions of the Rolandic area cause paralysis of voluntary motion on the opposite side, general or limited, according to the extent and locality of the lesion. The electrical reactions of the paralysed muscles are not affected. (ii.) When the whole motor cortex is involved the hemiplegia in all respects resembles that caused by destruction of the motor division of the internal capsule. (iii.) Frequently paralysis of cortical origin is dissociated, or consists of a succession of dissociated paralyses or monoplegias. Thus, a monoplegia may become a hemiplegia by advance of the lesion from its primary seat. (iv.) Paralysis of voluntary motion of the leg alone, or of the arm and leg, or of certain movements of the arm, or of the arm and face, or of the face alone, may be looked on as dependent upon lesions of the cortex of the Rolandic zone or of the subjacent medullary fibres. (v.) Hemiplegia from cortical lesion of the left hemisphere is almost invariably associated with aphasia. (vi.) Crural monoplegia, or this combined with a greater or less degree of paralysis of the arm also, indicates a lesion at the upper extremity of the central convolutions. (vii.) Brachial monoplegia indicates a lesion of the middle two-fourths of the Rolandic area. (viii.) Brachio-facial monoplegia indicates a lesion of the lower third of the Rolandic area. (ix.) Facial monoplegia (or this combined with aphasia, if the lesion is in the left hemisphere) indicates a lesion of the lower extremity of the Rolandic zone posterior to the third frontal. (x.) While it is probable that the centres of movement of the vocal cords, and of the muscles of mastication, are also situated at the lower extremity of the Rolandic area, there is as yet no case on record in which these muscles have been paralysed by cortical lesions, unilateral or bilateral. (xi.) Paralysis of cortical origin may be entirely independent of impairment of cutaneous or muscular sensibility. Not infrequently, however, and especially in connexion with lesions of an extensive and sudden character, or of such a nature as to produce dynamical disturbance of the sensory tracts and centres, it is associated for a time with impairment of sensibility in the paralysed limbs. This rarely extends to the sense of pain or temperature, and is mostly confined to tactile sensibility, and the sense of position and movement. These disturbances are, however, essentially fugacious; and, although at first general, they tend to become restricted to the distal extremities of the paralysed limbs, and ultimately to disappear, even though the motor paralysis remains. (xii.) But in a considerable percentage of cases, and apparently when the lesion invades the postcentral gyrus, cortical hemiplegia or monoplegia is associated with permanent astereognosis of the paralysed limb—specially the hand. When the motor cortex is destroyed, secondary sclerosis ensues in the pyramidal tracts, and contracture in the paralysed limbs.

REFERENCES

1. Albertoni und Brigatti. *Neurol. Centralbl.*, Leipz., 1894, xiii. 221.—2. Ballet. *Arch. de neurol.*, Paris, 1883, v. 283.—3. Barlow. *Brit. Med. Journ.*, 1877, ii. 103.—4. Beevor and Ballance. *Brit. Med. Journ.*, 1895, i. 5.—5. Beevor and Horsley. *Phil. Trans.*, 1890, B. clxxxi. 49.—6. Bennett. *Brit. Med. Journ.*, 1891, i. 528.—7. von Bergmann. *Arch. f. klin. Chir.*, 1887, xxxvi. 864.—8. Bidwell and Sherrington. *Brit. Med. Journ.*, 1893, ii. 988.—9. Bourdon. *Centres moteurs des membres*, 1877, Ob. 2.—10. de Boyer. *Étude typographique sur les lésions corticales*, 1879.—11. Bramwell. *Brit. Med. Journ.*, 1875, ii. 275.—12. *Idem. Edin. Med. Journ.*, 1879.—13. Brown-Séquard. *Lancet*, 1876, i. 2, 79, 159.—14. Charcot et Pitres. *Rev. mens. de méd. et chir.*, Paris, 1877, i. 1.—15. *Idem. Arch. clin. de Bordeaux*, 1894, 43.—16. Cunningham. *Journ. Anat. and Physiol.*, 1879, xiii. 508.—16*a*. Cushing. *Brain*, London, 1909, xxxii. 44.—17. Dana. *Med. Rec.*, N.Y., 1893, xlii. 578.—18. *Idem. Cortical Localisation of Cutaneous Sensations*, 1888.—19. Davy and Bennett. *Brain*, 1887, ix. 74.—20. Dieulafoy. *Bull. Soc. anat.*, 1886, 139.—21. Diller. "Two Cases of Astereognosis," *Brain*, London, 1901, xxiv. 649.—22. Diller and Buchanan. *Amer. Journ. Med. Sc.*, 1893, cvi. 14.—23. Dreschfeld. *Lancet*, 1887, i.—24. Exner. *Localisation der Functionen in der Grosshirnrinde*, 1881.—25. Ferrier. *Localisation of Cerebral Disease*, 1878.—26. *Idem. Cerebral Localisation*, 1890.—27. *Idem. Functions of the Brain*, 2nd ed., 1886.—28. *Idem. Brain*, 1888, iii. 128.—29. *Idem. Brain*, 1888, x. 26, 94.—30. *Ibid.* 29.—31. Friedländer. *Neurol. Centralbl.*, 1883, ii. 241.—32. Garel et Dor. *Ann. des mal. de l'oreille*, 1890, xvi. 209—33. Goldscheider. *Fortschr. d. Med.*, 1895, xiii. 233.—34. Gowers. *Diseases of the Nervous System*, ii. 20.—35. Grasset. *Localisation dans les maladies cérébrales*, 1880.—36. Grünbaum and Sherrington. *Proc. Roy. Soc.*, 69 and 72.—37. Head, Rivers, and Sherren. "The Afferent Nervous System from a New Aspect," *Brain*, 1905, xxviii. 99.—38. Head and Thompson. "Afferent Impulses within the Spinal Cord," *ibid.*, 1906, xxix. 537.—39. Hirt. *Berlin. klin. Wchnschr.*, 1887, xxiv. 488.—40. Hitzig. *Arch. f. Psychiat.*, 1872, iii. 231.—40*a*. Holmes and May. *Brain*, London, 1909, xxxii. i.—41. Horsley. *Brit. Med. Journ.*, 1886, ii. 670.—41*a*. *Idem. Ibid.*, 1909, ii. 125.—42. Horsley and Beevor. *Phil. Trans.*, 1890, B. clxxxi. 49.—43. Jackson, Hughlings. *Medical Mirror*, 1869, vi. 126.—44. Jones, Sidney. *Lancet*, 1873, ii. 449.—45. Keen. *Amer. Journ. Med. Sc.*, 1888, xcvi. 329.—46. *Ibid.*, Case 3.—47. Lloyd. "Faradisation of the Motor Cortex in the Human Brain," *Journ. Amer.*

Med. Assoc., 1906, xlii. 670.—48. Lloyd and Deaver. *Amer. Journ. Med. Sc.*, 1888, xcvi. 564.—49. *Ibid.*, 477.—50. Luciani und Seppilli. *Die Functions-Localisation auf der Grosshirnrinde*, 1886.—51. MacCormac. *Brain*, 1879, i. 26.—52. Macewen. *Lancet*, 1888, ii. 257, Case 8.—53. M'Nutt. *Amer. Journ. Med. Sc.*, 1885, lxxxix. 58.—54. Magnan. *Brain*, 1879, i. 562.—55. Mills. "Cerebral Localisation in its Practical Relations," *ibid.*, 1889, xii. 233.—56. *Idem. Univ. Penna. Med. Bull.*, 1905, xviii. 134.—57. Mills and Keen. *Amer. Journ. Med. Sc.*, 1891, cii. 587.—58. von Monakow. *Gehirnpathologie.*—59. Mott. *Journ. Physiol.*, 1894, xv. 464.—60. Nancrede. *Med. News*, 1888.—61. Nothnagel. *Topische Diagnostik d. Gehirnkrankheiten*, 1879, 473.—62. Parker (Rushton) and Gotch. *Brit. Med. Journ.*, 1893, i. 1101.—63. Petrina. *Ztschr. f. Heilk.*, 1881, ii. 375.—64. Pitres. *Arch. de neurol.*, Paris, 1882, iii. 319.—65. Proust et Terillon. *Bull. Acad. de méd.*, 1876, 2. s., v. 1115.—66. Ransom. *Brain*, 1892, xv. 437.—67. Redlich. "Über Störungen des Muskel-Sinnes bei der cerebral Hemiplegie," *Wien. klin. Wchnschr.*, 1893, vi. 429, 456, 477, 493, 513, 552.—68. Samt. *Arch. f. Psychiat.*, Berl., 1875, v. 209.—69. Schäfer. *Phil. Trans.*, 1888, xx. 303.—70. Seguin. *Trans. Amer. Neurol. Ass.*, 1877.—71. Semon. *Brain*, 1892, xv. 500.—72. Sherrington. *Journ. Physiol.*, 1890, xi. 121, 399.—73. Simon. *Berlin. klin. Wchnschr.*, 1871, viii. 537, 549, 586, 597.—74. Souques et Charcot. *Bull. Soc. anat.*, Paris, 1891, v. 279.—75. Starr. *Amer. Journ. Med. Sc.*, 1884, lxxxvii. 366 ; lxxxviii. 114.—76. Starr and M'Burney. *Brain*, 1891, xiv. 284.—77. Starr and M'Cosh. *Amer. Journ. Med. Sc.*, Phila., 1894, cviii. 517.—78. Stewart, Purves. *Rev. Neurol. and Psychiat.*, Edin., 1908, vi. 379.—79. Verger. *Arch. gén. de méd.*, Paris, 1900, clxxxvi. 513, 641.—80. Walton and Paul. "Cortical Sensory Areas," *Brain*, 1901, xxiv.—81. White, Hale. *Brit. Med. Journ.*, 1893, ii. 225.—82. Wilson, S. A. K. "A Study of Apraxia," *Brain*, London, 1908, xxxi. 164.

Lesions of the Falciform Lobe.—A considerable body of evidence has been adduced in the preceding section in favour of the localisation of the sense of movement and tactile discrimination in the postcentral gyrus of the Rolandic area. But there is still much controversy respecting the cortical representation of tactile and general sensibility—pain, temperature, etc. In 1875 I recorded a series of experiments on monkeys in which, after destructive lesion of the internal aspect of the temporal lobe, there was profound impairment or annihilation for the time being of all forms of cutaneous sensibility on the opposite side, and a condition of the limbs such as would result from anaesthesia apart from motor paralysis. Similar results were obtained by G. Yeo and myself in 1884 after lesions of the hippocampal region established from the convexity of the temporal lobe and inwards towards the gyrus hippocampi, and without any discoverable lesion of the sensory tracts of the internal capsule or tegmentum. The subject was re-investigated by Horsley and Schäfer, who obtained similar results when the hippocampal region was entirely destroyed. But as the anaesthesia and analgesia tended to wear off, they, at my suggestion, extended their experiments into the gyrus fornicatus as forming part of the limbic or falciform lobe. They thus describe the results of their experiments: "Any extensive lesion of the gyrus fornicatus is followed by hemianaesthesia, more or less marked and persistent. In some cases the anaesthetic condition has involved almost the whole of the opposite side of the body; in others it has been localised to either the upper or lower limb, and to particular parts of the trunk; but we have not yet succeeded in establishing a relationship between special regions of the body and the parts of the convolution which have been destroyed. Moreover, the anaesthesia was frequently very pronounced and general during the first three or four days after the operation, and, indeed, in several instances took the form of complete insensibility to both tactile and painful impressions, so that even a sharp prick or the contact of a hot iron would produce no indication of sensation, but after that time this general condition would become gradually in great part recovered from or more localised in definite regions. In all cases, however, in which the diminution of sensibility was well marked during the first few days, it persisted, although with lessened intensity, for many weeks in those instances in which the animals had been preserved for so long. In other cases in which, apparently, the lesion was slight, the diminution of sensibility, though at first well marked, subsequently disappeared entirely. However, persistent loss of all forms of cutaneous sensibility has never been caused by destructive lesions of the falciform lobe; no doubt because it has been impossible to destroy both limbs of the falciform lobe completely in any one animal."

Further confirmation of these results was given by Dr. Turner and myself, and we also found, as did Horsley and Schäfer, that the anaesthesia was occasionally much more marked in one limb than the other. In particular we found that lesion of the gyrus fornicatus at the point where it passes into the precuneus caused loss of sensation, especially in the opposite leg, which endured long after it had entirely disappeared from the face and upper limb. It has been assumed by many who do not accept our conclusions that the results are due either to dynamical disturbance of the hemisphere as a whole, or direct injury to the sensory tracts. But the anaesthesia caused by lesions of the falciform lobe is of a much more profound character than ever occurs from extensive lesion of the frontal, Rolandic, or other cortical area, and no evidence has been adduced of implication of the sensory tracts at any part of their course towards the cortex. That it is to dynamic disturbance of the falciform lobe and its tracts where they are not actually the seat of lesion that the temporary impairment of tactile and common sensibility is due in connexion with lesion of the Rolandic and parietal areas is, I think, fully established by the experimental results. But the clinical evidence on this head is admittedly only meagre. This is no doubt explicable by the comparative rarity of lesions restricted to the mesial aspect of the hemisphere. There are a few cases on record which confirm the experimental results, if it cannot be said that they are of themselves sufficient to establish the functions of the limbic lobe.

Nor should we, from the facts of experiment, be led to expect any noteworthy defect of cutaneous sensibility except in connexion with lesions of a considerable extent in this lobe. Among the reported cases of lesion of the gyrus fornicatus the following are worthy of mention: Savill has recorded a case of almost complete loss of sensation on the left side, and partial loss on the right, without any apparent loss of power in the arm or leg. The patient recovered from this attack, and again two years later was readmitted with paralysis of the arm and leg. After death an old haemorrhagic cyst was found lying underneath the gyrus fornicatus, part of the marginal convolution, and anterior half of the quadrate lobule, which passed as far inwards as the roof of the right lateral ventricle. The brain was otherwise healthy. The same observer has recorded another case of sarcoma disorganising the posterior third of the gyrus fornicatus, the greater part of the quadrate lobule, and posterior extremity of the marginal convolution, in which, with slight loss of power in the opposite arm and leg, there was almost complete loss of tactile sensibility in the arm. Dr. Churton has reported a case of anaesthesia of the left foot, without motor paralysis, and without any local vascular or other lesion in this limb, in which, after death, there was found a clot the size of a small walnut in the right calloso-marginal fissure, with erosion and discoloration of the subjacent gyrus fornicatus, but without any affection of the suprajacent marginal convolution.

In addition to these instances, in which the lesion was of a stationary character, some cases of tumour may be mentioned. Dr. Handford has recorded a case of tumour occupying the greater part of the left paracentral lobule, with bulging of the gyrus fornicatus, which caused almost complete paralysis of the left leg, and eventually some weakness in the arm and side of the face also. Cutaneous sensation, tactile, and painful, and thermal, was greatly impaired over the whole of the left side. The impairment of sensation in this case was much greater than that which occurs from tumours in the Rolandic area. In a similar case reported by Ackermann, a tumour on the mesial surface of the left hemisphere involved the greater part of the gyrus fornicatus and quadrate lobule. The brain was slightly softened immediately round the tumour, but both the convexity and base of the brain, as well as the centrum ovale and basal ganglia, were quite normal. The symptoms were weakness of the right leg, followed by a similar condition of the arm, together with general hyperaesthesia. Under observation the patient fell into complete hemiplegia and analgesia, with conjugate deviation of the head and eyes to the left. Muratoff reports a case of tumour in the mesial aspect of the left hemisphere, which largely destroyed the gyrus fornicatus, and was adherent to the corpus callosum. Above, it was limited by the calloso-marginal sulcus, and the paracentral lobule was intact. In this case there was some degree of weakness and awkwardness of the limbs on the right side, and touch and pain were abolished. At a later date it was noted that tactile sensibility was normal, but the sense of pain diminished. The author is of opinion that the affection of sensibility observed in this case was not the direct result of the focal lesion, but probably due to interruption of the associating fibres, and compression of the paracentral lobule.

But though it is necessary to be cautious in drawing conclusions from cases of tumour, the four above mentioned exhibit a remarkable agreement as regards the affection of sensation on the opposite side of the body when lesions of this character invade the falciform lobe. Taken with the symptoms observed in the two cases of stationary lesion, they lend at least considerable support to the experimental results obtained by Horsley, Schäfer, and myself.

That the motor centres of the Rolandic area are in intimate organic and functional relation with the sensory centres there can be no doubt, and they probably form a couple connected by associating fibres. Beevor shewed that the falciform lobe is connected with the motor cortex by means of the fibres of the cingulum. It is probable that these fibres form the nexus between the Rolandic zone and the falciform lobe, and that it is through these, or similar connexions, that irritative lesions of the motor zone are apt to excite eccentric sensations in the limbs, and that destructive lesions may produce temporary anaesthesia by causing dynamical disturbance of the centres of the falciform lobe.

According to the observations of Flechsig, with which those of Turner and myself coincide, the falciform lobe is the terminus of a system of corticipetal fibres (system II.) which ascend from the optic thalamus, and are probably the direct continuation of the short longitudinal fibre-system of the tegment. We are of opinion, from the results of our experiments, that in these lie the true cutaneous sensory paths. Though the fibres of the mesial fillet ascend to the postcentral region, there is no satisfactory evidence that they are concerned with the transmission of any of the forms of cutaneous, though they are in all probability the tracts of muscular, sensation. The mesial fillet may be entirely degenerated without loss or impairment of cutaneous sensibility (7).

REFERENCES

1. Ackermann. *Deutsche med. Wchnschr.*, 1893, xix. 253, Case 3.—2. Beevor. *Phil. Trans.*, London, 1891, B. clxxxii. 135.—3. Churton. *Trans. Med. Soc.*, London, 1892, xv. 230.—4. Ferrier. *Functions of the Brain*, 1876.—5. *Idem. Croonian Lectures on Cerebral Localisation*, 1890.—6. *Idem. Phil. Trans.*, London, 1875, B. clxv. 433.—7. Ferrier and Turner. *Ibid.*, 1894, clxxxv. 719.—8. *Idem. Proc. Roy. Soc.*, 1897, lxii. 1.—9. Ferrier and Yeo. *Phil. Trans.*, London, 1884, clxxv. 479.—10. Flechsig. *Localisation der geistigen Vorgänge*, 1896.—11. Handford. *Brain*, 1891, xiv. 296.—12. Horsley and Schäfer. *Phil. Trans.*, London, 1888, B. clxxix. 20.—13. Muratoff. *Neurol. Centralbl.*, 1895, xiv. 398.—14. Savill. *Brain*, 1891, xiv. 270, and *ibid.*, 1892, xv. 448.

Lesions of the Parietal Lobe.—The parietal lobe embraces the *superior* and the *inferior* parietal lobule—the latter formed by the *angular gyrus* and the *supramarginal lobule.* The parietal and the greater part of the temporal lobe constitute Flechsig's *posterior association* area. The functions of the parietal lobe are not agreed on, nor are the effects of lesions uniform or constant. The parietal lobe is frequently invaded by lesions which also implicate the Rolandic area, temporal or occipital lobes, and many of the symptoms which have been observed, more particularly in connexion with sudden lesions, or those which invade not only the cortex but subjacent tracts are without doubt largely of a dynamic character, and not dependent on lesion of the parietal lobe strictly speaking. Irritative lesions frequently cause Jacksonian epilepsy, and the attacks are more often ushered in by a sensory aura than when the lesion is in the motor area itself. Tumours, abscesses, and deeply penetrating lesions frequently cause hemiplegia, hemianaesthesia, and hemianopsy as well as various forms of sensory aphasia and agnostic apraxia. The occurrence of impairment of the muscular sense or astereognosis has been specially noted by Nothnagel, Redlich, and many subsequent observers, and it is still undecided how far these symptoms can be ascribed to lesion of the parietal lobe as such apart from implication or dynamic disturbance of the postcentral convolution or sensory tracts. The superior parietal lobule may perhaps, from the evidence given in the preceding section, be regarded as forming part of the postcentral zone, though it differs somewhat in histological structure. Astereognosis has also been observed in several cases of lesion apparently restricted to the supramarginal lobule. The occurrence of astereognosis in lesions of the parietal lobe, whether a direct or indirect symptom, is of considerable diagnostic value, and more especially if the hemiplegic symptoms are absent or relatively slight. In addition to those already referred to, a case recently reported by Kudlek may be mentioned. This was a case of Jacksonian epilepsy for which an operation was performed but no lesion was discovered. In the operative procedure the cortex was injured in the anterior part of the supramarginal lobule. The result of this was facial paresis of the opposite side, and a noteworthy impairment of sensibility of the opposite hand and arm with loss of the muscular sense and astereognosis. Bechterew has also reported a case of atrophic sclerosis of the left supramarginal lobule and postcentral gyrus associated with sensory aphasic symptoms, Jacksonian epilepsy of the opposite side, and apraxia of the right arm. Though this is described by Bechterew as a case of motor apraxia, it is in my opinion more of the nature of an agnostic apraxia.

The *angular* gyrus is frequently involved in lesions implicating the other parts of the parietal as well as the adjacent portion of the temporal lobe, but owing to the peculiarity of its vascular supply may be affected by thrombotic softening in a more limited manner than the rest of the parietal lobe. Respecting the functions and effects of lesions of the angular gyrus and their interpretation there is considerable divergence of opinion. In the lower monkeys—macaques—the angular gyrus belongs undoubtedly to the excitable region of the cortex. I have never failed to obtain positive results from faradisation of both the anterior and posterior limbs of this gyrus. The effects are conjugate movements of the eyeballs, and occasionally of the head, to the opposite side, with an upward or downward direction according as the anterior or posterior limb is stimulated. The pupils also were frequently observed to contract. Luciani and Tamburini obtained similar results, but Prof. Schäfer could only obtain them on irritation of the posterior limb, and also on stimulation of various parts of the occipital lobe. Profs. Grünbaum and Sherrington have observed them only on stimulation of the calcarine area and apex of the occipital lobe, and then not easily. It is obvious that these differences must be ascribed to varying conditions of the animals operated on rather than to anatomical or functional differences in the corresponding parts. This illustrates how hazardous it is to make a map of the excitable areas of the cortex on the strength of one or two experiments.

My hypothesis that the movements of the eyeballs and contraction of the pupils were indicative of subjective visual sensation was justified by the effects of destruction of the angular gyrus. In my early experiments (1875), again (with Yeo) in 1884, again in 1890 (Croonian Lectures), and again with Turner, I found that unilateral destruction of the angular gyrus caused temporary complete loss of vision in the opposite eye; and that bilateral destruction, temporary complete blindness giving place to an impairment of vision characterised by an inability on the part of the monkey to put its hand precisely on or pick up minute objects. The symptoms were such as can be best explained on the view that there was loss or impairment of central vision. Similar symptoms were also observed by Schäfer and by Munk in their experimental animals, though interpreted differently by each. The results obtained by myself and my colleagues have been controverted by some, and by others attributed to lesion, actual or dynamic, of the subjacent optic radiations. Had this been the case, the result would have been hemianopsy, not amblyopia or complete blindness of the opposite eye. It is quite certain, in contradiction to the statements of Munk, that destruction of the angular gyrus does not cause any loss of sensibility in the eye or any impairment of the ocular movements. There is no ptosis, the conjunctival reflex is active, and the eyeballs are capable of movement freely in all directions.

It has been maintained, however, by several observers (Landouzy, Grasset, Wernicke, etc.) that clinical facts indicate that the *pli courbe*, or angular gyrus, is the cortical centre for the levator palpebrae superioris, as well as for the conjugate movements of the eyes to the opposite side; and they adduce cases of unilateral ptosis, and conjugate deviation of the eyes to the side of lesion, in which the angular gyrus has been found to be the seat of lesion. Thus Chauffard has recorded a case of word-blindness and -deafness due to lesion of the left angular and supramarginal gyri in which there was some degree of ptosis of the right eye. Lemoine has reported a case of slight paresis of the left limb, with ptosis and slight external strabismus of the right eye. The patient died some years after of apoplexy and left hemiplegia. In addition to the lesions which caused the left hemiplegia there was in the left hemisphere an old lesion exactly occupying the angular gyrus, and extending towards the parallel and intra-parietal fissures. There was said to have been no alteration in the pons, medulla, or oculomotor nerves. Surmont has reported a case of ptosis of the left eye, and rotation of the head to the right, without paralysis of the limbs. In the right hemisphere there was found a yellow softening occupying the upper half of the posterior third of the first temporal convolution and the lower half of the inferior parietal lobule, extending slightly into the ascending parietal convolution. In a case of successive lesion of both supramarginal gyri (implicating also the postcentral gyrus in the right) reported by Schaffer, the patient observed anaesthesia of the opposite hand after each attack. Subsequently, after an injury, the patient was found by Schaffer to be anaesthetic over the whole body, except when her attention was excited, in which case sensory impressions were generally felt, but altogether erroneously localised. Schaffer regards the supramarginal gyrus as an association-area for superficial and deep sensibility.

Grasset has reported a case of ptosis of the left eyelid, in which after death was found a patch of exudation, with intense congestion of the cortical substance, at the upper extremity of the parallel fissure in the immediate neighbourhood of the right angular gyrus. There were, however, in this case, as Charcot and Pitres remark, signs of diffuse meningitis on the convexity of both hemispheres. In a case of conjugate deviation of the eyes to the right, reported by Wernicke, in addition to lesion of the medullary fibres of the inferior parietal lobule in the right hemisphere, there was a lesion also in the right half of the pons. In most cases in which conjugate deviation of the eyes has been observed, there has also been hemiplegia; and the lesions, if they involved the inferior parietal lobule, invaded the motor region also; whilst in some cases there was no lesion whatever in the angular gyrus or in its neighbourhood.

On the other hand, a large number of cases are on record in which, notwithstanding extensive lesions of the angular gyrus, no ptosis was observed. Thus Surmont has found only 11 cases of blepharoptosis, against 50 in which this symptom did not occur. In a collection of strictly cortical lesions implicating the angular gyrus (analysed for me by Dr. Ewens), in 30, in which the angular gyrus was affected along with the Rolandic zone, there was no ptosis or conjugate deviation of the eyes; in 16 cases of lesion of the parietal lobe, in 6 of which the angular gyrus was alone affected, ptosis occurred only in 2; whilst in 13 cases of lesion of the parieto-temporal region, in 10 of which the angular and upper temporal gyri were affected, only one shewed conjugate deviation of the eyes.

It is obvious, from this analysis, that neither ptosis nor conjugate deviation of the eyes is a common symptom in connexion with lesions of the inferior parietal lobule; and that clinical facts do not justify the assumption of any causal connexion between them.

The facts of experiment, however, would lead us to expect that irritation of the inferior parietal lobule might cause conjugate deviation of the eyes to the opposite side. And a case in confirmation of this hypothesis has been recorded by Thompson. This was a case of fracture of the skull in the left parietal region—cured by trephining—in which there was marked conjugate deviation of the eyes to the right. On the other hand, a similar symptom was produced by a patch of tuberculous meningitis over the upper part of the middle frontal convolution, in a case reported by Chouppe.

Wernicke's hypothesis, that double lesion of the angular gyrus causes a pseudo-nuclear ophthalmoplegia, is supported neither by experimental physiology nor by clinical evidence of any value.

Hughes Bennett has shewn that irritative lesions of the angular gyrus give rise occasionally to optical illusions or flashes of light. The only symptom that has been established with any degree of uniformity in connexion with destructive lesion of the angular gyrus is the occurrence of *word-blindness* when the lesion is in the left hemisphere. This may be associated with right hemianopsy if the lesion extends deeply into the substance of the hemisphere; but hemianopsy is not constant, nor has it any causal relation to the condition. The word-blind is not blind *presentatively*, for he may be able to see minute objects; but he is blind *re-presentatively*, and fails to read or interpret the meaning of written or printed characters or symbols.

Occasionally his agnosia may extend to objects and their uses (agnostic apraxia), but permanent and complete visual agnosia can only occur in connexion with complete destruction of the visual centres on both sides. Many instances of this affection are on record. One of unusual interest has been reported by Macewen. This was a case of traumatic lesion of the left angular and supramarginal gyri causing word-blindness and mental disturbance, but no other symptoms. The patient recovered after trephining. In a case recorded by Henschen (11), softening of the left angular gyrus was associated with word-blindness. A similar condition existed in Chauffard's case together with slight ptosis of the right eyelid. A large number of instances might be quoted (*vide* Starr), all bearing out the fact that lesions of the angular gyrus and its neighbourhood in the left hemisphere cause, often without any paralytic symptoms whatever, the special form of sensory aphasia called word-blindness. Various explanations of word-blindness have been given—all more or less speculative. Niessl v. Mayendorf is of opinion that it is in reality due to a subcortical or medullary lesion which cuts off a special bundle of fibres proceeding from the posterior and lower thalamic region towards the outer basal half of the occipital lobe. My own view is that it is merely a special manifestation of the more general condition *visual agnosia*[1]; and is an illustration of the laws of evolution and dissolution of the nerve-centres so ably expounded by Dr. Hughlings Jackson. As evolution is from the most simple and most stable up to the most complex and least stable, so destructive processes annihilate first the higher and last of all the lower functional manifestations. The functions of the visual centres in respect to visual sensation or simple presentation are much more stably organised than those that imply visual representation (apperception) and ideation. Most specialised and therefore least stable are the associations between written or printed characters and articulations or things signified, and hence lesion of the angular gyrus, the area of clearest vision, in the left hemisphere, causes the special form of visual agnosia termed word-blindness, just as lesion of the left auditory centre causes the similar condition word-deafness.

---

[1] This is commonly termed *mind-blindness* (Seelenblindheit)—an unphilosophical term coined by Munk which ought to be expunged from medical nomenclature.

The bilateral relations of the angular gyri are sufficient to explain the absence of any permanent or noteworthy impairment of visual sensation in cases of unilateral lesion.

There is comparatively little clinical evidence in favour of the occurrence of crossed amblyopia from unilateral cortical lesion. But Dr. Sharkey has recorded a case of this kind in which the angular gyrus of the opposite hemisphere was for the most part atrophied. And cases of crossed amblyopia from unilateral organic cerebral lesion have also been recorded by Féré, Gowers, and myself (7). The facts of hysterical hemianaesthesia, in which vision is lost, or impaired, in the eye on the anaesthetic side, also postulate the existence in the opposite hemisphere of a centre related to the whole visual field of the opposite side, as well as to some extent on the same side.

Bilateral destruction of the angular gyrus is a rare occurrence. I know of only one case on record—Shaw's case—and the lesion (atrophic softening) invaded not only the angular gyri, but also the superior temporal convolutions on both sides. Probably in this case the lesions were not strictly cortical, but extended deeply below it. The patient, who suffered also from mental aberration, was totally blind as well as deaf.

Not infrequently, where the lesion of the angular gyrus and adjacent portions of the temporo-sphenoidal and occipital lobes has been of the nature of tumour, or has invaded also the medullary substance, there has been observed, in addition to word-blindness, hemianopsy, or hemianopsy associated with a greater or less degree of impairment of tactile and muscular sensibility of the opposite side. Many cases of this kind might be cited (Henschen (12)). In all of them no doubt the optic radiations, together with the posterior part of the internal capsule, were involved to a greater or less extent; for an equal number of cases might be quoted in which similar lesions, not implicating the optic radiations or internal capsule, did not cause hemianopsy, or any apparent affection of sensation.

Summary.—Lesions of the inferior parietal lobule are diverse in their symptomatology. They may be entirely latent, or associated with ptosis, hemianopsy, or impairment of cutaneous and muscular sensibility of the opposite side.

They can only be diagnosed with certainty when situated in the left hemisphere, in which case the pathognomonic symptom is the form of sensory aphasia termed "word-blindness," or inability to read or comprehend the meaning of written language. This always indicates implication of the angular gyrus.

REFERENCES

1. Bechterew. *Monatsschr. f. Psychiat. u. Neurol.*, 1909, xxv.—2. Bennett Hughes. "Sensory Cortical Discharges," *Lancet*, 1889, i. 619, 672.—3. Chauffard.

Rev. de méd., 1881, i. 940.—4. Chouppe. *Bull. Soc. anat.*, 1871, 2. s., xvi. 380.—5. Féré. *Troubles fonctionelles de la vision*, 1882, 305.—6. Ferrier. *Croonian Lectures on Cerebral Localisation*, 1890.—7. *Idem. Brain*, 1881, iii. 456.—8. Gowers. *Diseases of the Nervous System*, ii.—9. Grasset. *Prog. méd.*, Paris, 1876, iv. 406.—10. *Idem. Déviation conjugée de la tête et des yeux*, 1879.—11. Henschen. *Neurol. Centralbl.*, 1886, v. 424.—12. *Idem. Path. d. Gehirns*, 2. Teil, 1892, 280.—13. Jackson, Hughlings. "Croonian Lectures on Evolution and Dissolution of Nervous System," *Brit. Med. Journ.*, 1884, i. 591, 660.—14. Kudlik. *Deutsche med. Wchnschr.*, 1908, xxxiv. 722.—15. Landouzy. *Blépharoptose cérébrale*, 1877.—16. *Idem. Déviation conjugée des yeux*, 1879.—17. Lemoine. *Rev. de méd.*, 1887, vii. 579.—18. Macewen. *Brit. Med. Journ.*, 1888, ii. 302.—19. Niessl v. Mayendorf. *Arch. f. Psychiat.*, Berlin, 1908 xliii. 659.—19*a*. Schaffer. *Monatsschr. f. Psychiat. u. Neurol.*, Berlin, 1910, xxvii. 53.—20. Sharkey. *Med.-Chir. Trans.*, 1883, lxvi. 293.—21. Shaw. *Arch. of Med.*, New York, 1882, vii. 77.—22. Starr. "Sensory Aphasia," *Brain*, London, 1889, xii. 82.—23. Surmont. Quoted by Charcot et Pitres, *Arch. clin. de Bordeaux*, 1894, iii. 389.—24. Thompson. *Brain*, 1883, vi. 99.—25. Wernicke. *Arch. f. Psychiat.*, Berlin, 1889, xx. 243.

Lesions of the Occipital Lobe.—As already stated, electrical irritation of the cortex of the occipital lobe in monkeys gives rise to movements of the eyes similar to those which I first described on irritation of the angular gyrus. Professor Schäfer obtained upward movements on stimulation of the under surface of the occipital lobe and lower part of the mesial surface, but simply lateral movements of the eyes on excitation of the convex aspect of the occipital lobe and margin of the longitudinal fissure. These results have been considered by Schäfer as indicating a special relation of the different portions of the visual field to respective regions of the occipital cortex. Whether this be so in reality has not been proved; and the results of destructive lesions shew that the relation, if it exist at all, cannot be regarded as an exclusive one, inasmuch as the greater portion of both occipital lobes may be removed without causing more than transient visual disturbances, and without producing permanent blindness in any part of the visual field. Unless the lesions are such as to implicate the parieto-occipital fissure, and thus in all probability to cut off the whole of the optic radiations, monkeys at least continue to see; and Schäfer's experiments shew that in order to cause complete loss of vision, not only must the occipital lobes, as such, be cut off, but the lesion must extend well forward into the occipito-temporal convolutions. I have shewn (5) that a transverse incision a few millimetres in depth on the under surface of the occipital region, anterior to the lower extremity of the parieto-occipital fissure, without the infliction of any injury upon the angular gyrus or occipital lobe, can cause persistent hemianopsy; and my view is that by removal of the occipital lobe in this plane the whole of the optic radiations are severed. The exact destination of the optic radiations is a problem which has been much discussed, but that they distribute themselves in the occipito-parietal region of the brain there can be no doubt; for when not only the occipital lobe is destroyed, but also the cortex of the angular gyrus, complete and persistent hemianopsy ensues if the lesion is unilateral, and total blindness if the lesion is bilateral. Practically the same result is obtained by section in the plane of the parieto-occipital fissure, by which the whole of the optic radiations are severed. In harmony with the fact that in monkeys a large proportion of the occipital lobe may be destroyed without producing obvious disturbance of vision, we find many clinical cases in which the occipital lobe has been the seat of lesion, unilateral or bilateral, without any impairment of vision. Lesions of the occipital lobe may, in fact, be entirely latent. Such lesions have been found on the superior, lateral, and inferior aspect, as well as at the apex of the occipital lobe, or even of the mesial aspect or cuneus. Thus, Sir William Gowers has reported a case of tumour of the right occipital region involving the first and second occipital convolutions, the superior and inferior parietal lobules, together with the cuneus and half of the precuneus, in which careful investigation failed to discover any indications of hemianopsy. Brill has reported a case of thrombotic softening of the mesial aspect of the left occipital lobe, including the cortex bordering on the calcarine fissure, the upper edge of the lingual gyrus, and the lower half of the cuneus, in which there was no absolute defect in the visual field, though the patient had diplopia and was colour-blind as regards green.

In the majority of cases in which hemianopsy has been found in association with lesions of the occipital lobe, the lesion has either been of the nature of a tumour, cyst, or abscess, or, if invading the cortex, has also extended into the optic radiations. In many the lesions have been multiple and not confined to the occipital region; and in the great majority also, in addition to hemianopsy, there has been hemiplegia, hemianaesthesia, aphasia, or other symptom of implication of the internal capsule or cortical centres beyond.

The clinical facts, however, seem to shew that, so far as lesions apparently cortical are concerned, hemianopsy is most frequently associated with those affecting the mesial aspect of the occipital lobe.

On the mesial aspect of the occipital lobe, within the calcarine fissure and in the convolutions bounding it, viz. the cuneus above, the lingual lobule below, and the occipital pole posteriorly, the cortex is characterised by the line of Gennari and the presence of a peculiar layer of stellate cells, which occupy the external layer of large pyramidal cells in other regions. In the deeper layers are also the solitary cells of Meynert, which differ from the homonymous cells in other parts of the cortex. The cortex so characterised is the *calcarine* or *striate* (Elliot Smith) area. The calcarine area is surrounded by an area of a somewhat similar cellular arrangement, but without the line of Gennari—the *peristriate* or *pericalcarine* area. The embryological researches of Flechsig shew that the projection-fibres of the optic radiations which distribute themselves to the calcarine area are medullated at full term, and before any of the others which are distributed to the occipital lobe. There are thus, apart from pathological evidence, strong grounds for assuming that the calcarine area is specially related to half vision. Most of the apparently cortical lesions of the calcarine area associated with hemianopsy have, however, been cases of thrombotic occlusion of the branches of the posterior cerebral artery, which supplies not only the cortex, but the subjacent optic radiations which are almost necessarily involved by such lesions.

Examination of 23 cases which have been quoted by Henschen (*op. cit.* p. 319) as purely cortical lesions of the mesial aspect, reveals the fact that in at least 6 of them (namely, those of Sänger, Curschmann, Huguenin, Henschen's case 21, Reinhardt's case 9, and Wilbrand) the optic radiations were also involved; in those of Leegaard, Féré, and Noyes there was no investigation into the condition of the optic radiations; whilst in 14 there was, in addition to hemianopsy, also some degree of hemiplegia, or hemiplegia with hemianaesthesia.

Of three cases of hemianopsy, reported by Vialet, attributed to purely cortical lesion, the first was one of softening occupying principally the internal perpendicular fissure, but extending also into the calcarine fissure. At the same time the cortex of the cuneus was atrophied in its anterior fourth. The lesion, however, was not strictly cortical, but penetrated to the depth of a few millimetres into the zone of the optic radiations. The second case was one of right hemianopsy associated with a lesion which had destroyed the cuneus, extended to the lips of the calcarine fissure, and slightly affected the white substance of the lingual lobule. The internal capsule was stated to have been intact, and yet in this case there was, at the beginning of the illness, turning of the head to the left and rigidity of the right limbs; and marked defect in the sense of position of the right arm persisted. In the third case there was right hemianopsy together with word-blindness. The lesion in this case was recent softening of the whole of the inferior parietal lobule and *pli courbe*, together with *plaques jaunes* occupying the lingual lobule, the fusiform lobe, the cuneus and the apex of the occipital lobe, as well as the splenium of the corpus callosum. The optic radiations were, however, also found softened in this case, though this was attributed to recent lesion.

It is evident from this analysis that in the great majority of the so-called cortical lesions of the mesial aspect of the occipital lobe the lesion extended more deeply than was apparent on the surface; and even when the optic radiations were not demonstrably affected, they were in all probability directly implicated along with the internal capsule, as otherwise it would be impossible to explain the hemiplegia and hemianaesthesia with which the hemianopsy was so often associated.

There are few, if any, cases on record in which coincident lesion of the optic radiations can be excluded. One of the most important is the case of Nordensen-Henschen, in which left hemianopsy was associated with cortical softening of the right calcarine and upper part of the hippocampal fissure. Microscopical examination revealed degeneration, but apparently no primary softening of the optic radiations. This, however, is disputed by von Monakow. If we assume that the degeneration in the Nordensen-Henschen case was merely secondary to the cortical lesion, this case might be regarded as a proof that in man the cortex of the calcarine fissure is pre-eminently the half-vision centre. But in a well-observed and carefully investigated case Beevor and Collier have shewn that the whole of the cortex of the calcarine fissure may be destroyed, as well as a large portion (posterior 2 cm.) of the fusiform lobe, the lingual lobule from the junction of the calcarine and parieto-occipital fissures, and the greater part of the inferior cuneal gyrus, with the production only of quadrantic hemianopsy of the upper part of the opposite visual fields. But, in contrast with this, is a case reported by Hun (not, however, microscopically examined) of softening of the lower half of the cuneus associated with quadrantic hemianopsy of the lower visual fields of the opposite side. It is difficult to harmonise these discrepancies. If, however, we assume, with Beevor and Collier, that the lesion of the inferior cuneal gyrus cuts off the optic radiations proceeding to the cuneus as a whole, we may conclude that the half-vision centre embraces, on the mesial aspect, the whole of the calcarine area, and that the lower quadrant of the retina is represented in the lingual lobule, and the upper quadrant in the cuneus, and that the half-vision centre is not restricted, as Henschen believes, to the cortex in the depth and lips of the calcarine fissure. But, though the calcarine area may be the primary half-vision centre, it is obvious, from experimental and pathological data, that it does not constitute the whole of the visual sphere. The atrophy which occurs from anophthalmos and long-standing blindness does not limit itself, as Dr. Bolton states, to the region of the line of Gennari, but affects also, as von Monakow has found, the whole of the occipital gyri, as well as the angular gyrus, in part at least. The researches of Vialet and von Monakow, as well as those of Dr. Turner and myself, shew that the optic radiations have a much more extensive distribution than the walls of the calcarine fissure. A considerable proportion, the middle stratum, passes to the calcarine fissure ; an inferior layer can be traced to the lingual and fusiform lobules, and a superior layer to the angular gyrus and convex aspect of the occipital lobe. The visual sphere must embrace, therefore, not merely the calcarine area, but the whole of the occipito-angular region (5*a*).

An examination of the clinical facts supports the view that the macula lutea is innervated from both cerebral hemispheres. The dividing line between the sensitive and blind halves of the retina in homonymous hemianopsy of cerebral origin probably never passes through the fixation point. Round this there is always an area of greater or less extent of clear central vision. I have only once seen the dividing line pass through the fixation point, and this was in a case of hemianopsy from lesion of the optic tract itself. It is frequently stated, however, in cases of cerebral hemianopsy that the dividing line had passed through the fixation point ; but such statements are to be accepted with great caution, as mistakes in perimetric investigations are easily made.

In cerebral hemianopsy from lesion of the calcarine area, whether single or double (*i.e.* from successive lesion in each hemisphere), central vision is retained, and may not be far short of normal. In a case of double hemianopsy, reported by Förster, from bilateral lesion of the calcarine area, the patient could read and write without difficulty. There was no word-blindness or alexia. In a case of a similar character, reported by Laquer and Schmidt, central vision remained good. It has been maintained by Henschen that the macula is represented in the anterior part of the calcarine fissure, because in his case this region was intact, whereas Laquer and Schmidt argue on similar grounds that the macula is represented in the posterior extremity of the calcarine fissure, because this escaped in their case. In Beevòr and Collier's case, however, the whole of the calcarine fissure was destroyed, and yet central vision was retained. It is obvious, therefore, that central vision is not represented in the calcarine area. Central vision, however, is always markedly affected in visual agnosia due to bilateral lesions of the occipito-angular regions.

I have in the preceding section adduced evidence in favour of the representation of the macula in the angular gyrus, and endeavoured to shew that the peculiar defects in visual recognition and ideation observed in connexion with lesion of the angular gyrus of the left hemisphere can be most satisfactorily explained in accordance with this view.

Cases have been recorded by several clinical observers (Henschen, Eperon, Dejerine, Mackay, etc.) in which there has been observed a homonymous hemichromatopsy, while the perception of light and form was not obviously or correspondingly impaired. The lesions in these cases have been found in various parts of the occipital lobe, mesial, lateral and occipito-temporal, and no constancy has been established between colour perception and lesion of any particular area. Mackay has furnished strong grounds for regarding such cases as merely varying degrees of essentially one affection; for careful investigation has always revealed not loss of colour perception only, but also distinct impairment of light- and form-perception; and, as the lesion becomes more pronounced, hemichromatopsy passes into complete hemianopsy. In cerebral hemianopsy the pupils react in the usual manner when a pencil of light is thrown on the blind halves of the retinae. The non-occurrence of the pupil reaction when the retinae are so tested (Wernicke's hemiopic pupillary reaction) is significant of lesion of the optic tract. (On this subject see Vol. VII. p. 507.)

Summary.—(i.) Lesions of the occipital lobe may be entirely latent.

(ii.) Homonymous hemianopsy results from destructive lesions of the occipital lobe and optic radiations. This is often associated with a greater or less degree of hemiplegia and hemianaesthesia, from implication of the internal capsule. When the lesion affects also the cortex of the angular gyrus, right hemianopsy is generally associated with word-blindness.

(iii.) Word-blindness alone indicates lesion of the left angular gyrus.

(iv.) Uncomplicated hemianopsy, occurring suddenly with symptoms of vascular obstruction, is indicative of lesion in the calcarine fissure and its neighbourhood.

(v.) In cerebral hemianopsy central vision is unimpaired, and the reaction of the pupils is normal.

REFERENCES

1. Beevor and Collier. *Brain*, 1904, xxvii. 153.—2. Bolton, J. S. "Exact Localisation of the Visual Area," *Phil. Trans.*, 1900, B. cxciii. 165.—3. Brill. *Amer. Journ. Neurol.*, 1882, i. 300.—4. Eperon. *Arch. d. ophthal.*, 1884, iv. 356—5. Ferrier. *Cerebral Localisation*, 1890, 53.—5*a*. Ferrier and Turner. *Phil. Trans.*, 1898, B. cxc. 1.—6. Förster. *Arch. f. Ophthalmol.*, 1890, xxxvi. 94—7. Henschen. *Pathologie des Gehirns*, 2. Teil, 1892.—8. Hun. *Amer. Journ. Med. Sc.*, Phila., 1887, xciii. 140—9. Laquer u. Schmidt. *Virchows Arch.*, 1900, clviii. 466—10. Mackay. *Brit. Med. Journ.*, 1888, ii. 1033.—11. von Monakow. *Arch. f. Psychiat.*, 1889, xx. 714; 1892, xxiv. 229, and *Gehirnpathologie*, 1905.—12. Nothnagel. *Neurol. Centralbl.*, 1885, iv. 41.—13. Schäfer. *Brain*, 1888, x. 368.—14. Seguin. *Amer. Journ. Med. Sc.*, 1887.—15. Vialet. *Les Centres cérébraux de la vision*, 1893.

LESIONS OF THE TEMPORAL LOBE.—Owing to the community of vascular supply between the parietal, occipital, and temporal lobes, the last is not infrequently implicated in lesions due to vascular disease affecting either the parietal or occipital lobe, or both together. The middle cerebral artery, which supplies the Rolandic area, also supplies the angular gyrus and the convex aspect of the temporal lobe; namely, the first and second temporal convolutions. The inferior and inner aspect of the temporal lobe is supplied by the posterior cerebral artery, which is also distributed to the cuneus and lingual gyrus on the internal aspect of the occipital lobe. The temporal lobe is specially connected with the outermost fibres of the foot of the crus cerebri, which pass up into the internal capsule, and then bend outwards and downwards to this region. In cases of lesion of the temporal convolutions these fibres have been found by Dejerine to degenerate downwards as far as the upper region of the pons. This has been confirmed by Dr. Turner and myself after lesion of the first temporal gyrus.

Lesions of the temporal lobe, of whatsoever character, may be entirely latent. There are so many such cases on record that it is unnecessary to allude to them in detail. This latency is more particularly notable when the lesions involve the right lobe, the explanation of which is to be found in the greater preponderance of the left hemisphere in speech processes.

The pathognomonic symptom of lesion of the left temporal lobe is amnesic aphasia (Wernicke) and the condition termed *word-deafness* (Kussmaul). This signifies an inability to interpret the meaning of articulate sounds, though the sounds as sounds are heard. It is the counterpart in the auditory sphere of word-blindness in that of vision. It may exist independently of word-blindness, though, owing to conjoint lesion of the inferior parietal and temporal lobes, the two conditions are often associated together. In all cases in which word-deafness has been a prominent symptom a lesion has been found in the superior temporal convolution. Thus, Seppilli found that in every one of seventeen cases there was lesion of the superior temporal convolution;

and in twelve in the second or middle convolution also. Of twenty-five recorded cases, analysed for me by Dr. Ewens in 1890, ten were due to lesion of the temporal lobe alone. In seven of these the first temporal was particularly affected ; but in the remaining three the exact limits of the lesion were not stated. Eight were due to lesions implicating the angular gyrus, as well as the upper temporo-sphenoidal. Six were due to lesions invading the upper temporal gyrus and adjoining portions of the occipital and parietal lobes ; and in one it was stated that the angular gyrus was affected alone. In this case, however, both word-blindness and word-deafness were present. This was the only case in which there was no obvious lesion of the superior temporal convolution, but since then other cases have been put on record. Thus, Mader reports the case of a patient, aged fifty, who was suddenly seized with disturbance of speech and complete word-deafness. After death a yellowish-white softening was found affecting the left superior and middle temporal convolutions. A similar case, due to lesion of the posterior part of the first temporal convolution and adjoining portion of the inferior parietal lobule, has been recorded by Fraser. Perhaps the most precise case, with the characters of an experimental lesion, has been recorded by Hammond. It was that of a young man who received a blow on the left temple which rendered him temporarily unconscious. On recovery he was found to be aphasic and word-deaf. On trephining over the superior temporal gyrus an extravasation of blood was found and a splinter of bone embedded in the posterior part of the superior temporal gyrus. Many other cases, all indicating the posterior half of the superior temporal gyrus as the special seat of lesion in word-deafness, have been collected by Mirallié, and by Niessl v. Mayendorf, to which reference may be made.

The relation of word-deafness to lesion of the left hemisphere appears to follow the same rule as obtains in reference to aphasia and lesion of the speech-centre of the left side. Thus, Seppilli (36) has recorded in a left-handed man atrophy and sclerosis of the first and second temporal gyri in the left hemisphere without any disturbance of speech or hearing ; and a case of a tumour invading the whole of the left temporal lobe, without affection of speech or hearing, in a patient supposed to be left-handed has been reported by Westphal.

The facts of word-deafness indicate that the region in which the lesions are habitually found constitutes the cortical centre of auditory perception and ideation, the non-recognition of articulate sounds being only a special form of auditory agnosia, like the cognate defect word-blindness in the visual sphere. Experimentally I have shewn that electrical irritation of the posterior half of the superior temporal gyrus in monkeys and its homologue in lower animals causes reactions—pricking of the opposite ear and movements of the head and eyes—indicative of subjective sounds in the opposite ear, and I first pointed out (1875) that destruction of this gyrus on both sides caused loss of auditory perception. My earlier experiments were amply confirmed by others made in conjunction with Yeo and demonstrated at the International Congress in 1881, and others detailed in my Croonian Lectures on Cerebral Localisation (1890). Thus, one of the experimental animals in which the superior temporal gyri were almost completely destroyed on both sides at first failed to respond to any of the sounds which formerly excited active reaction, and which invariably attracted the attention of normal monkeys. After some weeks it seemed to be in some degree sensible to sonorous vibrations, but it never seemed to recognise the sounds which formerly were of significance to it. The obscure sensibility to sounds which was observed in this animal might have been due either to incomplete removal of the auditory region or to the functional activity of lower centres. Destruction of the rest of the temporal lobes except the superior gyri did not cause any impairment of hearing. Similar but less exact experiments have been made on dogs by Munk, Luciani and Tamburini, and Larionow.

Professor Schäfer was unable to detect any signs of impairment of auditory perception in his animals after destruction of the superior temporal gyri, but the data of anatomy and pathology fully confirm the experimental results I obtained. The embryological researches of Flechsig have shewn that a relatively early myelinating strand of projection-fibres connects the corpus geniculatum internum with the inner aspect of the superior temporal gyrus and more especially with the anterior transverse gyrus of Heschl. This strand is the central cortical auditory tract, and is the indirect continuation of the cochlear division of the auditory nerve. The region to which it is distributed possesses certain peculiarities of structure which differentiate it from the rest of the temporal lobe—a richness in cells and the presence of curious giant-cells above the well-developed stellate layer (Campbell).

Apart from the phenomenon of word-deafness from lesion of the left superior temporal gyrus, there are now several cases on record in which bilateral lesion of this region has caused deafness. One of the most carefully observed cases of this kind has been recorded by Wernicke and Friedländer. A woman, aged forty-three, who had never suffered from deafness or affection of vision, was attacked on June 22, 1883, with right hemiplegia and aphasia. She remained in the hospital until August 4. At this time she could speak, but she spoke unintelligibly and was sometimes believed to be intoxicated. She not only could not make herself understood, but she could not understand what was said to her. She was received into the hospital again on September 10, with slight paresis of the left arm. The right hemiplegia had entirely disappeared. The patient was looked upon as insane; she was absolutely deaf, and could not be communicated with. She died on October 21. An extensive lesion was found invading the superior temporal convolution on both sides. The rest of the brain did not exhibit any abnormality, nor were there any conditions leading to increase of the intracranial pressure or secondary affection of the cranial nerves. The patient had previously enjoyed excellent health, her total deafness occurring suddenly in connexion with the other indications of cerebral disease. The result of the examination of the ears was practically quite negative; a slight dry catarrh was found, but locally nothing to account for deafness.

Shaw has recorded a case of a woman, aged thirty-four, who, two months before her admission into his asylum, lost power in the right arm, and soon after had a sudden apoplectic seizure, resulting in loss of speech and deafness. On admission she was found to be perfectly deaf and blind. She had occasional fits, and ultimately died of pneumonia a year after her admission. There was complete atrophy of the angular gyri and superior temporal convolutions in both hemispheres. The optic nerves exhibited increase of connective tissue, but the other cranial nerves were normal in appearance. It may perhaps be questioned if the blindness were due to the lesion of the angular gyri as such; but the sudden onset of deafness in this case, coincident with symptoms of cerebral lesion, and the condition of the brain, point to the destruction of the superior temporal convolutions as its cause.

A third case of a similar nature has been recorded by Mills (22). The patient was a woman, aged forty-six, who fifteen years before her death had an apoplectic attack which left her word-deaf, but not paralysed. She could hear sounds, though she could not understand what was said to her. Six years later she had a second apoplectic attack, causing partial left hemiplegia, together with total deafness for sounds as well as words. She was examined by Mills a few days before her death, which occurred from asthenia and emaciation. In the left hemisphere the posterior two-thirds of the first temporal convolution was reduced to a thin strip; and at the posterior quarter of the second temporal convolution was a cavity, evidently the remains of an old embolic softening. The lobe was otherwise normal. There was, however, some degree of atrophy of the lower portion of the central convolutions and hinder part of the first frontal. In the right hemisphere was an old haemorrhagic cyst destroying the first and second temporal convolutions, the insula and the lower end of the central gyri, together with the lenticular nucleus and external capsule. The auditory nerves were atrophied, and the striae acusticae, usually so easily seen, could not be made out with the naked eye. Dr. Mott has recorded a case of cortical deafness, sensory aphasia, and partial right hemiplegia and anaesthesia. The lesion was softening and atrophy of the left paracentral lobule and gyrus fornicatus, as well as of the posterior third of the superior and middle temporal gyri and the greater part of the supramarginal gyrus. In the right hemisphere the lesion was of similar extent on the lateral aspect, but the mesial surface was intact. Similar cases, but less precise, have been also recorded by Pick and Serieux.

Owing to the bilateral relations of the auditory centre one would not expect to observe anything more than merely temporary disturbance of hearing in the opposite ear from unilateral destruction. But even in cases of unilateral lesion of the superior temporal convolution, some observers have noted deafness in the opposite ear; but the cases are not altogether free from doubt, as the condition of the ears does not appear to have been quite satisfactorily examined. Thus, Kaufmann has reported a case of obliteration of the chief branch of the right Sylvian artery, causing softening of the first and second temporal gyri, together with atrophy of the lower end of the posterior central and supramarginal gyri. The symptoms in this case were left hemiplegia without aphasia, and total deafness of the left ear, though the patient was said to have heard well before the attack. Ferguson has recorded a case in which there was already long-standing deafness in the right ear from otitis media. Two years before death convulsive movements occurred on the left side accompanied by an auditory aura, and followed ultimately by entire loss of hearing in the left ear. The necropsy revealed the existence of a tumour affecting the first and second temporal convolutions in the right hemisphere, destroying the first entirely, and the second partially. Some clinical observers (Edgren, Probst) have endeavoured to correlate *tone-deafness* with lesions of the temporal lobe distinct from those causing word-deafness, and specially localise this faculty in the anterior two-thirds of the superior temporal gyrus. But Mills and Spiller have recorded a case of lesion of this region causing sensory aphasia without tone-deafness. Tone-deafness may occur with word-deafness and general impairment of hearing from cortical disease. In a patient under my own care, presumably with bilateral lesion, there is total inability to perceive the sound of a tuning-fork, though he hears the human voice. His word-deafness, at first complete, is now only partial. There is some paraphasia and paragraphia.

Irritative lesions in this region occasionally cause, as in Ferguson's case, an auditory aura, whether the lesion be in the right or the left hemisphere. Sir W. Gowers has recorded two cases of this nature: in the one a tumour, of which the oldest part was beneath the superior temporal convolution, caused convulsions beginning with an auditory aura referred to the opposite ear; in the other a tumour affecting the superior temporal gyrus caused unilateral convulsions preceded by a loud noise as of machinery. Hughes Bennett has reported several cases of auditory sensory discharges followed by temporary loss of hearing in the opposite ear or in both. Thus, a woman, subject to epileptic attacks preceded by a loud noise like the ringing of a bell in the left ear, became temporarily deaf in both ears after each attack. Both ears were defective in hearing, but the left more so. And in a case of abscess in the right temporal lobe, due to otitis media, under my care the prominent symptom which indicated the seat of the lesion was the occurrence of auditory hallucinations towards the left, so vivid that the patient under examination would occasionally suddenly turn to the left, pointing and saying, "Didn't you hear that?" The abscess was evacuated, but the patient succumbed.

Of important bearing on the question of the position of the auditory centre are the facts relating to the condition of the brain in deaf-mutes and in cases of deafness of long standing. Mills has described the brain of a woman who was deaf for thirty years, which, otherwise normal, exhibited extensive atrophy of both superior temporal gyri, particularly of the left. And Broadbent has described the brain of a deaf-mute in which, in addition to some defect of the annectant convolutions and supramarginal lobule, there was atrophy of both superior temporal gyri, especially of the left.

It has been found by von Monakow, Zacher, and Dejerine, and confirmed by Turner and myself, that lesions of the first temporal gyrus lead to degeneration downwards in the outer portion of the pes cruris traceable as far as the upper pontine region, but not affecting the so-called central auditory tract.

The temporal lobe is a frequent seat of abscess in connexion with disease of the middle ear (*vide* Vol. IV. Part II. p. 475). When the left temporal lobe is so affected the diagnosis is frequently rendered easy by the occurrence of word-deafness and sensory aphasia. When the abscess is situated in the right temporal lobe the diagnosis is more difficult, and may be impossible unless there be some degree of hemiplegia, hemianaesthesia, hemianopsy, or pressure on some of the cranial nerves, of which the third is specially liable to suffer. Apart from such conditions the diagnosis cannot be made with certainty, as the whole of the temporal lobe, even of the left side, may be destroyed without obvious symptoms, provided the superior temporal gyrus is unaffected.

A case has been described, however, by Mills and M'Connell which they consider indicative of the existence of a "naming" centre in the third temporal gyrus. The case was that of a woman, aged forty-one, whose symptoms began with vertigo, followed by word-blindness and right hemianopsy. She could not name objects either by sight or touch, though she understood what they were and what were their uses. Right hemiplegia set in, followed by death in a state of coma. The necropsy revealed a glioma involving the posterior part of the third temporal convolution, and extending slightly into neighbouring parts. The postulation of a "naming" centre on facts such as these appears to me to involve far greater difficulties than to explain them by direct or dynamic disturbance of the centres of visual ideation, or their connexions with the motor speech-centres.

The facts of comparative anatomy, as well as of physiological experiment, indicate that at the lower extremity of the temporal lobe, more particularly in the hippocampal lobule (lobus pyriformis, caput hippocampi), or anterior extremity of the hippocampal gyrus, to which the external root of the olfactory tract is traceable, is situated the cortical centre of smell. The pyriform lobule has a peculiar histological structure, and varies with the development of the sense of smell as does no other part of the limbic lobe, with the exception perhaps of the fascia dentata, which, as Dr. A. Hill has pointed out, is entirely absent in completely anosmatic animals such as the narwhal.

Electrical irritation of this lobule causes reactions such as are indicative of olfactory stimulation, and I have shewn that bilateral destructive lesions abolish or profoundly impair the sense of smell. In connexion with lesions in this neighbourhood I also observed loss or impairment of the sense of taste, so that the animal would unconcernedly eat substances which excite nausea and disgust in normal monkeys. The exact localisation of the sense of taste is surrounded by great experimental difficulties. Gorschkow, from his experiments on dogs, places it in the anterior and lower extremities of the third and fourth primary convolutions, which are supposed by Bechterew to be homologous with the region of the operculum, and by Campbell with the anterior portion of the insula.

There are several clinical cases on record which confirm the localisation of the sense of smell in the region indicated above.

Churton and Griffith report a case in which smell was impaired on the side of lesion, by a tumour which caused erosion of the uncus, and which did not appear, directly at least, to have affected the olfactory tract; and several cases have been recorded in which olfactory auras, or crude sensations of smell, have been observed in connexion with lesions implicating this part of the temporal lobe. M'Lane Hamilton reports a case of this kind due to meningitis involving the inferior and anterior extremity of the temporal lobe, without implication of the olfactory tract. Jackson and Beevor have described a case of olfactory aura occurring in connexion with a tumour of the anterior end of the right temporal lobe involving the nucleus amygdalae and hippocampal lobule. A similar case has been recorded by West, one by Siebert, and another by Sander. In the last case epileptiform convulsions were preceded by an unpleasant odour. The lesion was a tumour, which involved the anterior extremity of the temporal lobe, but the olfactory tract was also directly involved; and several other instances of a similar character might be referred to. Owing, however, to the implication of the olfactory tract, some of them at least might be ascribed to direct irritation of this structure; but in others this was not evident, and the aura probably depended upon irritation of the olfactory centre itself. In some cases of tumour invading the temporal lobe and implicating the uncinate region there have been observed, as Dr. Hughlings Jackson has pointed out, "dreamy" epileptiform attacks, with crude discharges of smell and taste. In addition to those already mentioned others have been referred to by Drs. Jackson and Purves Stewart, and more recently by Mills. The records of the National Hospital shew that in 9 cases of tumour of the temporo-sphenoidal lobe operated on, these dreamy epileptiform attacks and olfactory and gustatory discharges formed important elements in the regional diagnosis.

Summary.—(i.) Lesions of the temporal lobe may be entirely latent.

(ii.) Word-deafness is indicative of destructive lesion involving the superior temporal gyrus of the left hemisphere.

(iii.) Subjective sensations of smell and taste are indicative of irritative lesions involving the lower extremity of the temporal lobe.

Lesions of the Insula.—Little is positively known in respect to the functions, and effects of lesions, of the insula. It forms one of Flechsig's so-called association-areas, and has peculiarities of structure differentiating it from the frontal operculum and from the auditory area proper. The posterior division approximates in type to the temporal cortex (Campbell). Owing to its concealed position it is not readily accessible to experimental lesions without risk of injury to the adjacent and subjacent structures, and owing to the community of vascular supply it is liable to softening along with the anterior and posterior boundaries of the fissure of Sylvius. Electrical irritation of the exposed insula I have found to be entirely negative.

The insula is frequently the seat of lesion both in motor and sensory aphasia, but whether it has any special relation to factors concerned in speech, either motor or sensory, is extremely doubtful. The probability is that the disturbances of speech which have been found in connexion with lesions of the insula are due to implication either of Broca's or Wernicke's region, or of association-fibres between these centres. As already stated, Dr. Campbell suggests that the centres of taste are situated in the anterior part of the insula, but there is no clinical evidence in favour of this hypothesis.

REFERENCES

1. ANDERSON, JAMES. *Brain*, London, 1887, ix. 385.—2. BECHTEREW. *Neurol. Centralbl.*, Leipzig, 1900, xix. — 3. BENNETT, HUGHES. "Sensory Cortical Discharges," *Lancet*, 1889, i. 619, 672.—4. BROADBENT. *Journ. Anat. and Physiol.*, 1870, iv. 157, 302.—5. CHURTON and GRIFFITH. *Brit. Med. Journ.*, 1887, i. 1161. —6. DEJERINE. *Compt. rend. Soc. de biol.*, 1894, xlvi. 626—7. EDGREN. "Amusie," *Deutsche Ztschr. f. Nervenh.*, Leipzig, 1895, vi. 1.—8. FERGUSON. *Journ. Anat. and Physiol.*, 1890-91, n.s., v. 292. — 9. FERRIER. *Cerebral Localisation*, 1890. — 10. FLECHSIG. "Die Hörsphäre," *Neurol. Centralbl.*, 1908, xxvii. 2.—11. FRASER. *Glasgow Med. Journ.*, 1893, xxxix. 81.—12. GORSCHKOW. *Neurol. Centralbl.*, Leipz., 1902, xxi. 287.—13. GOWERS. *Diseases of the Nervous System*, ii. 24.—14. HAMILTON, M'LANE. *New York Med. Journ.*, xxxiv.—15. HAMMOND. *Med. Rec.*, New York, 1900, lviii. 1010.—16. HILL, A. "The Hippocampus," *Phil. Trans.*, 1893, B., clxxxiv. 389.—17. JACKSON (HUGHLINGS) and BEEVOR. *Brain*, 1889, xii. 346.—18. JACKSON (HUGHLINGS) and PURVES STEWART. *Brain*, London, 1899, xxii. 534.—19. KAUFMANN. *Berlin. klin. Wchnschr.*, 1886, xxiii. 541.—20. LARIONOW. *Neurol. Centralbl.*, 1899, xviii. 144.—21. MADER. *Wien. med. Bl.*, 1894, xvii. 101.—22. MILLS. *Brain*, 1891, xiv. 465.—23. *Idem.* *Univ. Med. Mag.*, 1889-90, ii. 61.—24. *Idem.* *Journ. Amer. Med. Assoc.*, Chicago, 1908, li. 879.—25. MILLS and M'CONNELL. *Journ. Nerv. and Ment. Dis.*, N.Y., 1895, xxii. 1.—26. MILLS and SPILLER. *Ibid.*, 1907, xxxiv. 622.—27. MIRALLIÉ. "L'Aphasie sens.," Thèse, 1896.—28. VON MONAKOW. *Arch. f. Psychiat.*, 1891, xxii. 1.—29. MOTT. *Arch. of Neurol.*, London, 1907, iii. —30. NIESSL V. MAYENDORF. *Monatsschr. f. Psychiat. u. Neurol.*, 1909, xxv.—31. PICK. *Arch. f. Psychiat.*, Berlin, 1892, xxiii. 896.—32. PROBST. *Ibid.*, 1899, xxxii. 387.—33. SANDER. *Ibid.*, 1894, xxv. 234.—34. SCHÄFER. *Brain*, 1888, x. 362.—35. SEPPILLI. *Riv. sper. di freniat.*, 1884, x. 94.—36. *Idem.* *Ibid.*, 1892, xviii. 591—37. SHAW. *Arch. of Med.*, New York, 1882, vii. 77—38. SIEBERT. *Monatsschr. f. Psychiat.*, 1899. —39. WERNICKE und FRIEDLÄNDER. *Fortschr. d. Med.*, 1883, i. 177. — 40. WESTPHAL. *Berlin. klin. Wchnschr.*, 1884, xxi. 777.—42. ZACHER. *Arch. f. Psychiat.*, 1891, xxii. 654.

LESIONS OF THE CORPUS CALLOSUM.—The commonly received view of the constitution of the corpus callosum is that it forms a great commissure connecting identical points of the two hemispheres with each other. The parts that are not connected together by the corpus callosum, namely, the hippocampal lobules, are, according to Flower, connected by the anterior commissure, which thus forms a supplement to the corpus callosum, and varies inversely with it. This view was contested by Hamilton, who held that the corpus callosum is not a commissure in the ordinary sense, but rather a decussation of certain cortical fibres which do not decussate lower down. He did not, however, adopt Foville's view that the corpus callosum is a decussation of the peduncular fibres. The fibres described by Hamilton are probably corticipetal fibres passing from the optic thalamus across the corpus callosum to the opposite cerebral hemisphere (Ferrier and Turner). It has been maintained by Schnopfhagen, and also by Sachs, that the corpus callosum is a system of associating fibres connecting, not homologous parts of the hemispheres, but regions quite distinct in situation and in function.

The experimental and histological investigations of Muratoff (15) are, however, in favour of the commonly adopted view as to the commissural function of the corpus callosum. Thus, he found, after excision of different portions of the motor cortex, that degenerated fibres could be traced from the excised area across the corpus callosum as far as the cortex of the identical contralateral area,—the number of degenerated fibres being directly proportional to the extent of the cortical lesion, and always limited to a particular region of the corpus callosum, according to the site of the lesion. The degeneration was always more extensive when an identical lesion was made on both sides than on one side only. On section of the corpus callosum, complete or partial (17), he found degenerated fibres traceable to the cortex on both sides, quite identical on the two sides, and lying in a strictly defined field corresponding to the extent and site of the lesion of the corpus callosum. He also found that a simultaneous destruction of the cortex and section of the corpus callosum caused a degeneration similar to each of these experiments alone, but more extensive ;—a fact which he explains on the supposition that half of the fibres of the corpus callosum have the left hemisphere as their trophic centre, and the other half the right hemisphere. Hence, on cortical lesion of one hemisphere the fibres from that hemisphere degenerate ; whilst on section of the corpus callosum the fibres of the opposite hemisphere also degenerate, and thus cause a double degeneration of the callosal fibres on the side of cortical lesion. The experiments of Turner and myself on removal of the occipital lobe and division of the corpus callosum confirm as regards the visual centres those of Muratoff on the motor centres.

Mott and Schäfer found that on stimulation of the uncut surface of the corpus callosum movements were caused on both sides of the body; whilst on irritation of the cut surface movements were obtained only on the side opposite that of irritation. From these experiments they conclude that the movements depend on indirect excitation of the motor centres through the callosal fibres,—a conclusion which was confirmed by the fact that the movements did not occur if the motor centres were extirpated. It would appear, therefore, from these experiments, that the corpus callosum must contain commissural fibres connecting the motor centres with each other; but, inasmuch as the movements were not in all cases strictly localised, they are of opinion that the fibres, though mainly massed in definite parts of the corpus callosum, are not entirely confined to those parts, but are, to a certain extent, scattered along the middle thinner part of the commissure.

I have found that section of the corpus callosum in monkeys does not cause any obvious symptoms, sensory or motor; and Korányi, from similar experiments on the corpus callosum of dogs, has arrived at the same conclusion.

In man pure uncomplicated destructive lesions of the corpus callosum are practically unknown. Several cases are on record of congenital absence or defect of the corpus callosum. In some, associated with malformation of the brain otherwise, there has been notable mental defect, but in others this has not been observed. On this point reference may be made to a paper by Dr. A. Bruce, who has described a case in which the physical and mental powers were practically normal; and to a case reported by Banchi, in which all the cerebral commissures were wanting, and yet without obvious defect, physical or mental.

Tumour is the chief form of disease affecting the corpus callosum, whilst vascular lesions are exceedingly rare. Erb and Hougberg have, however, each recorded a case of complete, or almost complete, destruction of the corpus callosum by haemorrhage; and Kaufmann has described a case of complete destruction of the corpus callosum by embolic softening, secondary to aneurysm with meningitis. The last had been very imperfectly observed during life, but practically no symptoms referable to the corpus callosum were found in any of the three cases. Erb concludes, in accordance with my own and Korányi's experimental results, that in an adult with no previous brain disease, almost the entire corpus callosum can be destroyed without any disturbance of motion or co-ordination, or of sensation, general or special; and without any noteworthy disturbance of intelligence.

Tumours of the corpus callosum, however, almost necessarily invade one or other or both hemispheres, and give rise to a group of symptoms in which one would scarcely expect to find much uniformity. Bristowe, however, from a comparison of the symptoms which he observed in 4 cases, gave the following as the most noteworthy characteristics:—(1) an ingravescent character; (2) the gradual onset of hemiplegia; (3) the association of hemiplegia on the one side with vague hemiplegic symptoms on the other; (4) the supervention of stupidity, generally with extreme drowsiness; a puzzled inquiring look, difficulty of swallowing, and speechlessness due mainly to incessant drowsiness and apathy without actual paralysis; (5) absence of direct implication of the cranial nerves; (6) death from coma. There was also in Bristowe's cases absence or insignificance of the more striking symptoms of cerebral tumour. Headache was not severe; there was practically no vomiting; optic neuritis was absent in one case, and there were no epileptiform

KING'S COLLEGE HOSPITAL
MEDICAL SCHOOL.

convulsions. Bristowe's syndrome has, in the main, been accepted as a fairly accurate picture of the symptoms of tumour of the corpus callosum by Bruns and Giese. Ransom, from observation of a case, and from analysis of 11 recorded cases, attaches great importance to the early occurrence of mental impairment; and Schuster found mental symptoms in 31 out of 33 cases. On analysis of 16 recorded cases I found (1899) impairment of intelligence, chiefly in the form of progressive mental dulness—or dementia more or less in all; optic neuritis in 6 certainly, and in 3 doubtfully; headache in 7; vomiting in 3; hemiparesis in 6, and paraparesis in 4.

An exhaustive analysis of the recorded cases of tumour of the corpus callosum up to 1908 has been made by Lippmann. He also lays special stress on the profound impairment of intelligence and the bilateral affection of the motor powers. In a remarkable case of glioma of the whole corpus callosum, recently described by Catòla, these were the most characteristic symptoms.

In connexion with the subject of apraxy mention is made of the relation of the cortical motor centres to each other through the corpus callosum, and Förster succeeded in diagnosing a case of tumour of the corpus callosum, by the fact that, in addition to the general apathy common to those cases, there was more obvious apraxia in the left hand.

Summary.—There are no special symptoms due to absence or lesion of the corpus callosum apart from implication of the cerebral hemispheres. Tumours of the corpus callosum may be occasionally diagnosed; the most characteristic symptoms are a gradually increasing mental impairment, with affection of the motor powers on both sides of the body.

REFERENCES

1. Banchi. Abstract, *Neurol. Centralbl.*, 1906, xxv. 176.—2. Bristowe. *Brain*, London, vii. 315.—3. Bruce. *Ibid.*, 1890, xii. 171.—4. Bruns. *Berl. klin. Wchnschr.*, 1886, xxiii. 340, 364.—5. Catòla. *Neurol. Centralbl.*, 1909, xxviii. 120.—6. Erb. *Virchows Arch.*, 1884, xcvii. 329.—7. Förster. *Neurol. Centralbl.*, 1908, xxvii. 540.—8. Giese. *Arch. f. Psychiat.*, 1892, xxiii. 961.—9. Hamilton. *Textbook of Pathology*, vol. ii. part ii. 644.—10. Hougberg. Reference in *Neurol. Centralbl.*, 1894, xiii. 229.—11. Kaufmann. *Arch. f. Psychiat.*, 1888, xix. 237.—12. Korányi. *Pflügers Arch.*, 1890, xlvii. 35.—13. Lippmann. *Arch. f. Psychiat.*, 1908, xliii.—14. Mott and Schäfer. *Brain*, 1890, xiii. 165, 174.—15. Muratoff. *Neurol. Centralbl.*, 1892, xi. 194.—16. *Idem. Arch. f. Anat. u. Physiol.*, 1893 (anat. Abt.), 97.—17. *Idem. Neurol. Centralbl.*, 1893, xii. 316, 714.—18. Ransom, W. B. *Brain*, London, 1895, xviii. 531.—19. Sachs. *Das Hemisphärenmark d. Hinterhauptlappen*, 1892, 22.—20. Schnopfhagen. *Die Entstehung der Windungen des Grosshirns*, 1891, 103.—21. Schuster. *Psych. Störung. bei Hirntumoren*, 1902.

LESIONS OF THE CENTRUM OVALE AND INTERNAL CAPSULE.—The centrum ovale, or white substance of the cerebral hemispheres, contains not only projection-fibres, which by means of the internal capsule connect the cortex cerebri with the periphery, but also fibres which connect the cortex with the optic thalamus, and, according to some views, but less certainly, with the corpus striatum also. Besides this mass of fibres, which constitutes the corona radiata, the centrum ovale contains several systems of fibres, some of which are intra-hemispherical, or associating and connecting different parts of the hemisphere with each other; whilst others are inter-hemispherical or commissural, such as the corpus callosum and anterior commissure, which probably connect corresponding parts of the hemispheres with each other.

As to the functions of the various systems of fibres, which together constitute the centrum ovale, we know nothing with certainty beyond those which form the internal capsule; nor is it always possible to distinguish between lesions of the cortex itself and the subjacent fibres of the corresponding lobes or convolutions. It is desirable for topographical purposes to indicate the position of lesions of the centrum ovale with reference to definite regions of the cortex. For this purpose Pitres has suggested a convenient method of dividing the hemisphere into a series of sections parallel to the fissure of Rolando, or vertical to the long axis of the hemisphere.

The first section through the prefrontal area forms the *prefrontal* section.

The second section through the base of the frontal convolutions forms the *pediculo-frontal* section, which is divided into superior, middle, and inferior fasciculi corresponding with the respective frontal convolutions.

The third section through the ascending frontal or precentral convolution forms the *frontal* section, which is also divided into a superior, middle, and inferior fasciculus.

The fourth or *parietal* section is formed in a similar manner by section through the ascending parietal or postcentral convolution. This also is divided into a superior, middle, and inferior fasciculus.

The fifth is formed by dividing the hemisphere three centimetres posterior to the fissure of Rolando, and, passing through the superior and inferior parietal lobules, constitutes the *pediculo-parietal* section, which is composed of a superior and inferior pediculo-parietal and *sphenoidal* fasciculus.

The sixth is formed by similar division through the occipital lobe, and constitutes the *occipital* section, in which no separate fasciculi are differentiated.

What has been said above with respect to the symptomatology of the prefrontal, occipito-angular, and temporo-sphenoidal lobes is essentially true of lesions of the prefrontal, pediculo-parietal, and occipital sections of the centrum ovale. It is only lesions of the medullary fasciculi which correspond to the fronto-parietal area which cause paralysis of motion and degeneration of the motor tracts of the internal capsule. Such lesions cause symptoms in most respects like those of lesions of the cortex itself in the corresponding regions; that is to say, they cause complete hemiplegia, similar to that resulting from total destruction of the Rolandic area, or of the motor division of the internal capsule; or monoplegia, according as the lesion is limited to the tracts

COLLEGE
LIBRAR

corresponding with the respective cortical centres. Many cases of this kind have been recorded by Pitres, and a large number of those which have been referred to in the previous pages have involved the medullary fibres of this region as well as the cortex itself.

There are few cases on record of monoplegias or dissociated paralyses due to limited lesions of the centrum ovale, not also invading the cortex. Most of such lesions have been situated at the foot of the corona radiata, just where the projection-fibres enter the internal capsule. An interesting case of this kind has been reported by Bennett and Campbell. This was a case of complete paralysis of the left arm, with unimpaired sensibility, which after death was found to be due to a circumscribed softening of the centrum ovale at the point where the medullary fibres of the middle parietal fasciculus converge into the internal capsule, just posterior to the knee. Cases have been reported, by various authors, which shew that lesions limited to the lower fasciculus of the frontal section,—that is, the medullary fibres subjacent to Broca's convolution,—may cause aphasia without any obvious affection of the cortex itself.

It is probable that in these cases the lesion is such as to impair the nutrition, and therefore the functions of the cortex itself; or that it destroys the emissive fibres or those which connect the motor centre with those of sensory perception.

Whilst symptoms of destructive lesion of the centrum ovale of the fronto-parietal or Rolandic area cannot be easily distinguished from those affecting the cortex itself, it is otherwise when the lesions are of an irritative character. Irritative lesions of the motor cortex are, as we have seen, characterised by the occurrence of tonic, followed by clonic, spasms of an epileptiform character. Irritative lesions of the medullary fibres of the Rolandic area cause only tonic, and not the repeated or clonic spasms of the cortical type. It has been proved experimentally that electrical irritation of these fibres causes only tonic contraction proportional in duration to the duration of the stimulus; and there is reason for believing, as Pitres has shewn, that the early rigidity which is seen in some cases of cerebral haemorrhage, and which has been generally attributed to the inundation of the ventricles, is in reality due to irritation of the fronto-parietal fibres of the centrum ovale in the course of their convergence into the internal capsule.

If, however, the lesions are immediately subjacent to the cortex, and are such as to cause irritation of the cortical grey matter also, convulsions of the usual epileptiform type may ensue. This occurs not infrequently in haemorrhage of the centrum ovale; and the occurrence of convulsions of the Jacksonian type in cases obviously haemorrhagic in character has been considered by some to be diagnostic of haemorrhage immediately subjacent to the cortex (von Bamberger).

Of the symptomatology of lesions of the anterior, or *lenticulo-caudate*, division of the internal capsule we know little with certainty. The fibres of this portion radiate into the prefrontal and postfrontal lobes, and they degenerate downwards towards the pons when those regions are the seat of destructive lesions. Their position in the foot of the crus cerebri and probable distribution will be described in connexion with the lesions of the crura themselves (p. 116).

Horsley and Beevor have shewn that by electrical irritation of the internal capsule of the monkey, movements of the head and eyes were caused on irritation of the posterior part of the lenticulo-caudate segment, just anterior to the knee. It would appear, therefore, that this region of the internal capsule corresponds to the cortical centres of the head and eyes, situated, as we have seen, in the postfrontal area.

The posterior, or *lenticulo-optic*, segment of the internal capsule forms with the anterior segment an acute angle termed the *knee*, as seen in horizontal sections. The anterior two-thirds of this segment, including the knee, contains the pyramidal fibres of the motor cortex; and, according to the experiments of Horsley and Beevor, the fibres are so arranged that those from the inferior cortical centres are situated most anteriorly, while those from the superior are situated most posteriorly. Hence irritation from the *knee* backwards causes, in order, movements of the tongue, mouth, upper limb (shoulder preceding the thumb), trunk, and lastly lower limb (hip preceding the toes).

It is theoretically possible that lesions of the internal capsule may be so limited as to cause monoplegia of the cortical type, but this must be exceptional, as the pyramidal fibres of the different cortical centres are so closely congregated together that a focus even of small magnitude must necessarily involve several tracts. The case reported by Bennett and Campbell (*vide* p. 102) was of this exceptional character, and the position of the lesion at the point where the coronal fibres converge into the internal capsule corresponded very closely with the position of the arm fibres in Horsley and Beevor's scheme. That the tracts for the mouth and tongue are situated in the knee of the internal capsule is verified by the occurrence of secondary degeneration in this region in cases of long-standing aphasia, such as have been reported by Charcot and Brissaud, Ross, and others. This tract has been termed by Charcot and Brissaud "the geniculate fasciculus," and the fibres from this region are continued downwards in the mesial portion of the crus cerebri. The position of the pyramidal fibres for the leg at the junction of the posterior with the anterior two-thirds of the lenticulo-optic segment is supported by the fact that in hemiplegia, due to lesions of the internal capsule, in which sensation is notably affected, the leg is frequently more severely and more enduringly paralysed than the arm; thus reversing the order of events in ordinary hemiplegia.

The posterior third of the lenticulo-optic or hinder segment of the internal capsule constitutes the chief path by which the impressions of tactile, muscular, and general sensibility ascend to the cortical centres. With these appear to be associated also the tracts of special sensation, namely, hearing, taste, and smell, together with the optic radiations, situated most posteriorly, which ascend to the occipito-angular region. There is still some doubt as to the path by which olfactory impressions are conveyed to the opposite hemisphere, as we have already seen that the anatomical facts are more in favour of a direct connexion of each olfactory tract with the hemisphere on the same side, and a decussation in the anterior commissure is far from clearly established. It is to injury of this portion of the internal capsule, in all probability, that the hemianaesthesia is due which occurs in connexion with lesions due to blocking or rupture of the lenticulo-optic artery, and in connexion with diseases affecting the optic thalamus. The hemianaesthesia does not necessarily affect all forms of sensibility alike. The most common form is hemianaesthesia of tactile and general sensibility, as well as of the so-called muscular sense. In many cases there is also homonymous hemianopsy, owing to implication of the optic radiations; and that hemianopsy is in the vast majority of instances due to lesion in this region is proved by its common association with hemianaesthesia and a gréater or less degree of hemiplegia.

Hemianaesthesia is frequently of a functional character, as has been pointed out by Charcot and his pupils. In this condition there is loss not only of general and muscular sensibility, but also loss or diminution of hearing, taste, and smell on the opposite side, as well as profound impairment of vision in the opposite eye, characterised by diminution of visual acuity, and concentric contraction of the visual field with dyschromatopsy. The contraction of the visual field occurs to some extent also in the eye on the same side. The visual defect in this form of hemianaesthesia is not of the homonymous hemiopic, but of the cross-amblyopic form.

That conditions similar to those of hysterical hemianaesthesia may occur in connexion with actually demonstrated or highly probable organic lesions of the internal capsule is evidenced by cases reported by Féré, Gowers, and myself already referred to (p. 84).

REFERENCES

1. Von Bamberger. Reference in *Neurol. Centralbl.*, 1895, xiv. 225.—2. Bennett and Campbell. *Brain*, 1885-86, viii. 78.—3. Horsley and Beevor. *Proc. Roy. Soc.*, 1890, xlvii. 21.—4. Pitres. *Lésions du centrum ovale*, Paris, 1877.

Lesions of the Corpora Striata.—The special functional significance of the corpora striata is still doubtful. They cannot be exposed for experimental purposes without inflicting a certain amount of damage on other centres. When exposed in monkeys and other animals by division of the corpus callosum, I found that electrical irritation of the free nucleus caudatus gave rise to contraction of a tonic character of all the muscles on the other side of the body, except in rabbits, in which clonic movements of the jaws occurred during continuance of the stimulation. This general pleurotonic spasm instantly ceased when the electrodes were transferred to the surface of the optic thalamus. From these phenomena I have argued that the corpora striata are related to the movements of the opposite side of the body.

Frank and Pitres and others are of opinion that the movements which I have described are not the direct result of irritation of the grey matter of the corpus striatum, but are due to the diffusion of the irritation to the adjacent motor tracts of the internal capsule. But this explanation does not appear to me consistent with the fact that similar irritation of the optic thalamus, which is quite as close to the motor tracts of the internal capsule, is entirely negative.

Nothnagel found a certain spot in the nucleus caudatus, situated close to the third ventricle, to be mechanically irritable. He states that puncture of this spot in rabbits with a fine needle, or injection therein of a few drops of chromic acid, causes an irresistible tendency on the part of the animal to run or jump until it becomes exhausted. The fact that the internal capsule is undoubtedly excitable by electrical stimulation does not, therefore, exclude the possibility of a direct irritability of the grey matter of the corpus striatum any more than that excitability of the corona radiata excludes the direct excitability of the motor cortex.

Destructive lesions of the nucleus caudatus and nucleus lenticularis cause, according to Nothnagel's experiments, motor disturbances of the opposite side, but more marked in the case of the lenticular than of the caudate nucleus. And Carville and Duret found that destruction of the ganglia of the corpus striatum, together with the tracts of the internal capsule which lie between them, causes a much more complete hemiplegia than that due to the lesions of the motor cortex or pyramidal tracts. The more recent experiments of Mingazzini are also in favour of the view that the ganglia of the corpus striatum are related to the movements of the opposite side of the body, though the exact nature of this relation is not clearly indicated.

The facts of clinical observation in man are somewhat conflicting. The corpora striata, with adjacent tracts, are frequently the seat of destructive lesion, of which the most common is haemorrhage. The favourite source of cerebral haemorrhage is rupture of the lenticulo-striate branches of the middle cerebral artery. This is apt to destroy the grey matter both of the caudate and lenticular nuclei, as well as the tracts of the internal capsule. Less frequently the haemorrhage occurs from rupture of the lenticulo-optic arteries which supply the lenticular nucleus and anterior part of the optic thalamus. Haemorrhage in this region is especially apt to injure the posterior or sensory tracts of the internal capsule. More rarely it occurs in the anterior part of the caudate nucleus from rupture of some of the branches of the anterior cerebral artery.

Haemorrhagic lesions of the corpus striatum, in the first instance at any rate, invariably produce hemiplegia, of motion alone, or of motion and sensation; the latter more particularly when the rupture occurs in the lenticulo-optic artery. Inasmuch, however, as haemorrhagic lesions are sudden, and affect directly or indirectly not only the grey matter of the ganglia, but also the tracts of the internal capsule, it is impossible to distinguish hemiplegia so caused from that occasioned by lesion of the internal capsule itself. An examination, however, of the facts of stationary lesions, such as softening confined to the grey matter of the ganglia, and cysts due to antecedent haemorrhage, has shewn that extensive lesions of this character may be found in the ganglia of the corpus striatum without any symptoms of paralysis, motor or sensory ; and the same is true of tumours. Many examples might be cited from clinical records, which it would be impossible to refer to in detail. But it seems to be well established that stationary lesions of variable size may be found in the grey matter of the corpus striatum, either in the nucleus caudatus or nucleus lenticularis, without the manifestation of any motor paralysis during life.

Pseudo-bulbar Paralysis.—In relation, however, with affections of the corpus striatum, it will be convenient to discuss in somewhat greater detail the pathology of pseudo-bulbar paralysis, which by several observers has been attributed to lesions of the lenticular nucleus. I have already (p. 65) described a group of symptoms simulating bulbar paralysis, resulting from bilateral lesion of the cortical centres of articulation. A similar group of symptoms has also been frequently observed associated with lesions, more especially bilateral, affecting the region of the lenticular nucleus and motor segment of the internal capsule.

Generally there are two distinct attacks of hemiplegia, often separated by a long interval ; and it is only after the second attack, as a rule, that the characteristic bulbar symptoms become manifested. In this condition there is generally a dull, apathetic expression and impaired articulation, the speech being slow, indistinct, monotonous, and with nasal intonation. There is paresis or paralysis of the lips, tongue, and palate, generally also some difficulty in swallowing. Though the vocal cords are, as a rule, unaffected, a few cases have been reported in which paresis or paralysis of one or both vocal cords has been noted (Oppenheim and Siemerling, and Münzer). The limbs are generally more affected on one side than the other (the legs more than the arms) ; and there is often a tendency to convulsive laughing or crying, especially on attempts to speak. Respiratory and circulatory disturbances are, as a rule, absent, though they have been observed in some cases.

Of great importance as a means of distinction between pseudo-bulbar and true bulbar paralysis is the absence in the paralysed parts of atrophy, fibrillar tremors, alteration in the electrical contractility of the affected muscles, or affection of sensation. The reflexes of the palate and pharynx are, as a rule, unimpaired, but they have occasionally been found diminished or even lost. (See cases reported by Lépine, Ross, Colman, Oppenheim and Siemerling, Hughlings Jackson.)

In most of the recorded cases the lesions, generally haemorrhagic or foci of softening, have been bilateral, and more or less symmetrical in the two hemispheres. In a few instances, however, the lesion has been found in one hemisphere only. (See cases recorded by Magnus, Kirchhoff, Ross, Drummond, Bamberger, Wharton Sinkler.) The lesions have been found in the cortex, in the subcortical white matter, or in the central ganglia. The most common seat is the basal ganglia and neighbouring white substance. As a rule the lesions are more or less extensive and diffuse, implicating the central ganglia as well as the capsular fibres; at other times they may be circumscribed, and then they are usually in the lenticular nucleus in its outer segment or putamen. But in these cases it is difficult to eliminate possible implication of the internal capsule, especially in the region of the knee. Cases, however, in which the lesions appear to have been strictly confined to the external division of the lenticular nucleus have been recorded by Lépine and Leresche. Eisenlohr's case is one in which the lesion was confined to the optic thalamus and caudate nucleus, without affection of the lenticular nucleus. Though the pons and medulla have been stated to have been entirely free from lesion in many recorded cases, yet from the degree of paralysis of the palate, pharynx, and vocal cords, which have been reported in some of them, one cannot help suspecting, in accordance with the views of Oppenheim and Siemerling, that there have been undetected lesions in this region. These authors, from an examination of 5 cases observed by themselves, as well as a review of the literature on the subject, have arrived at the conclusion that in the majority of carefully examined instances there are found, in addition to lesions in the hemispheres and ganglia, also small foci of softening in the medulla and pons. These were present in all the 5 cases examined by them. There is no doubt that in a large number of cases of pseudo-bulbar paralysis—in more than half, according to Boulay—there is sclerosis of the basilar vessels, thus favouring the occurrence of haemorrhage, or softening of a multiple character, not only in the central ganglia, but also in the pons and medulla.

The occurrence of symptoms of pseudo-bulbar paralysis from bilateral lesion of the centres and conducting tracts of articulation is readily understood, but it is more difficult to give a satisfactory explanation of the cases of unilateral lesion, as the muscles of articulation and deglutition are innervated from both hemispheres. It is possible that the centres of one side, and, as has been supposed, the left in particular, may predominate; but it is more probable that there have been undetected lesions also on the other side. In those cases in which the lesions have been apparently limited to the lenticular nucleus, which has been regarded as an independent centre for the movements of articulation and deglutition, it is difficult to harmonise the symptoms with the negative results of similar lesions; and it is difficult to disprove that, even where not demonstrated, there have been lesions also of the internal capsule, if we assume that they have been entirely absent from the pons and medulla.

In this connexion, however, great importance is to be attached to a *familial* disease first described by Homén as observed in three members of the same family. The symptoms were giddiness, a drunken-like gait, indistinct speech, impairment of memory and gradually increasing dementia, with rigidity of the whole body, especially of the legs, which became fixed in the flexed position. The necropsy in two of the cases revealed softening in each lenticular nucleus. Some microscopical changes were, however, also demonstrated in the cells and fibres of the cortex.

A few other similar cases have been reported (Ormerod, Gowers (a brother and sister), and Anton). Dr. S. A. K. Wilson has furnished me with the particulars of two, in which he has made a minute pathological examination. The clinical picture is one of slowly progressive tremor and spasticity of the limbs, the tremor being more marked in the arms, sometimes in the head and trunk. At the same time there is progressive difficulty in articulation, resulting eventually in absolute anarthria, and this may be accompanied by dysphagia. The patient is usually emotional, and has ultimately a rather silly look. The cases have been mistaken for disseminated sclerosis, but there is no nystagmus, no affection of sensation, and no change in the discs. The plantar reflex may be normal. Wilson has found bilateral and almost symmetrical softening of the lenticular nuclei, the inner divisions being more affected than the outer; whilst the internal capsules, though undermined by the softened areas, have been found intact. Other parts of the basal ganglia have been normal, and it is remarkable that all the cases have exhibited cirrhosis of the liver. The facts of these cases seem to demonstrate conclusively the relations of the lenticular nuclei to articulation, as well as to steadiness of motor innervation, and explain the occurrence of post-apoplectic tremor which has been observed, among others, by Rhein and Potts in connexion with softening of the lenticular nuclei.

Some facts of experiment, as well as of clinical observation, have been quoted as shewing a relation between the corpus striatum and the temperature of the body. Aronsohn and Sachs found that mechanical and electrical irritation of the caudate nucleus, near the so-called *nodus cursorius* of Nothnagel, caused a notable elevation of temperature, with sensible increase of the respiratory energy and excretion of urea. Dr. Hale White (26) found that puncture of the corpus striatum in rabbits caused a temporary rise in temperature, the highest rise being when the puncture was made anteriorly, averaging in this case about 1° F. To produce this effect it was necessary that the lesion should be bilateral. Ott, however, found that in birds puncture of the corpora striata generally caused a fall of temperature, except when the hinder part was injured, in which case it rose about 1° F. The experimental evidence is thus conflicting.

Several clinical cases have been quoted by Dr. Hale White in support of his experimental results. Cases have also been reported by Nothnagel, Kroemer, and others, in which marked vasomotor disturbances on the opposite side of the body were associated with lesions in the corpus striatum. Thus, Kaiser reports a case of vasomotor paralysis of the right limbs with a temperature in the right axilla varying from 0·1° to 0·15° C. higher than in the left, associated with softening in the left gyrus supramarginalis, the left caudate nucleus, and a small cyst in the left lenticular nucleus. The lesion in the supramarginal gyrus was old, and Kaiser attributes the rise of temperature and the vasomotor paralysis to the lesions in the corpus striatum, especially of the caudate nucleus, which were more recent; and the oedema was only observed about two months before death. This case confirms a similar one reported by Guicciardi and Petrazzani, in which a small haemorrhagic cyst in the left caudate nucleus was associated with right hemiplegia, hyperaemia, and hyperthermia. Dana calls attention to the fact, which has also been observed by several others (Klebs, Kahler, Porechin, Kolisko), that in gas-poisoning there is often softening in the lenticular nuclei associated with vasomotor and trophic disturbances of the skin. He considers that these conditions in connexion with a history of coma from gas-poisoning form a pathognomonic group or "syndrome of the corpus striatum."

Beyond quoting these facts and opinions, however, it does not seem possible, in the present state of our knowledge, to express any definite opinion on the relation of the corpus striatum to thermal and vasomotor innervation.

Summary.—(i.) Lesions of the lenticular and caudate nuclei may be entirely latent.

(ii.) Lesions, such as haemorrhage, involving the internal capsule as well as the nuclei of the corpus striatum, cause hemiplegia of the opposite side, with or without affection of sensation, according as the lesion invades both the anterior and posterior division of the lenticulo-optic segment of the internal capsule or the anterior division only.

(iii.) Pseudo-bulbar paralysis results from lesions in the region of the lenticular nuclei; and progressive softening of the lenticular nuclei causes impairment of articulation, tremors, and spasticity of the limbs and trunk (Homén's syndrome).

REFERENCES

1. ANTON. *München. med. Wchnschr.*, 1908, lv. 2369.—2. ARONSOHN und SACHS. *Pflügers Arch.*, 1885, xxxvii. 232.—3. BAMBERGER. *Jahrb. d. Wiener Krankenanstalten*, 1893, ii.—4. BOULAY. *Gaz. des hôp.*, 1891, lxiii. 793. Abstract in *Brain*, 1891, xiv. 352. —5. COLMAN. *Brain*, 1894, xvii. 86.—6. DANA. *Journ. Nerv. and Ment. Dis.*, 1908, xxxv. 65.—7. DRUMMOND. *Lancet*, 1887, i. 12, Obs. 5.—8. EISENLOHR. *Arch. f. Psychiat.*, 1879, ix. 43, Case 9.—9. GOWERS. *Rev. Neurol. and Psychiat.*, Edin., 1906, iv. 249.—10. GUICCIARDI e PETRAZZANI. *Riv. speriment.*, 1887, xiii. 399.—11. HOMÉN. *Neurol. Centralbl.*, 1890, ix. 514.—12. JACKSON and TAYLOR. *Lancet*, 1892, ii. 1320.—13. KAISER. *Neurol. Centralbl.*, 1895, xiv. 457.—14. KIRCHHOFF. *Arch. f. Psychiat.*, 1881, 132.—15. LÉPINE. *Rev. mens.*, 1877, i. 309.—16. LERESCHE. Thèse de Paris, 1890. Abstract in *Neurol. Centralbl.*, 1891, x. 80.—17. MAGNUS. Müller's *Arch. f. Anat.*, 1837, 258, 567.—18. MINGAZZINI. *Sintomatol. delle lesione del nucleo lenticolare*, 1902.—19. MÜNZER. *Prag. med. Wchnschr.*, 1890, xv. 363.—20. OPPENHEIM und SIEMERLING. *Berlin. klin. Wchnschr.*, 1886, xxiii. 791.—21. ORMEROD. *St. Bart.'s Hosp. Rep.*, 1890, xxvi. 57.—22. OTT. *Journ. Nerv. and Ment. Dis.*, 1893, xx. 1.—23. RHEIN and POTTS. *Ibid.*, 1907, xxxiv. 757.—24. ROSS. *Brain*, 1882-83, v. 145.—25. SINKLER, WHARTON. *Journ. Nerv. and Ment. Dis.*, 1890, xvii. 419.—26. WHITE, HALE. *Brit. Med. Journ.*, 1889, i. 1401.—27. *Idem. Ibid.*, 1891, i. 569.

LESIONS OF THE OPTIC THALAMI.—The special functional significance of the optic thalami, like that of the corpora striata, is still obscure. They have been the subject of experimental lesion by various methods (including electrolysis) by many physiologists, among the more recent of whom may be mentioned Lo Monaco, Sillier and Verger, Probst, Page May, Roussy, and E. Sachs. It is, however, impossible by any method absolutely to restrict experimental lesions to the ganglia, and the lesions of disease are rarely so limited as to exclude direct or indirect damage to neighbouring structures. Anatomically the optic thalami form ganglia of interruption of the sensory tracts, general and special, in their ascent to the cortex; and various cell-groups or nuclei, of which they are composed, are connected with all parts of the cortex by thalamo-cortical and cortico-thalamic fibres. It would be impossible, however, in this short article to give a detailed description of the relations of the optic thalami to the cortex and other structures, and for this subject reference must be had to works on cerebral anatomy (Dejerine, von Monakow, Roussy, E. Sachs). The most common lesions in the optic thalamus are:—(i.) Haemorrhage; (ii.) softening; (iii.) tumours.

(i.) *Haemorrhage.*—As has already been stated, the central ganglia are the most frequent seat of cerebral haemorrhage, and the rupture often extends into the optic thalamus, but does not often begin in it. Andral found that in 301 cases of cerebral haemorrhage affecting the corpus striatum and optic thalamus, only 35 were confined to the optic thalamus itself. In rupture of the lenticulo-optic arteries, the extravasation begins in the thalamus, or between it and the corpus striatum; and it often damages the posterior part of the internal capsule. The small vessels which proceed from the posterior cerebral or posterior communicating arteries to the inner portion of the optic thalamus usually cause haemorrhages near the surface, which are prone to rupture into the ventricle. Those from the posterior cerebral, which supply the hinder part of the optic thalamus, may give rise to haemorrhage into the posterior part of this ganglion, which extends either into the ventricle or downward into the crus or pons.

(ii.) *Softening* in the optic thalamus, as in the corpus striatum, is usually the result of arterial sclerosis, or syphilitic endarteritis.

(iii.) *Tumour* of the optic thalamus is relatively infrequent; and still more rarely is it limited to the optic thalamus.

Affections of Motion.—Hemiparesis or hemiplegia is generally present in lesions implicating the optic thalamus. Thus we find it in all of 13 cases of tumour, in all of 4 cases of haemorrhage, and in 5 out of 6 cases of softening. But there is reason to believe that the motor symptoms are not due to lesion of the optic thalamus as such, but to direct or indirect implication of the internal capsule or cerebral peduncle. When paralysis is found associated with old thalamic foci, we find them usually in the middle third or centre of the ganglion. This is in harmony with the view that the paralysis is in reality due to implication of the internal capsule, as lesions in this situation are more likely to affect the motor tracts. These views are fairly in accordance with the results of experiment. Electrical irritation of the part has always failed in my hands to produce muscular contraction on the opposite side of the body.

Nothnagel did not observe motor paralysis after lesions of the optic thalami. Dr. Turner and I found that almost total destruction of the optic thalamus in a monkey caused weakness on the opposite side of the body, which disappeared within a week; and the probability is that the hemiparesis in this case was due to indirect implication of the internal capsule. Roussy did not observe any true paralysis in his experimental animals; only a slight diminution of muscular resistance.

When paralysis occurs in man, with lesion of the optic thalamus, the clinical picture does not appear to be quite so uniform as that dependent on lesion of the corpus striatum. The face and tongue are less frequently affected than in the latter case, and sometimes isolated paralysis of the arm has been observed. In a case of tumour reported by Lloyd the arm was totally paralysed, the leg paretic, and the face normal; in one by Fisher the arm and face were early affected, and the leg at a later date. On the other hand, Eisenlohr records a case of symmetrical softening of the pulvinar, in which the legs were specially weak, the arms slightly so, and the face normal. The motor affection in connexion with lesion of the optic thalamus is seldom complete paralysis, and is more often a mere paresis, or paresis with rigidity. Thus, in a case of soft vascular tumour under my own care there appeared to be hemiparesis on the opposite side; but on testing the movements of the different segments of the limbs, I found a stiffness or rigidity rather than actual loss of power.

A curious combination of symptoms was observed by Meynert in 2 cases of lesion of the optic thalamus; namely, flexion of the arm opposite and extension of the arm on the side of lesion, the head in both cases being turned towards the opposite side. Schiff and Nothnagel have described similar symptoms after experimental lesions of the optic thalamus. Dr. Michell Clarke, however, has recorded a case of glioma affecting the left optic thalamus and pressing on the internal capsule, which caused right hemiparesis with flexion of the right arm and extension of the right leg, the head and eyes being turned at first to the left, but later to the right. This case differs from those of Meynert in that the arm on the side of lesion was not extended.

Relation to Mimetic Movements.—Nothnagel states that if, in a focal lesion with hemiplegia and facial paralysis, voluntary movement of the facial muscles is abolished, while the two sides of the face are alike in the expression of emotion, such as laughing, crying, pain, we may assume that the optic thalamus and tracts connecting it with the cortex are intact.

Many cases are on record which corroborate this view. Thus, Zenner reports a case of lesion of the optic thalamus in which paresis of the face was more marked in laughing than when shewing the teeth voluntarily. I have observed the same; and Kirilzew has reported a case in which, though the voluntary movement of the face was equal on both sides, the right was quite motionless in laughing or screaming. On the other hand, cases have been observed in which with lesion of the optic thalamus there has been no defect in the mimetic movements. Thus, Eisenlohr has reported the case of a cavity occupying the greater part of the left optic thalamus and posterior part of the internal capsule, in which there was no permanent affection of the mimetic action of the face. A similar case has been reported by Jakob; and Senator has described a case of tubercle invading the whole of the left optic thalamus, in which there was slight obliteration of the right naso-labial fold, which, however, disappeared on crying. Roussy, however, observed no defect of mimetic movements in his experimental monkeys. It would appear, therefore, on consideration of these somewhat contradictory data, that the relation of the optic thalamus to mimetic movements has not yet been satisfactorily established.

Bechterew, from experiments on rabbits, states that irritation of the optic thalami sets in action a complex of movements necessary for the nutrition of the organism, beginning with those of mastication and deglutition, and ending with those of the bowel. And he formulates the view that the optic thalami are the centres for the various vegetative functions of the organism, and of the involuntary movements expressive of feeling or emotion. In reference to this hypothesis it is enough to say that little or nothing beyond the doubtful relation of the optic thalamus to psycho-reflex mimetic movements has been established by clinical observation.

Post-hemiplegic Chorea.—Among motor disorders met with in connexion with lesions implicating the optic thalamus and neighbourhood, unsteady or chorea-like movements in the affected limbs have frequently been observed, more particularly in those cases in which there has been considerable recovery of volitional control. These movements may occur, to some extent, spontaneously; but they are especially marked on volitional exertion. They are allied to the mobile spasm of athetosis, and the condition is generally named "hemiplegic chorea." Hemiplegic chorea is usually, in the earlier stages of the affection at least, accompanied by some degree of hemianaesthesia. The seat of the lesion has been, for the most part, in or outside the optic thalamus, implicating, it may be, the caudate or lenticular nucleus also; and in many instances it has actually involved the internal capsule, more particularly its posterior or sensory division. It is doubtful how far we should ascribe the choreic movement to lesion of the optic thalamus, as such. Often, too, in connexion with tumour of the optic thalamus, the opposite limbs, more particularly the arm, are affected with inco-ordination varying from slight instability to pronounced ataxia, and frequently exhibit the characters of disseminated sclerosis. The choreic, unsteady, and ataxic movements are all probably but varying degrees of the same affection. Unstable or choreic movements in hemiparetic limbs, however, are not necessarily indicative of lesion in the optic thalamus, or external to it; as they may occur also in connexion with lesion of the cortex, corpora striata, midbrain, and other parts. Dr. Gordon Holmes, in an exhaustive analysis of these affections, finds 21 cases of disease limited to the optic thalamus, basal ganglia, and internal capsule; and in 11 of these athetosis resulted; in 7, choreiform movements; and in 3, tremor. In 31 cases the disease was in the mid-brain, and in these the relative frequency of the various forms was reversed, athetosis only appearing once. He concludes that athetosis almost always results from disease of the basal ganglia, but especially of the optic thalamus; choreiform movements occur with about equal frequency in lesions of the basal ganglia and mid-brain, whilst in the large majority of cases of tremor the lesion involves the mid-brain.

The pathology of these choreic and ataxic disorders has been the subject of considerable speculation, but it cannot be said to be yet definitely elucidated (see p. 123).

Sir J. Crichton-Browne is of opinion that considerable destruction of the optic thalamus causes diminution or loss of reflex excitability, but this is by no means a constant symptom, and where it has been observed the lesion has not been limited to the thalamus itself. The state of the knee-jerk varies. In some cases it has been found defective or absent, as in a case of tumour of the left optic thalamus reported by Lloyd. But again, as in a case, reported by Eisenlohr, of symmetrical softening of both pulvinars, both knee-jerks were increased, and double ankle-clonus set in. Hence, neither as regards the superficial nor the so-called deep reflexes can any definite relation to the optic thalamus be considered as established.

Affections of Sensation.—Nothnagel did not observe any obvious impairment of sensation in animals after experimental lesion of the optic thalamus; but hemianaesthesia of greater or less degree has been observed by Probst, Page May, and Roussy; and Roussy calls particular attention to the loss of deep sensibility and sense of position of the limbs. Hemianaesthesia has been frequently observed in connexion with lesions implicating the optic thalamus. Thus, of 4 recorded cases of haemorrhage which I have examined, there was hemianaesthesia as well as hemiplegia in all. Of 13 cases of tumour, 5 had hemianaesthesia, and 2 analgesia also: 1 had anaesthesia of the face only (Sinkler); 1 had anaesthesia of the leg only (Michell Clarke); 1 had anaesthesia and analgesia of the arm (Fisher); and in 5 there was no affection of sensation. Of 6 cases of softening, in 1 sensation was unaffected (Zacher); 1 had almost total hemianalgesia (Eisenlohr); 1 had severe paraesthesia (Edinger); whilst in the others the condition of sensation was not specially mentioned. Roussy has described 7, and referred to 6 others reported by other authors. In all these the symptoms were essentially alike, and were a combination of impairment of superficial and deep sensibility, including loss of muscular sense and astereognosis. Tingling, and other forms of paraesthesia, are not infrequent in cases in which there is no obvious loss of sensation; and in some cases there is a painful hyperaesthesia conditioned without doubt by irritation of the sensory tracts of the internal capsule. Thus, Edinger records a case of softening in the left optic thalamus in which the focus lay directly on the sensory fibres of the internal capsule. On the day after the onset there was right hyperaesthesia, followed by violent pains in the right half of the body, pains so severe as to lead the patient to commit suicide two years later. In Roussy's cases this eccentric hemiplegic pain was a marked symptom.

Meynert attributed the abnormal position assumed by the limbs in his patients to disturbance of the muscular sense, a view which is also shared by Nothnagel. Jackson records a case of softening of the right optic thalamus in which the patient could not distinguish between balls of different weight placed in his left hand. As the loss of muscular sense, astereognosis, etc., observed in this and other cases has always been associated with impairment also of the other forms of superficial sensibility, we are not justified in assuming that the optic thalamus has special relations to the muscular sense.

Affections of the Special Senses.—With superficial and deep hemianaesthesia the special senses are occasionally affected by lesions invading the optic thalamus. This is especially so in tumours. Of the special senses vision is most apt to suffer, and the affection is usually of the homonymous hemiopic character, such as results from lesion of the calcarine area or optic radiations. Thus, in 13 cases of tumour of the optic thalamus which I have examined, hemianopsy was present in 6, absent in 3, and not mentioned in 4.

Dr. Turner and I found that destruction of the left optic thalamus in a monkey (after division of the corpus callosum and exposure of the ventricle) caused total blindness of the right eye lasting for about a week, followed by right hemianopsy which persisted until the death of the animal some months later. There was no obvious injury to the corpus geniculatum externum, but the optic radiations were degenerated. Hemianopsy has also been observed after experimental lesions by Probst, Page May, and Roussy.

In many stationary lesions of the optic thalamus vision does not seem to have been impaired. And yet the obvious origin of some of the fibres of the optic tract from the pulvinar, and the secondary atrophy which ensues in it when the visual centres are destroyed, furnish grounds for assuming that lesions in this region may cause hemianopsy independently of lesion of the optic radiations or external geniculate body. Several instances of hemianopsy in relation with lesion of the pulvinar have been recorded (Jackson, Gowers, Edinger, and Henschen). Against these, however, must be set the cases reported by Zacher and Mills. In Mills's case there was a haemorrhagic cyst which had destroyed about two-thirds of the substance of the optic thalamus, including the entire external tubercle and a large portion of the pulvinar; and yet there was no hemianopsy. In Zacher's case there was almost complete degeneration of one pulvinar, also without hemianopsy. Henschen is of opinion, from his examination of all the recorded cases of hemianopsy in connexion with lesion of the optic thalamus, that no relation can be considered as proved between hemianopsy and lesion of the optic thalamus as such.

As regards affection of the other forms of special sense there are comparatively few observations. In a case reported by Dr. Hughlings Jackson hearing was equal on both sides, but taste was slightly diminished on the opposite side. The patient in smelling sniffed with one nostril only, but, as he remarks, this may have been due to the general anaesthesia rather than to any affection of smell proper. In a case of tumour of the right optic thalamus under my own care, hearing on the left side was much diminished; smell and taste were, however, normal. In a case reported by Henschen of haemorrhage in the left pulvinar, hearing in the right ear was defective; and in Zenner's case of tumour of the left optic thalamus hearing in the right ear was not so acute as in the left. In Zenner's case there were also subjective olfactory sensations, and smell in the left nostril was not so acute as in the right. In a case reported by Engel of a large gumma growing from the back of the fornix, and extending over both optic thalami, there was abolition of smell and taste, then of hearing, and lastly of sight.

The relation of lesions of the optic thalamus to **vasomotor** and **thermal** disturbances is very uncertain, and they have no diagnostic significance. The experiments of Dr. Hale White and of Ott on the influence of lesions of the optic thalamus on the temperature of the body are neither harmonious nor conclusive. White, on puncturing the optic thalami in rabbits, at first observed considerable elevation of temperature on both sides of the body; but from later experiments he came to the conclusion that the rise in temperature, which at most was only slight (0·9 F.) was really due to lesion of the corpus striatum. Ott found that puncture of the anterior part of the optic thalamus caused a rise in temperature, but he has since come to the conclusion that the puncture must enter the tuber cinereum, which he regards as the true centre of thermotaxis. Dr. Turner and I observed no affection of temperature on destruction of the optic thalamus in a monkey. An examination of the recorded clinical facts does not, in my opinion, shew any constant relation between lesions of the optic thalamus and changes in temperature, or disturbance of vasomotor innervation.

Wharton Sinkler has recorded a case of softening in the posterior part of the right optic thalamus in which death took place from profuse intestinal haemorrhage without any apparent ulceration of the intestinal mucous membrane. He connects the intestinal haemorrhage with lesion of the thalamus; and in support of this view he quotes the experiments of Brown-Séquard, Lussana, and others, who found that when lesions were made in the optic thalami ecchymoses occurred in the mucous membrane of the stomach and colon. If, however, there were any real causal relation between the phenomena we should expect it to occur more frequently, seeing that lesions of the optic thalami are by no means uncommon.

Summary.—(i.) Lesions of the optic thalamus cannot with certainty be distinguished from those involving the posterior lenticulo-optic and retro-lenticular division of the internal capsule.

(ii.) Paralysis of the mimetic movements of the face, and the occurrence of post-hemiplegic choreic disorders, are in favour of lesion implicating the opposite optic thalamus.

A lesion of the optic thalamus may be diagnosed, according to Roussy, when there is present the following "thalamic syndrome," viz. (1) A more or less marked hemianaesthesia superficial and deep, especially the latter; (2) A slight hemiplegia, usually without contracture and rapidly regressive; (3) A slight hemiataxy and astereognosis more or less complete; (4) Severe pain in the hemiplegic side, persistent, paroxysmal, and often intolerable; (5) Choreo-athetotic movements of the paralysed limbs.

REFERENCES

1. Bechterew. *Neurol. Centralbl.*, Leipzig, 1894, xiii. 580.—2. Clarke, Michell. *Brit. Med. Journ.*, 1891, i. 1283.—3. Crichton-Browne. *West Riding Asylum Rep.*, 1875, v. 227.—4. Dejerine. *Anat. des centres nerveux*, 1901.—5. Edinger. *Deutsche Ztschr. f. Nervenh.*, 1891, i. 262—6. Eisenlohr. *Deutsche Ztschr. f. Nervenh.*, 1891, i. 388.—7. *Ibid.*, 1893, iii. 260, Case 2.—8. Engel. Ref. in *Lancet*, 1891, i. 266.—9. Fisher. *Journ. Nerv. and Ment. Dis.*, 1895, xxii. 206.—10. Ferrier. *Functions of the Brain*, 2nd edition, 416.—11. Gordon Holmes. *Brain*, London, 1904, xxvii. 327.—12. Gowers. *Diseases of the Nervous System*, 1893, 2nd edit., ii. 316.—13. Henschen. *Pathologie des Gehirns*, zweite Teil, 1892.—14. Jackson. *London Ophth. Hosp. Rep.*, 1875, viii. 316.—15. Jakob. *Deutsche Ztschr. f. Nervenh.*, 1894, v. 188.—16. Kirilzew. Abstract in *Neurol. Centralbl.*, 1891, x. 310.—17. Lloyd. *Medical News*, 1892, lx. 129.—18. Lo Monaco. *Arch. di farmacol. sperim.*, 1904, iii. 475.—19. May, Page. *Brit. Med. Journ.*, 1905, i.—20. Major. *Brit. Med. Journ.*, 1892, i. 318.—21. Meynert. *Wien. psych. Centralbl.*, 1873.—22. Mills. *Journ. Nerv. and Ment. Dis.*, N.Y., 1893, xx. 718.—23. von Monakow. *Gehirnpathologie*, 1905.—24. Nothnagel. *Top. Diagnose der Gehirnkrankheiten*, 1879.—25. Ott. *Journ. Nerv. and Ment. Dis.*, 1891, xviii. 433.—26. Probst. *Arch. f. Psychiat.*, 1900, xxxiii. 1.—27. Roussy. *La Couche optique*, 1907.—27*a*. Sachs, E. "On the Structure and Functional Relations of the Optic Thalamus," *Brain*, London, 1909, xxxii. 95.—28. Sellier et Veyer. *Arch. de physiol.*, 1898, xxx. 706.—29. Senator. *Charité-Ann.*, 1888, xiii. 323.—30. Sinkler. *Journ. Nerv. and Ment. Dis.*, 1893, xx. 719.—31. *Ibid.*, 1894, xxi. 664.—32. White, Hale. *Brit. Med. Journ.*, 1891, i. 1401, and *Journ. Physiol.*, 1890, ii. 1.—33. Zacher. *Arch. f. Psychiat.*, 1891, xxii. 661.—34. Zenner. *Neurol. Centralbl.*, 1893, xii. 607.

Lesions of the Crura Cerebri.—The crura cerebri are the connecting link between the cerebral hemispheres and the lower centres, and form the paths by which sensory impressions are conveyed upwards and motor impulses transmitted downwards. Each crus is composed of two parts—a posterior, or dorsal, forming the tegmentum; and an inferior, or ventral, termed the "foot of the crus"; the two being separated by a layer of grey matter termed the "substantia nigra." The exact relations of the various centres and tracts which constitute the tegmentum, substantia nigra, and foot are as yet only imperfectly determined; but there is good reason to believe that the sensory tracts ascend to the cerebral hemisphere in the fillet and reticular formation. It is also certain, from embryological investigations as well as from a study of secondary degenerations, that the pyramidal tracts which convey impulses from the motor convolutions are situated in the foot of the crus, and more particularly in the middle third or middle two-fourths of this structure. That portion of the crus which lies external to the pyramidal tracts, which was at one time supposed to be the path of the sensory tracts from below, has been shewn by later investigations to contain fibres which connect the occipital and temporal lobes with the grey matter of the pons, and probably indirectly with the cerebellum. The region internal to the pyramids consists of tracts which descend from the lenticular nucleus and frontal convolutions, and include those which form the anterior division and knee of the internal capsule. In these are contained the tracts from the tongue and facial centres. Internal or mesial to these are fibres which, according to Flechsig, as well as according to Dr. Turner and myself, descend from the prefrontal regions; though this is doubted by Zacher, who traces them from the lenticular nucleus. All the tracts, except those which form the pyramids, appear to end in the grey matter of the upper parts of the pons.

The crura cerebri are subject to lesions of various kinds. Haemorrhage limited to the crus is comparatively rare. The same is true of thrombotic softening, dependent on obliteration of some of the branches of the posterior cerebral artery. Abscess is occasionally found invading the crus as well as the optic thalamus and pons. Tumours in the crus, as such, are mostly tuberculous nodules. More frequently the crura are invaded by tumours growing from the base of the skull. Most of the lesions described have been found in the foot or ventral part, and we know more of the symptoms caused by lesions here than in the tegmentum, to which they are very rarely limited.

The complex of symptoms which is characteristic of crus lesion is an alternate hemiplegia involving the limbs, and often also the lower facial region on the opposite side, and the muscles supplied by the third nerve on the same side (Weber's syndrome). When this combination occurs simultaneously, the diagnosis of a lesion of the crus is certain. It is less certain if the hemiplegia occur first, and the oculomotor paralysis at a later date or conversely, though several cases of this kind are on record (Putawski and Goldscheider).

The affection of the third nerve is often complete, involving both external and internal muscles of the eyeball. Thus there is ptosis, which is often the most prominent and earliest ocular symptom, external strabismus with diplopia and giddiness, and mydriasis. Occasionally the oculomotor paralysis is only partial, and in particular affects the levator palpebrae or the internal rectus. Cases have been observed in which the iris has escaped (Gowers). A superficial lesion may cause oculomotor palsy without hemiplegia by damaging the root-fibres of the third nerve; and partial lesions in the middle line, beneath the corpora quadrigemina, may cause symptoms of acute nuclear ophthalmoplegia.

A tumour growing from one crus may also invade the third nerve on the other side, and thus cause a double oculomotor palsy. So also the limbs of both sides may be affected by a tumour primarily affecting one crus, and gradually encroaching on the other; and Weissmann has recorded a case in which an intra-peduncular hydatid caused complete paralysis of all four limbs, without paralysis of any of the cranial nerves. In connexion with tumours, irritative symptoms may occur in the form of spasms and tremors of the paralysed limbs.

Along with motor hemiplegia there may be impairment or loss of sensation. This occurs more particularly when the lesion extends into the tegmental region of the crus; yet cases have been recorded in which lesions have been found in the tegmentum without causing any noteworthy affection of sensation. Thus, Griewe has recorded a case of tumour in the right tegmentum cruris, extending to the optic thalamus in front and the corpus quadrigeminum behind, which, though giving rise to weakness of the left side, did not cause any oculomotor palsy or obvious loss of sensation; the patient, however, complained of pins and needles at the tips of the fingers. Krafft-Ebing has related a similar case. Yet this does not negative the view that the tegmentum contains the sensory tracts, as it is well known that tumours may compress the tracts in which they occur without entirely destroying them. In Weber's well-known and much-quoted case of a large focus of haemorrhage primarily situated in the inner part of the crus, there was considerable loss of sensation—in all probability the indirect effect of pressure on the sensory tracts in the tegmentum. A case of a similar nature has been reported, by Mayor, in which the lesion was also limited to the inner half of the crus. There was motor but no sensory paralysis.

Besides the motor and sensory symptoms above described, there have been cases in which the affection of the opposite limbs was of an ataxic character. Thus, Krafft-Ebing has recorded a case of tubercle situated in the tegment of the right crus, which caused left hemiataxia; and he quotes similar cases reported by Buss and Kahler and Pick. Blocq and Marinesco observed a case of tubercle of the right crus, specially implicating the locus niger, which caused tremors of the left limbs, especially of the arm, similar to those of paralysis agitans, but increased on voluntary movement; and Sachs also has recorded a case of thrombotic softening of the crus, which caused wild ataxic movements of the left arm when voluntary movements were attempted. Further reference to these phenomena will be found in the following section (p. 123).

In lesions of the crus cerebri the special senses are, as a rule, unaffected, or, if affected, only indirectly; this serves to discriminate such lesions from those due to implication of the retro-lenticular division of the internal capsule. The immediate contiguity of the optic tract would lead one to expect occasional hemianopsy. A case of this kind has been reported by Rossi and Roussy.

The views of Budge and Afanasieff as to the relation of the crus to the functions of the bladder are not supported by clinical observations; but Nothnagel quotes cases which shew that vasomotor disturbances in the paralysed limbs, in the form of oedema with increased temperature, are not uncommon.

Summary.—Simultaneous paralysis of the third nerve on one side, and of the limbs on the other, is pathognomonic of lesion of the crus cerebri.

REFERENCES

1. Blocq et Marinesco. *Bull. de méd.*, No. 43.—2. Goldscheider. *Charité-Ann.*, 1892, xvii. 171.—3. Gowers. *Diseases of the Nervous System*, 1893, 2nd edit., vol. ii. 318.—4. Griewe. *Neurol. Centralbl.*, 1894, xiii. 130.—5. Krafft-Ebing. *Wien. klin. Wchnschr.*, 1889, ii. 897.—6. Mayor. *Bull. Soc. anat.*, Paris, 1877, 4. s., ii. 186.—7. Putawski. Abstract in *Neurol. Centralbl.*, 1895, xiv. 177.—8. Rossi et Roussy. *Nouv. icon. de la Salpêtrière*, 1907, xx. 185.—9. Sachs. *Journ. Nerv. and Ment. Dis.*, 1890, xvii. 759.—10. Weber. *Med.-Chir. Trans.*, 1863, xlvi. 121.—11. Weissmann. *Corr.-Bl. f. schweiz. Ärzte*, 1890, xx. 363.—12. Zacher. *Arch. f. Psychiat.*, 1891, xxii. 522.

Lesions of the Corpora Quadrigemina.—The anterior tubercles or nates are connected with the optic tracts by means of the anterior brachia and corpora geniculata externa. The posterior tubercles or testes are connected by the posterior brachia with the corpora geniculata interna, and thus indirectly with the cortex of the temporal lobe. The inferior connexions of the anterior tubercles are mainly through the mesial fillet, by means of which each tubercle is connected directly with the nuclei of the posterior columns on the opposite side. Those of the posterior tubercles are chiefly through the lateral fillet. Dr. Turner and I have found that section of the lateral fillet, including destruction of its nucleus, is followed by degeneration upwards into the posterior quadrigeminal tubercle on the same side, and downwards to the lateral medullary tract and spinal cord. By means of the lateral fillet also the posterior tubercle is connected with the cochlear root of the auditory nerve, the corpus trapezoides, and superior olivary body; for division of the auditory nerve between the accessory ganglion and the side of the pons causes degeneration in the corpus trapezoides and superior olives on both sides, together with bilateral degeneration of the lateral fillet, more marked on the opposite side. The corpora quadrigemina are connected with the spinal cord by tracts which descend in the opposite ventrolateral column.

Diseases affecting the corpora quadrigemina are rarely confined to these structures, and localised haemorrhage or softening is practically unknown. A unique case of traumatic lesion by a bullet has been recorded by Eisenlohr. The most common lesion is tumour, but even tumours occur in this situation more rarely than in other parts of the brain. In all I have been able to discover about thirty cases of tumour of the corpora quadrigemina on record. In the majority of cases of tumour of the corpora quadrigemina the neighbouring parts are also more or less implicated; especially the optic thalamus, tegmentum pontis, the red nucleus, the superior cerebellar peduncles, and often the cerebellum itself. Hence, in the complex of symptoms so caused, it is doubtful how much is due to lesion of the corpora quadrigemina as such, and how much to disturbance of function of the other structures mentioned. But whilst this is so, there are certain symptoms, or rather an assemblage of symptoms, which are suggestive of tumour in the corpora quadrigemina or the immediate neighbourhood. These are, as Nothnagel has pointed out, first, an unsteady reeling gait, especially if this appears as the first symptom; secondly, ophthalmoplegia, not quite symmetrical or implicating all the ocular muscles in equal degree.

The gait is usually described as being that characteristic of a drunken person. It resembles that of cerebellar disease rather than ataxy, though in a case reported by myself the opposite leg was lifted high and thrown forwards in an ataxic manner. Nothnagel found this symptom in all cases of tumour implicating the corpora quadrigemina examined by him, except in one recorded by Sir W. Gowers, in which the lesion was partial and affected only the anterior tubercles. It was present in all the cases that have been recorded since 1889, except in one described by Ruel in which the gait was not mentioned; and in another by Guthrie and Turner in which the patient, when he came under observation, was unable to stand; though probably his gait had been of the usual character in the early stages of the disease.

As the disease advances, walking or even standing becomes impossible; but even when this occurs there is no real motor paralysis except when the disease in its growth presses on the motor tracts of the internal capsule or crus cerebri.

The reeling gait and unsteadiness of equilibrium have been attributed by some to affection of the cerebellum itself, or of its superior peduncles, rather than to affection of the ganglia themselves; but the experiments of Rolando, Serres, Cayrade, and Goltz on the lower animals, as well as my own earlier experiments on monkeys, rendered it probable that lesions of the ganglionic masses of the corpora quadrigemina themselves are sufficient to induce these disorders (6). Nothnagel arrived at essentially the same conclusion from an examination of the clinical cases. But in later experiments Dr. Turner and I found that lesions strictly limited to the tectum of the corpora quadrigemina in monkeys caused no disturbance of equilibrium.

The reeling gait is not, however, pathognomonic, as it occurs also in disease of the cerebellum and in hydrocephalus; but if, as Nothnagel remarks, it occurs as the first symptom, and especially if it coexist with oculomotor disturbances, it points rather to affection of the corpora quadrigemina than of the cerebellum itself. As a rule it occurs before the oculomotor paralysis; but in some recorded cases the ocular symptoms, especially ptosis, shewed themselves first (J. Taylor, Ransom, and Bruns).

The Oculomotor Symptoms.—Nothnagel had never seen complete ophthalmoplegia in cases of tumour of the corpora quadrigemina. There was, however, complete external third nerve palsy in a case reported by Goldzieher; nearly complete double external ophthalmoplegia in Taylor's case; and in Bruns' case there was double oculomotor paralysis, external and internal.

The most common oculomotor symptoms are, first, ptosis on one or both sides; secondly, limitation or loss of the upward and downward movements; thirdly, limitation of the lateral movements. In a case reported by myself, however, the upward and downward movements were good, but there was limitation of the lateral movements. In the case of bullet wound reported by Eisenlohr there was divergent strabismus with defective internal movement of the left eye, the wound being on the right side. In Ransom's case there were constant slight clonic movements of the upper eyelids and of the internal recti.

Paralysis or paresis of convergence is not uncommon, as in the cases reported by myself, Bruns, Bristowe, Kolisch, and others. The sixth nerve may entirely escape; but in some cases there is paralysis or paresis of one or both external recti. Thus, in Ransom's case the left eye was turned strongly inwards, and the right eye also, but to a lesser extent. Nystagmus does not appear to be common, but it was noted in my own and Taylor's cases.

The oculomotor symptoms are not due to lesion of the ganglionic structure of the corpora quadrigemina, but to implication of the subjacent oculomotor nuclei. There were no oculomotor symptoms in a case reported by Nothnagel, nor in another by Weinland. Destruction of the colliculi of the corpora quadrigemina in monkeys does not abolish the reaction of the pupils to light.

Irritation of the corpora quadrigemina on one side causes dilatation of both pupils, especially of the opposite one. There appears, however, to be no definite or constant relation between lesions of the corpora quadrigemina and the state of the pupils. The pupils may remain active while there is almost complete external ophthalmoplegia, as in the cases reported by Taylor, Goldzieher, Bristowe, and others. In some cases they react sluggishly. In the case reported by Eisenlohr the reaction to light was lost in both eyes, and in Guthrie and Turner's case the light reaction was also probably lost. In Ruel's case the pupil on the side of the tumour was dilated and immobile, and at a later date both pupils became inactive both to light and accommodation. In my own case there was loss of the power of convergence; the pupils were equal and contractile to light, but did not contract on attempts at accommodation. In Weinland's case, in which the tumour was situated on the left side, the pupils were dilated, the right being larger than the left; the reaction to light was retained, but on convergence the reaction was feeble on the right side. The pupils are in many cases dilated or unequal.

Affections of Vision.—The obvious connexion of the anterior tubercles of the corpora quadrigemina with the optic tracts and the secondary atrophy which ensues when the corresponding optic tracts are divided, or the cortical centres of vision destroyed, shew these bodies to be important parts of the visual apparatus; but their exact relations to the sense of vision are not clear. Many experimenters have found that destruction of the optic lobes in fishes, birds, etc., or anterior corpora quadrigemina in lower mammals causes total blindness. Dr. Turner and

I, however, observed no affection of vision on destruction of the corpora quadrigemina in monkeys, and in man there is practically no evidence that the nates are concerned with visual sensation. Dr. Bastian mentions a case of total blindness apparently due to a patch of softening almost limited to the anterior quadrigeminal bodies. Nothnagel, however, as the result of his examination of reported clinical cases, makes the statement that vision or visual acuity may be unimpaired although the corpora quadrigemina be entirely destroyed. In Eisenlohr's case of bullet wound of the right corpora quadrigemina, there was no affection of vision immediately after receipt of the injury; but three months later vision in the right eye was diminished, and at a still later date visual acuity was diminished in both eyes. The probability is, therefore, that the defective vision was due to secondary changes in the optic nerves, as otherwise the affection of vision should have occurred on receipt of the lesion.

In almost all the cases of disease of the corpora quadrigemina, in which vision has been defective, the lesion has been of the nature of tumour, with coincident optic neuritis or internal hydrocephalus. In Taylor's case, however, there was no optic neuritis, and yet the child was blind; and Ransom was of opinion that in his case the defective vision was greater than could be accounted for by the neuritis. It should also be remembered in this connexion that there may be perfect vision, notwithstanding the existence of considerable neuritis; as was pointed out long ago by Dr. Hughlings Jackson. Defective vision from optic neuritis usually sets in only when the inflammation gives place to secondary atrophy. In most cases, however, of unilateral lesion of the corpora quadrigemina, with defective vision, there is little or no difference in the visual acuity of the two eyes; and this increases the probability that the true cause is either optic neuritis or internal hydrocephalus.

It is obvious from these facts, therefore, that a direct relationship between the anterior quadrigeminal tubercles and the sense of vision is far from being established by clinical observation.

Affections of Hearing.—Experimental observations, as well as a study of the course of degeneration in lesions of the temporal lobe (p. 90), render it probable that the central tracts of the cochlear nerve ascend through the opposite lateral fillet, posterior tubercle, brachium and internal geniculate body, and thence to the temporal cortex. It is important, therefore, to examine as to any evidences of affection of hearing in connexion with diseases of the corpora quadrigemina. Weinland has analysed the clinical records in this relation, and finds that in 19 cases of tumour of the corpora quadrigemina hearing was affected in 9; and of these, 5 had impairment of hearing on both sides. In 3 cases (reported by myself, Weinland, Ruel) hearing was impaired on the side opposite the tubercles affected; and Weinland concludes, therefore, that disease of the posterior corpus quadrigeminum causes affection of hearing on the opposite side. There are, however, many difficulties in the way of arriving at a correct conclusion on this point; chiefly dependent on the mental condition of the patients, who usually present a considerable degree of mental dulness, somnolence, and apathy. Hence impairment of hearing, even if observed, may be attributed to this cause; or, unless very pronounced, it may altogether escape observation. The experiments of Rothmann indicate that though there is temporary impairment of hearing from destruction of the posterior colliculus, the real primary auditory centre is the corpus geniculatum internum.

Affections of Motion and Sensation.—As a rule, in lesions of the colliculi of the corpora quadrigemina there is no motor paralysis or loss of sensation. In tumours, however, and haemorrhagic lesions involving the tegmentum there may be paralysis both of motion and sensation along with oculomotor paralysis. In the more chronic lesions tremors are not uncommon, chiefly of the arms; and especially on volitional movement, like those of disseminated sclerosis. Tremors of this character were observed in 10 out of 17 cases I have analysed (*vide* cases by Ferrier, Weinland, Bruns, Ilberg, Bristowe, Kolisch, Taylor, Eisenlohr, Pilz, Guthrie and Turner). In 5 of these there was also some degree of motor weakness; in the others this was not observed. The tremors were either bilateral, or, if unilateral, on the side opposite the lesion, except in Weinland's case. In those cases in which the tremor was bilateral the lesion was not strictly confined to one side. Oscillation of the head and neck, as well as of the limbs, was noted in the cases reported by Bristowe and by Taylor. In Ilberg's, and also in Eisenlohr's case, the tremors were described as being similar to those of paralysis agitans. Many similar cases have been reported by subsequent observers.

An examination of the cases, in which an exact necropsy was made, shews that in all in which tremors were observed the lesion was not confined to the corpora quadrigemina, but implicated also the optic thalamus, tegmentum cruris or pontis, red nucleus and the rubro-spinal tract, or superior cerebellar peduncles. In this connexion it is worthy of note that Dr. Turner and I have described similar tremors as resulting from section of the superior cerebellar peduncles in monkeys. In our experiments, in which the peduncle was divided between the cerebellum and its decussation in the tegmentum, the tremors were confined to the side of lesion. In the reported cases the tremors have been generally on the side opposite the lesion. This would be the case if the lesion implicated the red nucleus and its spinal tract above the decussation of the superior cerebellar peduncles. Dr. Gordon Holmes, from his analysis of the reported cases, connects the tremors which are so commonly observed in mid-brain lesions with affection of the cerebello-rubral system.

Electrical irritation of the corpora quadrigemina in monkeys and other animals gives rise to tonic or tetanic spasm of the neck, trunk, and limbs with opisthotonos. A similar condition, termed by Prof. Sherrington *decerebrate rigidity*, ensues when the forebrain is severed posterior to the diencephalon or optic thalami. A similar group of symptoms has been described by Dr. Hughlings Jackson in connexion with tumours of the middle lobe of the cerebellum. As the decerebrate rigidity does not cease on destruction of the cerebellum it would appear probable that this tetanic spasm is due to the unantagonised or positively excited action of the mesencephalic centres. A case supporting this view has been reported by Goldzieher of a small tumour of the corpora quadrigemina causing opisthotonic spasms.

The knee-jerks in lesions of the corpora quadrigemina are either normal or exaggerated. They may, however, be abolished even at an early date, just as in cerebellar disease. (Guthrie and Turner, Ransom.) They were exaggerated in the cases reported by Kolisch, Bristowe, and myself.

In connexion with lesions of the corpora quadrigemina it is only necessary to allude in this place to paralysis of the ocular muscles, external and internal, which occurs from lesions limited to the oculomotor nuclei, as the subject is discussed elsewhere at length (Vol. VII. p. 510). These are, with the exception of the nucleus of the sixth, situated in the central grey substance subjacent to the aqueduct of Sylvius, and consist of different cell-groups which have probably special relations to the various internal and external ocular muscles, and are connected with the sixth nuclei by the posterior longitudinal bundles.

Lesions of the oculomotor nuclei may be acute or chronic. The most common lesion is a chronic degeneration of the nerve-cells, similar to that which occurs in bulbar palsy and progressive muscular atrophy, and is mostly associated with or followed by symptoms of tabes. The oculomotor palsy may be limited to the external muscles, or affect also the internal muscles, so that the condition is one of partial or complete ophthalmoplegia externa as well as interna. An acute form is more rare, and in its onset and pathology resembles acute anterior poliomyelitis (p. 241). From its analogy with this affection Wernicke has named it acute polioencephalitis superior—the term superior being used to distinguish it from a similar affection of the nuclei of the medulla oblongata, which is named acute polioencephalitis inferior. In these cases ophthalmoplegia occurs suddenly with ptosis and paralysis of the conjugate movements of the eyes, together with loss of convergence and the power of accommodation. Such were the symptoms in a case under my care which ended in recovery in the course of a few weeks, with the exception of the powers of convergence and accommodation. The reaction of the pupils to light was sluggish, but not abolished. Several similar instances have been recorded. A third form of lesion, termed "sudden nuclear palsy," has been attributed to obstruction of the branches of the basilar artery which supply the oculomotor nuclei. The oculomotor paralysis in this case is usually irregular, and associated with some degree of hemiplegia, especially on the side opposite the greater lesion.

Summary.—A regional diagnosis of tumour of the corpora quadrigemina is probable when there is a combination of ophthalmoplegia with an uncertain or reeling gait. The probability is increased if there is also some affection of hearing in one or both ears.

REFERENCES

1. Bastian. *Paralysis from Brain Disease*, 115.—2. Bruns. *Arch. f. Psychiat.*, 1894, xxvi. 299.—3. Duffin. *Lancet*, 1876, i. 888.—4. Eisenlohr. *Neurol. Centralbl.*, 1890, ix. 747.—5. Ferrier. *Brain*, 1883, v. 123.—6. *Idem. Functions of the Brain*, 2nd edit., 159.—7. Ferrier and Turner. *Phil. Trans.*, 1894, B. clxxxv.—8. *Idem. Brain*, London, 1901, xxiv.—9. Goldzieher. *Centralbl. f. Augenh.*, 1893, xvii. 44.—10. Gowers. *Lancet*, 1879, i. 327, 363.—11. Guthrie and Turner. *Lancet*, 1895, i. 273.—12. Holmes, Gordon. *Brain*, London, 1904, xxvii. 327.—13. Jackson, Hughlings. *Brain*, London, 1906, xxix. 425, 441.—14. Nothnagel. *Brain*, 1889-90, xii. 21.—15. Ransom. *Lancet*, 1895, i. 1115.—16. Rothmann. *Neurol. Centralbl.*, Leipzig, 1907, xxvi. 922.—17. Ruel. Reference in *Neurol. Centralbl.*, 1890, ix. 630.—18. Sherrington. *Integrative Action of the Nervous System*, 1906.—19. Taylor. *Brit. Med. Journ.*, 1893, ii. 1102.—20. Weinland. *Arch. f. Psychiat.*, 1894, xxvi. 363.

Lesions of the Pons Varolii.—Lesions of the pons Varolii are especially diverse in their symptomatology owing to the complex relations of the pons itself. Not only is it an independent centre, but it contains the sensory and motor paths to and from the cerebrum; it is intimately linked with the cerebellum by means of the middle peduncles, and through it pass many of the cranial nerves in their course to or from their primary nuclei. The symptoms are therefore likely to vary greatly according to the position and extent of the lesion. To give an explanation of these in all their relations necessitates a knowledge of the minute anatomy of the pons, which it is necessary for the purposes of this article to assume on the part of the reader.

We may, however, somewhat simplify the symptomatology of lesions of the pons by classifying them with Markowski according as they affect the *tegmental* or *crustal* region. Lesions of the tegmental region are specially apt to affect the fifth, sixth, seventh, and eighth nerves, as well as to cause disturbances of sensation and co-ordination; lesions of the crustal region affect principally the movements of the limbs, as well as those supplied by the seventh, tenth, eleventh, and twelfth cranial nerves.

The pons is liable to various morbid processes, originating in it primarily or affecting it secondarily. Of the latter character may be specially mentioned aneurysm of the basilar artery, and tumours of the cerebellum or base of the skull.

Haemorrhage is by no means uncommon, though it occurs much less frequently here than in the central ganglia. The haemorrhagic extravasation may vary in size from a hemp-seed to one which causes complete disorganisation of the whole structure. Occasionally there may be several small haemorrhages quite distinct from each other (Russell and Taylor). Large haemorrhages not infrequently burst into the fourth ventricle, or even make their way through the iter into the lateral ventricles. Small haemorrhages into the pons are not necessarily accompanied by a loss of consciousness; but if the haemorrhage be large, profound coma occurs with great suddenness, and leads to death much more certainly and rapidly than similar effusions in other parts of the cerebrum. Death takes place in a few minutes (7 minutes, Mickle); or in a few hours (2 hours, Rorie; 4½ hours, Barr). Bod finds that of 78 cases 46 died within twenty-four hours.

In pontine haemorrhage, along with profound coma, there is often complete relaxation of the muscular system and contraction of the pupils, so that the condition is not unlike that found in opium poisoning. Tonic or clonic convulsions sometimes occur, rarely unilateral, and often irregular in type and distribution. The occurrence of paralytic or convulsive symptoms in the limbs of one side and the face on the opposite is pathognomonic of haemorrhage in the pons. When the limbs are paralysed on one side there is not infrequently conjugate deviation of the head and eyes from the side of the lesion; but if the symptoms are convulsive the deviation of the head and eyes may be towards the side of the lesion. Deglutition is difficult or impossible, and death occurs from cardiac or respiratory paralysis, irregularity of rhythm generally preceding the fatal issue. Hyperpyrexia is frequently observed, and the temperature may rise as high as 109° F. Haemorrhage may occur in the pons not only from diseased arteries, but, as has been shewn experimentally by Duret, from cranial injuries also, more particularly in the frontal region; and I have seen several cases of cranial injuries in man in which the symptoms were those of lesions of the pons, in all probability of a similar nature.

Necrotic Softening.—The most common cause of necrotic softening of the pons is thrombosis, due to atheromatous or syphilitic degeneration of the basilar artery or its branches. Embolic softening is exceedingly rare, though a case of this kind has been reported by Sir W. Gowers. If, however, as Nothnagel pointed out, an embolus sticks in the vertebral, the basilar artery may become gradually thrombosed and blocked.

Tumours occasionally grow in the substance of the pons, but more frequently the pons is pressed on by tumours originating at the base, or situated primarily in the cerebellum. Sometimes the whole pons may be enlarged by gliomatous infiltration, constituting so-called hypertrophy of the pons.

Abscess is rare, but the pons is a favourite seat of sclerotic foci, as in multiple sclerosis; though in this latter case the pons is only affected along with other encephalic centres.

Affections of Motion.—As a rule in lesions of the pons affections of motion predominate over affections of sensation. They may be variously grouped, and at the outset it may be mentioned that cases of lesions of the pons have been recorded in which no symptoms were noted either in the domain of motion or sensation. A remarkable instance of this kind has been recorded by Ladame, in which the whole pons appears to have been transformed into a new formation containing no nerve-elements, and covered over only by a layer of medullated fibres a line thick.

Type 1.—The limbs may be paralysed, but the cranial nerves and nuclei escape. In this group the lesion is generally in the upper part of the pons, and the resulting hemiplegia is indistinguishable from that due to lesion of the internal capsule.

Type 2.—The cranial nerves or nuclei may be affected alone without paralysis of the limbs. In Bennett and Savill's case of softening in the region of the sixth nucleus there was isolated conjugate deviation of the head and eyes. In Mierzejewski and Rosenbach's case of glioma there was right facial palsy and conjugate deviation to the left. In Moeli and Marinesco's case there was paresis of the lower half of the right side of the face, the right sixth, and right portio minor of the fifth, together with affection of sensation on the left side of the body. In Elzholz's case of haemorrhage there was complete paralysis of the right sixth, seventh, and of the twelfth, without palsy of the limbs, a rare condition in haemorrhage.

Type 3.—There may be paralysis of the face and limbs on the side opposite the lesion. In this case the lesion must be above the decussation of the central facial fibres, which is probably about the middle of the pons (see cases by Diller, Mills and Zinner, Markowski). The resulting hemiplegia may be indistinguishable from that due to lesion of the internal capsule, unless some localising symptom be present, such as implication of the fifth on the side of lesion, or of the sixth, as in Mills's and Zinner's case. In 10 cases under my observation 5 shewed this form of paralysis.

Type 4.—The most characteristic form of paralysis due to lesion of the pons is paralysis of one or more of the cranial nerves, especially the fifth, sixth, and seventh on the side of lesion, and of the limbs and tongue on the other side. This is the type of the so-called *alternate hemiplegia* (Grubler).

The most common variety of this alternate hemiplegia is paralysis of the face on the side of lesion, and of the limbs on the other. The facial paralysis is of the peripheral type, and implicates both the upper and lower facial regions; the paralysis of the limbs is of motion alone, or of this combined with anaesthesia. In order to cause this group of symptoms the lesion must be below the decussation of the facial fibres; that is, below the middle of the pons. This grouping of symptoms is practically pathognomonic of a pontine lesion, especially if it occur suddenly. It may, however, be simulated by a basal tumour, or chronic meningitis, compressing the pons and facial nerves; but in this case the onset is usually slow. Joffroy has seen similar symptoms produced by a double cerebral lesion.

(*a*) The seventh may be the only cranial nerve affected on the side of the lesion, but more often other nerves suffer, especially the sixth and fifth. Occasionally, but more rarely, the eighth is also implicated. If the lesion extend into the medulla oblongata, the eleventh and twelfth cranial nerves may likewise become affected, as shewn by unilateral paralysis of the palate, vocal cord, and tongue; and in some cases the ninth and tenth have been involved. Thus, we find the seventh and sixth (Porter, Williams); the seventh, sixth, fifth (motor and sensory), and twelfth

(Jolly); the seventh, sixth, fifth (motor and sensory), and palate (Bristowe); the seventh, fifth, and eighth (Miles); the seventh, fifth, and eleventh (vocal cord and palate) (de Havilland Hall); and among my own cases I have seen the seventh, sixth, fifth, eleventh (vocal cord and palate), twelfth, and eighth; and also a case of seventh, sixth, fifth, and partially also the opposite sixth. Oppenheim records a case in which tubercle of the left side of the pons caused paralysis of the left side of the face, left sixth and fifth, right internal rectus, right side of the face in the lower part, and the right limbs. In cases recorded by Martin Brasch and by Mills and Zinner the upper part of the face only was affected on the side of lesion. This is rare. Though in both cases the focus seems to have affected the issuing seventh, as there was also conjugate paralysis the lesion may have implicated the posterior longitudinal bundle, in which it is probable that the upper facial fibres from the third nucleus descend to the genu of the facial nerve. One side of the face may be paralysed and the other in a state of spasm.

(*b*) Next in frequency to affections of the seventh cranial nerve and limbs is paralysis of the sixth. It is often, as we have seen, affected along with the seventh; but the seventh may escape, as for example in the case of Blocq and Guinon. Usually the hemiplegia, associated with affection of the sixth nerve, does not involve the face; but Raymond records a case of supposed pontine lesion in which there was affection of the sixth on one side, and the face and limbs on the other; and a similar case has been reported by Mills and Zinner. This is a rare form of alternate hemiplegia. There are also cases of paralysis of the face, sixth nerve, and limbs on the side of lesion. When the root of the sixth nerve is implicated there is paralysis of the abducens on that side, with internal strabismus; but if the nucleus of the sixth is affected, there is loss of conjugate movements of the eyes towards the side of lesion, if it is destructive; and conjugate spasm to the side of lesion if it is of an irritative character. In all the cases that have been reported of this affection, the lesion has been in the sixth nucleus, or immediately above it, except in one case reported by Senator, in which the lesion was just below the lower end of the sixth nucleus. Siemerling, however, reports a case of isolated lesion of the sixth nucleus, in which the corresponding internal rectus was not paralysed. Wernicke and Hunnuis assume that there is a special conjugate centre near, but not actually the same as, the sixth nucleus. Various explanations have been given of the mechanism of the conjugate ocular action. The most probable explanation of this conjugate deviation from unilateral lesion of the sixth nucleus is that the cortical fibres for the opposite external rectus, and for the internal rectus of the same side, run down together into the region of the sixth nucleus of the opposite side, having decussated higher up, perhaps in the region of the corpora quadrigemina. Of these fibres some enter the sixth nucleus, and others ascend by the posterior longitudinal bundle into the third nucleus of the same or the opposite side. It is usually assumed that the fibres which ascend in the posterior longitudinal bundle cross over to the opposite third nucleus; but it is unnecessary to assume this decussation if, as Kölliker and Perlia hold, there is a partial decussation of the fibres of origin of the third nerve in the middle line. That the posterior longitudinal bundle contains fibres, interruption of which causes paralysis of the opposite internal rectus, may be regarded as well established, both by the results of experiment and also from clinical cases. Thus, Grubler records a case of tubercle on the left side, immediately under the anterior segment of the floor of the fourth ventricle, which caused paralysis of the right internal rectus without implication of the left abducens. As a rule, in these cases of conjugate deviation of the eyes from lesion of the sixth nucleus the power of convergence is retained.

(*c*) Affections of the fifth nerve are, as we have seen, very common on the side of lesion. The sensory division seems to suffer more than the motor, but not infrequently the two are affected together. The sensory and motor divisions were both affected in the cases reported by Jolly, Bristowe, de Havilland Hall, and in a case of my own; the sensory alone in cases reported by Kolisch, Brasch, Starr, Holmberg, Miles; and the motor alone in the case reported by Moeli and Marinesco, and in another case under my own care. The region of the skin supplied by the fifth usually escapes on the side of the hemiplegia, but it was affected in Bristowe's case, in which there was complete hemianaesthesia, including the face.

Tactile sensibility may be impaired in the region of the trigeminus on the side of lesion, and on the limbs of the opposite side; and thus we get crossed hemianaesthesia, as in the cases reported by Jolly and Miles. The trigeminal anaesthesia affects either the whole of the region innervated by the fifth, or only that supplied by special divisions. Thus in the cases reported by Bristowe, Holmberg, and Brasch, the affection of the fifth was limited to the upper area of distribution. Along with the face, the conjunctiva, nostril, and tongue are often anaesthetic. Cases have been reported of pontine lesion in which tactile sensibility in the region of the fifth was normal, but in which there have been complete analgesia and thermanaesthesia (Starr, Holmberg).

In some cases not only is the eye on the side of lesion anaesthetic, but it may become the seat of neuro-paralytic ophthalmia, as in the cases reported by Bristowe, de Havilland Hall, Miles, and Starr. It is, however, a question whether the trunk of the fifth was not affected in these cases, as the neuro-paralytic ophthalmia is more common in peripheral nerve lesions. When it does occur we may, according to my own and Dr. Turner's experiments (13), assume the existence of an irritative lesion, and not the cessation of a trophic influence.

Taste may be impaired on the side of the trigeminal anaesthesia, as in the cases reported by Kolisch and Bristowe. But in Dr. de Havilland Hall's case, though there was complete paralysis of the fifth, both motor and sensory, taste was normal. Instead of anaesthesia of the fifth there may be hyperaesthesia or neuralgic pains. Thus, in de Havilland Hall's case the earliest symptom was pain referred to the malar branch of the fifth, and the eye became so painful and inflamed as to require enucleation.

Affection of the motor division of the fifth is shewn by paralysis and wasting of the muscles of mastication. In some cases, however, there may be irritative symptoms in the form of trismus.

When the tongue is affected it is usually on the side of the hemiplegia, though it frequently escapes. If the lesion extend down to the medulla, so as to implicate the roots or nucleus of the twelfth, the tongue is affected on the side of lesion. Madame Goukovski records a case of paralysis of the limbs and tongue on the opposite sides, due to softening in the left olive and pyramid, which also affected the roots of the twelfth nerve; a condition of things readily understood from the anatomical relations of these structures.

Bilateral symptoms occur frequently in lesions of the pons, either acute or chronic. Bilateral paralysis is especially common in haemorrhage, and it may affect either the arms or legs or both, though bilateral paralysis of the legs is very rare. Bilateral paralysis is also common in cases of thrombotic occlusion of the basilar artery. In a case reported by Hoppe there was, at first, left hemiplegia, with double paralysis of the sixth and seventh cranial nerves, and twenty-four hours later also right hemiplegia. When the occlusion is confined to the branches of the basilar artery we may, as Sir W. Gowers has pointed out, get successive and irregular paralyses of the limbs and cranial nerves in almost every variety of distribution. In cases of tumour growing to any size in the middle of the pons we may have paralysis of all four limbs and of the tongue, with difficulty of articulation and deglutition, as well as double paralysis of the fifth, sixth, and seventh cranial nerves. At other times the symptoms are quite asymmetrical. Thus, the face may be paralysed on one side, together with all four limbs, the affection being greater on one side than the other; or the limbs may be affected on one side and the face on both. In the case of a tumour originating on the one side the symptoms may at first be unilateral, but ultimately become bilateral with the extension of the disease to the other side. Thus, in the case reported by Blocq and Guinon there was at first conjugate paralysis to the right, followed at a later date by bilateral conjugate paralysis. The tumour involved the right sixth nucleus and pressed on the left, and other similar cases have been reported.

Affections of Hearing.—Affections of hearing are not regarded as common in cases of lesions of the pons. Nothnagel refers to cases reported by Holmberg, Rosenthal, and Huguenin, in all of which hearing was affected on the side of lesion. Of 12 cases of tumour and 5 of softening of the pons, together with 10 others which have come under my own observation, I find that hearing was affected on one or both sides in 5. In Kolisch's case (tumour of the right tegmentum) hearing was affected chiefly on the side of lesion. In Bristowe's case (tuberculous mass chiefly in the left side of pons) hearing was affected especially in the right ear. In this case there was left hemiplegia. In Diller's case, in which there was left hemiparesis, hearing was defective on the left side. In Miles' case (tumour in the left half of the pons) hearing was defective on the side of lesion; and in a case under my own care, in which there was hemiplegia and paralysis of several of the cranial nerves on the right side, hearing was defective in both ears, beginning on the right side; that is, three times exclusively or mostly, on the side of lesion, and twice on the opposite side. In Jolly's case (tumour on the left side of the pons, just underneath the floor of the fourth ventricle) degeneration was found in the tracts now usually regarded as the central paths of the cochlear nerve; namely, the corpus trapezoides, striae acusticae, and crossed lateral fillet. This, theoretically, ought to have been associated with bilateral affection of hearing, which, however, does not appear to have been the case,—a fact difficult to explain, unless we assume that the tumour did not actually destroy the continuity of these tracts.

Besides the actual impairment of hearing, such as occurred in the above-mentioned cases, subjective sensations of sound have also been observed in several instances.

Affections of Sensation.—Affections of sensation are of frequent occurrence in connexion with lesions of the pons. Ladame found hemianaesthesia along with hemiplegia in about one-third of the cases. The hemianaesthesia may include the face and limbs on the same side, which is rare (Kolisch); or the face may be affected on the side of lesion, and the limbs on the other side, which is the common form, according as the lesion is above or below the point of junction of the ascending and descending roots of the fifth nerve (Starr (45)). Or we may get the fifth affected on both sides, and the limbs only on one side, as in Bristowe's case, in which there was anaesthesia of the face on the side of lesion, and hemianaesthesia of the opposite side, including the face. The degree of disturbance of sensation varies from slight diminution up to complete abolition, and all forms of sensibility appear to be about equally affected.

The lesions producing anaesthesia are, according to Nothnagel, usually situated laterally and near the floor of the fourth ventricle. In a later analysis of the recorded cases, Starr (46) by a process of exclusion arrived at the opinion that the sensory tracts in the pons must lie between the deep transverse fibres and the grey matter of the fourth ventricle; that is, in the lemniscus or formatio reticularis.

Moeli and Marinesco, also in an exhaustive paper, in which they describe one new case and examine all those previously recorded, conclude that in all cases in which the fillet and the ventral part of the formatio reticularis were affected there were disturbances of cutaneous sensibility; and that when these parts were not affected cutaneous sensibility was intact. Whence they argue: "We can localise the paths of cutaneous sensibility either in the mesial fillet alone, or in it together with the neighbouring part of the formatio reticularis." As the mesial fillet is the continuation of the posterior columns of the spinal cord which decussate in the inter-olivary layer after passing through the clavate and cuneate nuclei, it follows that this is the tract in which are conveyed the sensory impressions which do not cross in the spinal cord. These, as Dr. Head has shewn, are the impressions of muscular sense, and those underlying tactile dissemination. All the other forms of sensation pass up in the formatio reticularis. Dr. Turner and I have found that complete degeneration takes place in the mesial fillet after destruction of the clavate and cuneate nuclei in monkeys. This lesion caused no impairment in tactile sensibility, but as it caused temporary ataxic symptoms, this would be in harmony with the view that it interrupted the transmission of impressions subserving the muscular sense.

Ataxic Disorders.—Ataxic disorders have been frequently observed in connexion with lesions of the pons, and Nothnagel considered that these may be attributed to pontine lesions as such, apart from implication of the cerebellum or its peduncles. Moeli and Marinesco state that in all cases in which ataxia has been a noteworthy symptom, the middle and ventral parts of the tegmentum have been affected. In almost all these cases there were disturbances of cutaneous sensibility; but Buss and von Leyden have each recorded a case in which ataxia was well marked, but tactile sensibility apparently unimpaired. Goldscheider insists on the relation of ataxia to disturbance of the muscular sense, and records a case of ataxia with loss of so-called muscular sensibility due to lesion of the inter-olivary tracts between the roots of the twelfth nerve and the raphe. Reinhold has also recorded a case of focal lesion in the right side of the medulla associated with ataxia of the right arm, which he attributes to implication of the fibres of the fillet arising from the right post-columnar nuclei. Many other similar cases might be mentioned in favour of ataxia being specially related to lesion of the fillet.

The gait has been frequently described as of an uncertain or reeling character, similar to that which is seen in connexion with cerebellar disease. Many cases of this kind might be referred to. In Penzoldt's case there was, besides the reeling gait, a tendency to retropulsion; and in Ewald's case there were also pendulum-like oscillations of the limbs. In a case reported by Williams (gliomatous affection of the whole pons) the patient would tumble about, fall forwards, or spin round like a top. These disturbances of station and locomotion may be partly due to vertigo, with which they are often associated; or both symptoms may be due to the same cause, namely, implication of the cerebellum or its peduncles. As nearly all the well-marked cases of this affection have been of the nature of tumour, an indirect influence on the cerebellum must always be assumed as probable. Choreic movements, or jerky tremors similar to those seen in disseminated sclerosis, have also been noted in connexion with lesions of the pons, and are not characteristic of pontine lesions as such.

Affections of Articulation and Deglutition.—Affections of articulation and deglutition, particularly the former, are common in connexion with lesions of the pons; they are usually well pronounced in occlusion of the whole basilar artery, as in a case reported by Hoppe.

Impairment of articulation in pons lesion is, without doubt, due to affection of the central fibres of the hypoglossal; but dysphagia, when it occurs, is probably due to indirect implication of the medulla oblongata; or is a secondary result of the impairment of the movements of articulation which form the first part of the act of deglutition.

Anarthria occurs more particularly when the pontine lesion is bilateral. Unilateral lesions of the pons may exist without any impairment of articulation. From an analysis of 27 recorded cases Markowski finds that in 18 of unilateral lesion there were 10 in which there was no dysarthria; of these, 7 were lesions of the right half of the pons, and 3 of the left. In the remaining 8—or, on more critical analysis, 6—impairment of articulation was observed. In 9 cases of bilateral lesion there was only 1 in which anarthria did not occur. The conclusion, therefore, from these facts is that bilateral lesion of the pons almost always, and unilateral in less than one-half of the cases, cause impairment of articulation. The results are the same whether the lesion is in the right or in the left side; so that the view of Wernicke, that the articulatory tracts run specially in the left half of the pons, is not substantiated. The paths of articulation appear, from Markowski's observations, to run specially in the medio-dorsal portion of the pyramid. In his own case, in which there was well-marked dysarthria and dysphagia, there was total destruction of the left pyramid, and only a small focus in the medio-dorsal part of the right pyramid. He quotes a similar case, reported by Raymond, in which there were two small foci in the middle line of the pons which could only have injured the medio-dorsal part of the pyramidal tracts.

Hyperpyrexia.—Many other symptoms occur in connexion with pontine lesions, but are not peculiar to them. There is one, however, which deserves special mention, namely, hyperpyrexia. This occurs more particularly in connexion with sudden lesions, such as haemorrhage. The temperature not infrequently rises above the normal within an hour of the onset of haemorrhage, and speedily rises to 104° or 106° F., or even higher, before death. In a case reported by Barr it rose to 109°. A similar rise may occur in sudden obstruction of the basilar artery, preceded by an initial fall, such as not infrequently occurs at the onset of cerebral haemorrhage. Dr. Bastian reports a case of this kind in which there was an initial fall to 95° F. in the rectum. In chronic lesions of the pons, such as tumours and softening, there may be no material alteration of the temperature. In some cases it is actually subnormal.

The Pupils.—The state of the pupils has been already specially referred to in connexion with the symptoms of haemorrhage into the pons. In pontine haemorrhage the pupils are usually minutely contracted, as in opium poisoning. This, however, is not universal, and the state may vary according to the period at which they are observed. Thus, in a case reported by Edridge Green the pupils were at first minutely contracted, but they became dilated before death; and they were normal in a case recorded by Rorie.

Glycosuria and albuminuria have sometimes been observed in connexion with lesions of the pons, more frequently, perhaps, in cases of haemorrhage. It is doubtful if any causal relationship has been satisfactorily established between these symptoms and lesions of the pons as such. Lesions of the pons frequently occur in connexion with chronic renal disease and arterial sclerosis; so that the albuminuria, in some instances at least, has been pre-existent; and glycosuria may sometimes be found in other lesions of the nerve centres.

Summary.—Lesions of the pons Varolii are very varied in their symptomatology. The most characteristic indications are a combination of paralysis of some of the cranial nerves, especially the seventh, sixth, and fifth on the side of lesion, and of the limbs on the other.

REFERENCES

1. Barr. *Lancet*, 1890, i. 750.—2. Bastian. *Trans. Clin. Soc.*, 1885, xviii. 193.—3. Bennett and Savill. *Brain*, 1889-90, xii. 102.—4. Blocq et Guinon. *Arch. de méd. exp. et d'anat. path.*, 1891, iii. 74.—5. Bode. *Die Haemorrhagien der Varolsbrücke*, Würzb., 1877.—6. Brasch. *Neurol. Centralbl.*, 1892, xi. 226.—7. Bristowe. *Brain*, 1891, xiv. 289.—8. Buss. *Deutsch. Arch. f. klin. Med.*, 1887, xli. 241.—9. Diller. *Amer. Journ. Med. Sc.*, 1892, civ. 509.—10. Duret. *Traumatismes cérébraux*, Paris, 1878.—11. Elzholz. *Jahrb. d. Wien. Krankenanst.*, 1892, i. 635.—12. Ewald. *Deutsch. Arch. f. klin. Med.*, 1877, xix. 591.—13. Ferrier and Turner. *Brain*, 1895, xviii. 231.—14. *Idem. Phil. Trans.*, 1894, B. clxxxv. Part ii. 719.—15. Goldscheider. *Charité-Ann.*, 1891, xvi. 162.—16. Goukovski. *Nouv. icon. de la Salpêtr.*, 1895, viii. 178.—17. Gowers. *Brain*, London, 1883, v. 137.—18. Green, Edridge. *Brit. Med. Journ.*, 1895, i. 1035.—19. Grubler. *Gaz. hebdom.*, 1858, v. 721; 1859, vi. 3.—20. Hall, de Havilland. *Lancet*, 1895, ii. 263.—21. Holmberg. *Neurol. Centralbl.*, 1893, xii. 390.—22. Hoppe. *Berl. klin. Wchnschr.*, 1893, No. 10.—23. Hunnuis. *Brückenerkrankungen*, 1881.—24. Joffroy. *Bull. et mém. Soc. méd. d. hôp. de Paris*, 1885, 3. s., ii. 279, 1885.—25. Jolly. *Arch. f. Psychiat.*, 1894, xxvi. 619.—26. Kolisch. *Wien. klin. Wchnschr.*, 1893, vi. 14.—27. Ladame. *Symptomat. und Diagnose der Gehirngeschwülste*, 1865.—28. Markowski. *Arch. f. Psychiat.*, 1892, xxiii. 367.—29. Mickle. *Brit. Med. Journ.*, 1881, ii. 151.—30. Mierzejewski und Rosenbach. Reference in *London Med. Rec.*, 1886, xiv. 338.—31. Miles. *Arch. of Med.*, 1881, vi. 196.—32. Mills and Zinner. *Journ. Nerv. and Ment. Dis.*, 1894, xxi. 688.—33. Moeli und Marinesco. *Arch. f. Psychiat.*, 1892, xxiv. 655.—34. Nothnagel. *Top. Diagnose der Gehirnkrank.*, 1879.—35. Oppenheim. *Charité-Ann.*, 1883, x. 342.—36. Penzoldt. *Berl. klin. Wchnschr.*, 1876, xiii. 545.—37. Porter. *Lancet*, 1891, i. 1384.—38. Raymond. *Neurol. Centralbl.*, 1886, v. 404.—39. *Idem. Rev. neurol.*, 1895, iii. 193.—40. Reinhold. *Deutsche Ztschr. f. Nervenh.*, 1894, v. 351.—41. Rorie. *Journ. Ment. Sc.*, 1890, xxxvi. 354.—42. Russell and Taylor. *Brain*, 1890, xiii. 88.—43. Senator. *Berlin. psych. Gesellsch.*, July 9, 1883.—44. Souques. *Nouv. icon. de la Salpêtr.*, 1891, iv. 358.—45. Starr. *Med. Rec.*, N.Y., 1893, xliii. 168.—46. *Idem. Journ. Nerv. and Ment. Dis.*, 1884, xi. 327.—47. Wernicke. *Fortschr. d. Med.*, 1884, ii. 1.; *Gehirnkrankheiten*, 1881.—48. Williams. *Bristol Med.-Chir. Journ.*, 1891, ix. 163.

Lesions of the Cerebellum.—The cerebellum is liable to the same diseases as affect the other encephalic centres.

Haemorrhage.—Haemorrhage occurs much less often in the cerebellum than in the cerebrum, a fact which is in accordance with the relatively greater frequency of miliary aneurysms in the cerebrum. In 100 cases of cerebellar disease quoted by Krauss there was only one of haemorrhage. In many cases the extravasation is large and occupies the greater portion of one hemisphere, as well as part of the middle lobe.

According to Duret, the great effusions come from the chief stem of the branch of the superior cerebellar artery to the dentate nucleus. Small circumscribed foci of haemorrhage are comparatively rare. They are for the most part central, and usually implicate the nucleus dentatus. Haemorrhages restricted to the vermis appear to be exceptional (Nothnagel). Haemorrhage into the inner and hinder portion of the hemisphere may occur from rupture of a branch of the posterior cerebral artery (Gowers). Haemorrhagic extravasation may burst into the fourth ventricle and may fill all the ventricles, as in a case reported by Barling. The superior and inferior cerebellar peduncles are rarely the seat of haemorrhage; but extravasation may occur in the middle peduncle, generally from a haemorrhage primarily originating in the side of the pons; and conversely a haemorrhage into the cerebellum may extend into the side of the pons. In 10 cases of cerebellar haemorrhage, to which I have been able to refer, 5 were in one hemisphere, 3 were in the middle lobe and also in one or both hemispheres. In Mills' case the cerebellum, pons, and crura were quite ploughed up; and in Campbell's (24) case 3 there was perivascular haemorrhage in both corpora dentata and in the adjoining white matter. In 3 cases extravasation had occurred into the fourth ventricle.

Symptoms of Cerebellar Haemorrhage.—There may be premonitory symptoms, such as headache and giddiness for two days before the onset, as in Mills' case; and Ratcliffe has noted, as of probably some significance, an involuntary movement of the head to one side. When the haemorrhage occurs there is usually coma, as in intracranial effusions generally. Often it is sudden; this is so in half the reported cases, but the coma may be delayed, or, according to the observations of Ratcliffe, may cease and reappear several times. Pain in the head, especially occipital, is not infrequently a marked and early symptom. Vomiting is frequent and often very persistent; the symptom is so marked, that, according to Nothnagel, cerebellar haemorrhage may be expected if at the beginning there is repeated vomiting without any obvious motor paralysis. But haemorrhages of considerable extent have taken place without any symptoms during life (Oliver). Hemiplegia may or may not occur. Its absence is generally regarded as more typical than its presence. In the 10 cases to which I have referred there was no paralysis in 3; crossed hemiplegia in 1 (Farquharson); direct hemiplegia in Thacker's case; weakness of the arm on the side of lesion, and paresis of the face on the opposite in Friedeberg's case; and left hemiplegia in Mills' case, in which the cerebellum, pons, and crura were all torn up and disorganised. The state of the pupils is somewhat variable. Generally they are contracted and fixed, and in

Barling's case they were contracted at first, but became dilated before death. Not infrequently, in connexion with haemorrhage into the middle lobe, priapism has been observed, due without doubt, as Segalas has pointed out, to irritation of the subjacent pons-medulla, and not to any direct connexion between the middle lobe and the generative organs, as supposed by Serres.

Haemorrhage into the cerebellum is often very rapidly fatal, with severe respiratory symptoms; especially if effusion take place into the fourth ventricle. If recovery ensue the pressure symptoms pass away, and there remain only, with greater or less persistence, the symptoms specially characteristic of cerebellar lesion.

Softening.—Necrotic softening of the cerebellum from vascular occlusion, embolic or thrombotic, is rare. Even if a cerebellar vessel does become occluded the softening is comparatively limited, owing to the rich anastomosis between the cerebellar vessels. Softening is most common in the regions supplied by the posterior cerebellar artery, often secondary to occlusion of the vertebral, but sometimes originating in the posterior cerebellar, and extending downwards into the vertebral artery, as in a case reported by Menzies.

Abscess.—Abscess frequently occurs in the cerebellum, and, for the most part, in connexion with disease of the middle ear. Winter and Deanesly collected 23 cases of cerebellar abscess, and found purulent otitis media in all. In 19, which I have myself analysed, there was middle-ear disease in 16. In all these the abscess was situated in the hemispheres. In Winter and Deanesly's collection the abscess was situated in the anterior part of the lateral lobe in 21; in the inferior surface of the middle lobe in 1; and in the flocculus in 1. In a case reported by Friedeberg there was an abscess in each hemisphere.

The time that elapses between the onset of the otitis media and the development of cerebellar abscess is very variable—between three weeks and several years. Cerebellar abscess may also result from traumatic lesion and septic infection, as in a case reported by Krauss.

Tumours. — Tumour occurs very frequently in the cerebellum. Ladame in 1865 had collected 77 cases; Bernhardt between 1865 and 1881 had collected 90 cases; and Wetzel between 1881 and 1889 collected 59 cases. I have collected, mainly between 1890 and 1899, records of 53 cases, of which 8 are original and unpublished. Besides the 53 cases of tumour proper, 11 cases of cyst might be included. Simple cysts are not common. Most of the so-called cerebellar cysts are merely cystic degeneration of tumours; and Williamson rightly points out that a cerebellar cyst can only be classed as simple or serous when minute examination shews no trace of hydatid disease, cysticercus, haemorrhage, or tumour. He has himself recorded 2 cases of apparently simple serous cyst; but histological examination shewed gliomatous or glio-sarcomatous tissue in the walls of both, so that they were merely tumours, the proper tissue of which had almost completely disappeared.

Atrophy.—Atrophy of the cerebellum is not uncommon, and varies greatly in degree. The whole organ may be entirely atrophied, as in the much-quoted case of Alexandrine Labrosse, and a similar case, under the care of Dr. Shuttleworth, described by myself; or only one, or both hemispheres may be affected, the middle lobe being normal or relatively so. Atrophy of one hemisphere is usually consecutive to long-standing disease of the opposite cerebral hemisphere. Atrophy of the cerebellum may be due to arrest of development in intra-uterine life, of which the cause is unknown; it may be primary and familial, or it may be secondary to cerebral inflammation, generally meningitic (Hammarberg), in which case, in addition to atrophy, there is also sclerosis of a much more pronounced character than is seen in primary atrophy. Hypertrophy of the cerebellum has been described as occurring in cases of imbecility. Doursott has recorded eight such cases, in which the weight of the cerebellum and pons varied from 200 to 256 grams.

Symptomatology of Cerebellar Disease.—Diseases of the cerebellum vary in their symptomatology with the time at which they are observed, the rate of their development, the position in which they are situated, and according as they affect the cerebellum alone or implicate directly or indirectly neighbouring centres, tracts, or nerves.

The most characteristic symptoms are an instability of station and locomotion, similar to that of alcoholic intoxication in different degree.

These symptoms are always seen in mammals immediately after the infliction of lesions on the cerebellum, as originally described by Flourens, but they tend to diminish with time. I may here quote the description which I have elsewhere given of the effects of cerebellar lesions, as observed by Dr. Turner and myself:—

"When the cerebellum has been totally removed from monkeys—an operation which we have found to be a most formidable one, and more frequently fatal than successful—the animals exhibit the most tumultuous disorders of equilibrium, so that station and locomotion are, for the time, altogether impossible. Gradually, however, these tumultuous disorders subside to such an extent that the animal is able to sit up, with the help of some support to which it clings, and ultimately regains such a degree of stability that it may dispense with extraneous aid, but is so tottery that it falls over on the slightest disturbance or excitement. Its gait is of a peculiar sprawling character, at first barely raising its abdominal surface from the ground, and planting its limbs wide apart so as to increase the basis of support; these being in progression raised and set down in a brusque and characteristically ungraduated fashion, which character the movements of the limbs retain for an indefinite period. Notwithstanding this extraordinary unsteadiness and instability of equilibrium, the animals are, however, able to grasp tenaciously with both hands and feet, so much so that it is sometimes more easy to drag the chair to which they may cling than loosen their grip; and they are able to climb a rope with agility hand-over-hand, in no way differing in this respect from perfectly normal animals. But the most noteworthy and persistent feature is the remarkable astasia or unsteadiness of the head, trunk, and limbs, which are either agitated by constant fine tremors apart from obvious muscular exertion, or exhibit oscillations of the typical disseminated sclerosis type on volitional exertion; so that, for instance, an attempt to lay hold of a piece of fruit excites such wild oscillations of the hand and arm that the object aimed at is either knocked away or if seized, after many unsuccessful efforts, is with the utmost difficulty brought up and held to the mouth. This instability is a persistent feature, and though lessening to some extent, never entirely disappears, and was clearly evident in one of Luciani's monkeys a whole year after the operation.

"If only the lateral lobe, or one-half of the cerebellum, is removed, the persistent symptoms, after the cessation of the primary disorders, namely, the sprawling, ungraduated action of the limbs, and the tremors of instability on volitional effort, are confined to the same side as the lesion.

"If, on the other hand, the middle lobe is destroyed, or extensively injured, as by antero-posterior division, the symptoms are essentially of the same character as those which follow destruction of the whole organ, but they do not affect one side more than the other, and, according to our observations, are more pronounced in the head and trunk than in the limbs. They also appear to be less persistent, and, in the course of a few months, pass off to such an extent that, except on careful examination, it would be difficult to distinguish an animal so operated upon among its normal companions."

The disorders of station, locomotion, and movements in general have been variously explained. Luciani, whose views have met with much acceptance, is of opinion that the cerebellum normally exerts on the apparatus of movement a *sthenic*, *tonic*, and *static* influence, so that destructive lesions or defects of the organ exhibit themselves in a condition of *asthenia*, *atonia*, and *astasia*. According to Dr. Hughlings Jackson, "all the muscles of the body are innervated both by the cerebrum and cerebellum, but in an inverse order. The cerebellum regulates the muscular contractions necessary for our attitudes in space, while the cerebrum regulates the contractions necessary to effect all changes of attitude which are made in response to successive impressions occurring in time. . . . Speaking broadly, then, the cerebellum regulates continuous or tonic muscular contractions. It will be seen, therefore, that every combined muscular adjustment necessitates the co-operation of both these organs; no change of attitude can be effected by the cerebrum except in so far as a certain attitude was previously maintained by the cerebellum, and no steady movements can be produced by the alternate contractions of some groups of muscles, except in so far as other groups of muscles are maintained in a state of continuous contraction. Hence it may be inferred that all movements of the body are co-ordinated both in the cerebellum and cerebrum." The cessation of the tonic or continuous innervation by the cerebellum should, according to this hypothesis, exhibit itself in an unsteady and ungraduated character of all the motor adjustments, whether of the body as a whole or of its individual parts, and should theoretically induce a state of affairs which actually agrees well with the effects of cerebellar lesions in animals.

In Professor Sherrington's phraseology the cerebellum is the head ganglion of the *proprio-ceptive* system, *i.e.* of afferent impressions generated in the organism itself, chiefly from the labyrinth and from the muscles of the trunk and limbs. These are conveyed to the cortex of the cerebellum by the cerebello-petal tracts in the inferior and middle peduncles. From the cortex impulses pass to the central nuclei, whence emerge the efferent tracts (Horsley and Clarke) which, through the superior peduncles and red nuclei and the vestibulo-spinal tracts, influence the apparatus of movement. The influence of the cerebellum is mainly homolateral, whilst that of the cerebrum is mainly heterolateral—each cerebral hemisphere acting in conjunction with opposite cerebellar lateral lobe.

An analysis of the facts of the different forms of cerebellar disease shews that the characteristic symptoms have been observed only in a certain proportion of the recorded cases. For the most part the negative cases have been those of slow growth or long standing, in which the disease was confined to one hemisphere. The characteristic disturbances are manifested most frequently when the middle lobe is affected. On this fact Nothnagel has founded the conclusion that the middle lobe alone exercises those functions which we ascribe to the cerebellum; and that on lesion of this lobe, directly or indirectly, the symptoms of cerebellar disease exclusively depend. This view receives no support from the phenomena of experimental lesions, as has been shewn by Luciani, and Turner and myself. Indeed, as has been mentioned above, the effects of destruction of the middle lobe are not more pronounced and are not more enduring than those which follow extirpation of the lateral lobe. A lesion of the middle lobe is more calculated to affect the organ as a whole than one situated in the lateral lobe; and the standard by which it is sought to determine that the middle lobe is really implicated in all cases in which the characteristic symptoms are manifested is a purely arbitrary one. The apparent discrepancy which exists between the many negative cases of cerebellar lesion in man and the uniformly positive results of experimental lesions in the lower animals is largely, if not entirely, explicable by the principle of compensation effected both by the other parts of the cerebellum and by the higher centres. This principle, which I enunciated many years ago, has been experimentally verified by the researches of Luciani. The disturbances of equilibrium are always most marked immediately after the infliction of the injury on the cerebellum. This may be accounted for by the suddenness of the derangement of the self-adjusting mechanism on which the maintenance of the equilibrium mainly depends. As, however, the loss of this mechanism may be to some extent compensated by conscious effort, the animal acquires in process of time the power of voluntary adaptation by means of its cerebral centres, and is thus enabled to maintain its equilibrium, though with a less degree of security than before. The more extensive the lesion, the greater the disturbance and the greater the difficulty of effecting by conscious effort all the muscular adjustments which are necessary to maintain the balance. Hence, even when the utmost degree of compensation has been achieved, a greater or less degree of instability and unsteadiness of movement and an incapacity for prolonged exertion are manifested. When the cerebellum is extensively diseased, or largely atrophied, all the motor adjustments concerned in equilibration, formerly easy and automatic, are now performed by the voluntary centres under a laborious sense of strained attention and conscious effort. No compensation takes place if, as Luciani has shewn, the motor centres of the hemispheres are destroyed along with the cerebellum itself. Hence, congenital defect, or a slowly progressive lesion, affords the most favourable conditions for the supplementation by the higher centres of the automatic mechanism, which is originally deficient or gradually undergoing degeneration; and, as a rule, it has been found that congenital defect or extensive lesion of the cerebellum in early life has been associated with a tardy acquisition of station and locomotion; indeed it is questionable if perfect compensation is attainable.

We may now proceed to inquire how far the results of physiological experiment are in harmony with those of clinical observation.

The most characteristic symptom of cerebellar disease in man is a reeling, staggering, or drunken gait—the so-called titubation or cerebellar ataxia. The equilibrium is unsteady, and there is a continual tendency to stumble or fall over the most trifling obstacles, particularly on hurried movements or change of position. The patient, however, who is unable to stand or walk without assistance, may be capable in the recumbent position of making the finest volitional movements with force and precision. There is no true inco-ordination, as all the muscles concerned in any particular act co-operate together in perfect harmony. They have none of the brusque, irregular, and sprawling character of locomotor ataxia. But, though these symptoms are pathognomonic of cerebellar disease, clinical records undoubtedly shew that in a large number of cases of organic disease, or degeneration, of the cerebellum, they have not been observed.

In the first edition of this work a tabular abstract was given of all the cases of cerebellar atrophy that had been recorded since the publication of Nothnagel's well-known work on *Regional Diagnosis of Cerebral Disease*. It has not been considered necessary to reproduce this, or to analyse in detail all the cases that have been recorded since. There is no doubt, as Dr. Gordon Holmes has shewn, that under the same head have been included cases differing greatly in their pathogenesis. He classifies them under four principal heads:—(1) Primary parenchymatous degeneration of the cerebellum; (2) Olivo-ponto-cerebellar atrophy; (3) Progressive cerebellar disease due to vascular or interstitial lesions; (4) Acute cerebellar lesions. In these four groups, all cerebellar diseases, apart from tumours and focal lesions of vascular origin, may be included. In the following two groups the cerebellum is not primarily affected, though their most prominent symptoms have been described as those of cerebellar disease, viz. :—(5) Degeneration of the spino-cerebellar tracts, the cerebellum being normal or small only ; (6) Congenital smallness of the central nervous system associated with cerebellar symptoms.

Of the first type the best instances have been published by Dr. Gordon Holmes. In a family of eight, four presented the same symptoms which set in in early middle life, and progressed slowly till death at an advanced age. The first and most prominent symptom was a reeling or staggering gait like that of a drunk person, irregularity in or uncertainty of the movements of the arms, hesitating, scanning or explosive articulation, tremor of the head and limbs, nystagmus or irregular nystagmoid jerkings of the eyes. There was no defect of vision or hearing, and no motor paralysis or sensory defect. The tendon-reflexes were unaffected. The sphincters were intact, and there was no mental impairment. To the same category belong the cases of a brother and sister reported by Fraser, a case reported by Thomas, and another by Mingazzini. A similar condition has also been observed by Drs. Herringham and Andrewes in cats, and by Dr. Risien Russell in a puppy of a litter all similarly affected with an ataxic reeling gait, and constant oscillation of the head and trunk while at rest.

The second type of cerebellar degeneration originally described by Thomas, under the title olivo-ponto-cerebellar atrophy, is characterised anatomically by atrophy of the cerebellar cortex, of the olivary bodies and grey matter of the pons ; total degeneration of the middle cerebellar peduncles and partial degeneration of the restiform bodies, with practical integrity of the central nuclei. It is neither hereditary nor familial, it comes on at an advanced age and progresses slowly (*vide* also Vol. VII. p. 785).

The most prominent symptom in Thomas' case (109) was a drunken reeling gait ; in two of the cases the arms were normal in their movements, but in a third there was intention-tremor. Articulation was slow and scanning in all three cases, and nystagmus existed in two. Other cases similar to those described by Thomas have been reported by other clinical observers.

The third form of degeneration is that due to progressive vascular and interstitial lesions. In Schultze's case the onset occurred at the age of thirty-nine. Gait became of the drunken type, articulation was slow, and there was intention-tremor of the arms, with slight nystagmus. The symptoms progressed rapidly and death took place at the age of forty-three. There was considerable atrophy and sclerosis of the cerebellum and brain-stem. The Purkinje cells of the cerebellar cortex were scanty, and those which remained were atrophied ; the molecular and granular layers were less affected. The lesions were diffuse, with much secondary neuroglial sclerosis. The central nuclei were atrophic, and the superior peduncles degenerated. There was also degeneration, probably secondary, of the olives and transverse fibres of the pons. The spinal cord was normal except for slight pallor of the pyramidal tracts. Other cases somewhat similar have been reported by Dr. Michell Clarke, Southard, and Catòlo. In Catòlo's case the symptoms commenced at the age of thirty-eight after an attack of cholera. They were an ataxo-cerebellar and spastic gait, tremor of the head, intention-tremor of the limbs, nystagmus, diplopia, scanning speech, and increase of the tendon-reflexes. The post-mortem appearances were atrophy of the brain-stem and cerebellum, diminution of the Purkinje cells and patches of sclerosis throughout the cerebellum and brain-stem. There was also partial atrophy of the olivary bodies and pons, and pseudo-system-degeneration of the ventro-lateral columns of the cord. This seems to me to have been a case of disseminated sclerosis specially affecting the cerebellum and its peduncles, and is of interest more particularly in its symptomatology.

Under the fourth head Holmes describes cases of acute cerebellar symptoms, such as those described by Batten and others, as occurring frequently in children after zymotic diseases. The child seems to be making a normal convalescence, but when made to sit up in bed is unable to maintain his balance. He has marked inco-ordination of the hands, and if placed on his feet is wildly unsteady. There is also affection of speech. Recovery gradually ensues, but in most cases takes several months, and it may be two or three years. The case reported by Hammarberg seems to have belonged to this category, and several similar cases have been described by others. In the majority of the cases with acute onset the disease seems to have consisted in diffuse or focal lesions of the nature of encephalitis, whilst in others it may have been due to the action of toxins.

Under the title Hereditary Cerebellar Ataxia (hérédo-ataxie cérébelleuse) Marie described an affection which he considered dependent on atrophy of the cerebellum. He founded his nosological classification on certain cases observed by himself, together with others reported by Fraser, Nonne, Sanger Brown, etc. As the name denotes the disease is hereditary, and tends to affect several members of a family. The leading symptoms are of the cerebellar type, viz. difficulty in maintaining the equilibrium, and an unsteady, reeling gait. The resemblance between the symptoms and those caused by experimental lesions and diseases of the cerebellum, indicated, according to Marie and Londe, that the cerebellum was the seat of the disease. Dr. Gordon Holmes, however, has shewn that the cases which have been included in Marie's disease are not characterised by constancy in their clinical symptoms, morbid anatomy, or pathogenesis. The disease was hereditary in the cases described by Sanger Brown, and by Klippel and Durante, but there was no similar case in the ascending or collateral lines of the families observed by Fraser and Nonne. The morbid anatomy has now been determined in one or more members in each family. From this it appears that in the cases described by Klippel and Durante, and in the remarkable series of 21 cases observed in four generations of the same family reported by Sanger Brown, the structure of the cerebellum was normal, but there was degeneration of the dorsal and spino-cerebellar tracts of the spinal cord.

In two of the three brothers described by Nonne no pathological changes were found, but in both the cerebellum was abnormally small, and in one of them the whole central nervous system was considerably below the normal size. Only in Fraser's case, previously referred to under the head of primary parenchymatous degeneration, was there clear evidence of acquired cerebellar disease.

It would appear, therefore, that there is no form of cerebellar disease to which the term Hereditary Cerebellar Ataxia is correctly applicable. The majority of the cases of progressive cerebellar disease belong to Thomas' group—olivo-ponto-cerebellar atrophy, but in none of those published were the symptoms either hereditary or familial, and in almost all the onset occurred in advanced life. The best examples of familial degeneration strictly limited to the cerebellar cortex are those which have been described by Dr. Gordon Holmes.

Cerebellar Tumours.—The following account of the symptomatology of cerebellar tumours is based mainly on the careful analysis by Drs. Stewart and Holmes of a series of 40 cases occurring during the last few years in the National Hospital for the Paralysed and Epileptic. The diagnosis was confirmed by necropsy or operation in 22 cases, and all cases were excluded in which the disease invaded other parts, or were such as to induce complications. Cerebellar tumours are either *intra-cerebellar or extra-cerebellar*; the latter are tumours occupying the angle between the cerebellum and pons, and arise either from the ventral aspect of the cerebellum or grow from the adjacent cranial nerves. The symptoms in both forms are essentially the same *qua* the truly cerebellar functions, but in extra-cerebellar tumours there are more often complications dependent on pressure or irritation of the cranial nerves or motor tracts of the pons. In the cases carefully investigated in the National Hospital there has rarely been a total absence of the characteristic symptoms of cerebellar lesion as determined by experiments on animals.

Affections of Motion.—The *attitude* in many cases of unilateral cerebellar tumour is peculiar. When the patient sits or stands, the head is held slightly inclined to the side of lesion and the occiput approximated to the shoulder, while the chin is directed towards the opposite shoulder. This, however, is not constant, and the position of the head seems to be the reverse of that seen in monkeys after extirpation of the lateral lobe. In some cases of tumour of the middle lobe there may be a tendency to retraction of the head; in others the head may be bent forwards on the chest. In a large proportion of cases there has also been lordosis in the dorso-lumbar region on assuming the erect position.

The *gait* is of the characteristic drunken reeling type. When more carefully analysed, it seems to comprise a tendency to stagger and stumble to the side of the lesion and a tendency to deviate from the desired direction towards the side of lesion. The patient in walking suddenly totters as if drawn by some unseen influence, and makes a circle towards the side of lesion with his shoulder pointing towards the centre of the circle. But when the disease has been of considerable duration, he endeavours to correct this tendency and frequently overdoes it, so that he bends from the direct line towards the sound side. In most cases the patient holds the leg on the side of lesion more abducted than its fellow and rotated outwards at the hip, so that in this way the base is widened. He can also stand less steadily on this leg than on the sound side. When the feet are placed together, the tendency is to fall backwards and to the side of lesion. In mesial tumours, or those involving both lateral lobes, the gait is more drunken and ataxic, and the patient stumbles and reels from side to side. In these cases the tendency is to fall forwards or backwards rather than to either side.

Drs. Stewart and Holmes have also observed, in accordance with the observations of Luciani in animals, a very decided loss of power or *asthenia* on the side of the lesion; this is sometimes called paresis, but the term in my opinion is a misnomer, seeing that it affects the proximal and distal muscles alike. In paresis proper, or partial paralysis, the distal movements are more affected than the proximal. The asthenia is always on the side of lesion. It is not due to indirect lesion of the pyramidal tracts. The superficial reflexes are normal, and there is no degeneration discoverable in the pyramidal tracts. The weakness is relatively greater in acute lesions, and especially after extensive destruction of cerebellar tissue by operation. After operations there is often a condition of general muscular asthenia, but always more evident on the homolateral side. The opposite limbs speedily regain their normal strength, but the limbs on the side of lesion may remain weak for a considerable period, but not to any great degree. The trunk muscles are notably affected, and the patient may be unable to sit up from inability of the spinal muscles to bear the weight of the body. Occasionally the weakness is decidedly more obvious on the side of the lesion. The asthenia on the side of lesion is always more marked in intra-cerebellar than in extra-cerebellar tumours. In the latter case there is sometimes distinct spastic paresis of the opposite limbs with alterations of the reflexes characteristic of organic hemiplegia. It is always due to pressure and degeneration of the pyramidal tracts in the pons.

Associated with the unilateral weakness there is often a defective tone or *atonia* of the homolateral limbs, as described by Luciani. The limbs are limp or flaccid, so that when shaken the distal segments flop like a flail apparently unrestricted by any active or passive tone of the muscles. The greater degrees of this hypotonia are met with in cases of acute onset, and especially after operations in which the cerebellum has been considerably damaged.

The state of the so-called tendon-reflexes in cerebellar disease is not a measure of the tone of the muscles as it is in tabetic affections. The variability of these reactions is a remarkable feature of cerebellar lesions. In some cases they are increased, whilst in others they are diminished or lost, and any case may present both extremes within short periods. In unilateral cerebellar tumours Stewart and Holmes found diminution on the side of lesion most common, though an increase on both sides or on the side of lesion was often seen. In extra-cerebellar cases the tendon-reflexes were rarely diminished; in some they were normal, but the rule is that they are increased, especially on the side opposite the lesion. In cases of spasticity due to pressure on the pyramidal tracts ankle-clonus may be present on the opposite or on both sides. In all the cases of operation the tendon-reflexes were absent for some days after the operation. This coincided with the extreme asthenia and atonia which was also observed at the same time, but the reappearance of the reflexes was not synchronous with improvements in the muscular tone.

Dr. Turner and I found that after experimental ablation of the cerebellum in monkeys there was no appreciable alteration of the reflexes, but they gradually became increased. After extirpation of a lateral lobe the tendency was for the knee-jerk to become increased on the opposite side. Russell found in rabbits and dogs increase of the knee-jerk on the side of lesion and diminution on the opposite side. Later the knee-jerk on the same side became less active until they became nearly equal. In the cases of cerebellar atrophy reported by Holmes the knee-jerks were brisk. It appears, therefore, that in cerebellar lesions and degenerations of almost any extent or position the knee-jerks may be normal, diminished, or increased, and may vary at different times in the same case. Thus, in a case under my own care of carcinoma of the left side of the cerebellum, the knee-jerks were at first equal and normal, at a later date they were somewhat increased, and still later very slight. In another case in which there was a cyst of the left lateral lobe excavating the middle portion of the right lobe, the knee-jerks were equal and normal; at a later date they were diminished; still later they were active and equal, and ultimately the left was almost gone with a tendency to ankle-clonus.

Drs. Stewart and Holmes have also recorded the occurrence of a *dysmetric* or *ataxic* condition of the limbs in cerebellar tumour and especially after operative procedure. It consists in a deficient control of the various component muscular contractions by which a complex act is executed, and in a disturbance of the normal reciprocal innervation of the muscles and their antagonists. It is not increased like the ataxia of tabes when the eyes are shut. It is most marked on the side of lesion, but may occur on both sides, though always less on the opposite side. When the tumour is in the middle lobe or extends into both lateral lobes the inco-ordination is bilateral, but greater on the side of the greater lesion of the lateral lobe. It affects the arm more than the leg.

Babinski has described as pathognomonic of cerebellar disease a symptom to which he has given the name of *dys-diadocho-kinesia.* This consists in the inability of the patient to perform with precision rapid alternate movements, such as those of pronation and supination of the wrist. The arm on the side of lesion performs these movements awkwardly and irregularly. This symptom was observed in cases both of intra- and extra-cerebellar tumours, but it was not constant, nor can it be considered altogether pathognomonic. It apparently depends on the same conditions as give rise to the dysmetria or ataxia above described.

One of the most striking symptoms following extirpation or extensive lesion of the cerebellum in monkeys is the *astasia* or unsteadiness of the head, trunk, and limbs after the primary tumultuous disorders of equilibrium have passed away. If only one lateral lobe is destroyed, this astasia occurs specially in the limbs on the side of lesion. There is either a constant fine tremor apart from obvious muscular exertion, or oscillation of the disseminated sclerosis type on volitional movement. Dr. Turner and I found these tremors to be most marked in the upper limb, and that they tend to subside in course of time. This astasia is also a prominent symptom in all cases of familial degeneration of the cerebellum, as has already been mentioned, and was observed in many cases of cerebellar atrophy. It is remarkable, however, that in the uncomplicated cases of cerebellar tumour analysed by Drs. Stewart and Holmes it was not a noteworthy symptom. On the contrary, as described by Babinski, and confirmed by them, there was an almost unnatural steadiness of the homolateral arm when extended horizontally forward. After operation, however, astasia of the head and limbs occurred, but did not as a rule persist long. In one case, however, under the care of Dr. Turner, which I examined, of removal of tumour occupying the left lateral lobe, vermis, and part of the right lateral lobe tremor was very pronounced four years and a half after the operation. When the patient was resting in bed there was no tremor, but when she sat up a fine rotato-lateral tremor was observed in the head. This increased on walking and extended to the whole body. When the hands were stretched out horizontally a fine tremor shewed itself in the right hand. There was also considerable instability in walking.

Other clinical observers have only occasionally described tremors in connexion with cerebellar disease. In 100 cases referred to by Krauss tremors were noted in two or three only. In Wetzel's collection they were observed once in the limbs, and twice in the hands only. Among those analysed by myself they were observed three times. In 10 cases of tumour that have come under my own observation tremors were noted in six, but not always on the side corresponding to the primary seat of the disease. It may be stated as the result of the most recent observations that in man ataxia, atonia, and asthenia are the most essential symptoms of cerebellar tumour.

In their series of cerebellar tumours Drs. Stewart and Holmes seldom observed either the spasm or the rotatory movements which have been described by so many experimenters. That electrical irritation of the cerebellum causes muscular spasms of the same side of the body I have myself shewn. Forced movements of peculiar character which are regarded by Luciani as the result of irritation are seen in animals after removal of one hemisphere, but more especially as Dr. Turner and I have found after division of the peduncles, more particularly the inferior. In the latter case there is in monkeys pleurosthotonos to the side of lesion, with adduction and flexion of the limbs on this side and abduction and extension on the opposite. In dogs, according to Luciani, instead of flexion of the fore limb there is extension. The spasm, however, is not of a rigid character and can be easily overcome. It is probably only a dynamic effect and due to the suddenness of the lesion. Occasionally also there are rotatory movements which tend to carry the animal round on its vertebral axis or cause rolling on the floor. The direction of these movements has been variously described.

In my own and Dr. Turner's experiments, which correspond with those of Magendie and Hitzig, the almost invariable direction while the animal was sprawling on its abdomen was a tendency to roll, or actual and apparently irresistible rolling, towards the side of the lesion. This rolling tendency is really the same as a rotation round the vertebral axis from left to right. Others have described it as in the inverse sense. Probably it may not be altogether uniform, and the difference may depend on the degree in which the peduncle is injured.

The most definite rotation movements observed by Drs. Stewart and Holmes were in a case of cerebellar fits following operation on the left cerebellar lobe causing haemorrhage into the dentate nucleus. Rotation occurred from the side of the lesion towards the sound side. In several cases there was a tendency to lie on or roll to the sound side after operation on one cerebellar hemisphere. These patients would complain of being forced to assume this position apart from vertigo. In states of acute vertigo, however, where extra-cerebellar tumours have pressed on the auditory nerve or middle peduncle, the tendency has been to rotate on the vertical axis, as well as to fall towards the side of disease. The direction of the subjective vertigo in intra-cerebellar tumours is always *from*, whilst in extra-cerebellar tumours it is always *towards* the side of lesion. Stewart and Holmes have never observed in their series of cases fits of the nature described by Hughlings Jackson as occurring with tumours of the middle lobe, viz. rigidity of the muscles of the back of the neck, retraction of the head, opisthotonos, flexion of the elbows, and extension of the legs with pointing of the toes. It is questionable if these phenomena are the direct result of cerebellar irritation proper. Convulsions and retraction of the head with rigidity of the masseters have been observed more particularly in abscess, and have been considered by Friedeberg as pathognomonic. These symptoms, however, occur most commonly when inflammation has been set up about the pons-medulla.

Ocular symptoms occur in the majority of cases both of intra- and extra-cerebellar tumours. Abnormal positions of the eyes have been generally observed after experimental lesion of one-half of the cerebellum. The eyes are deviated to the sound side, and not infrequently there occurs skew deviation, the homolateral eye being directed downwards and inwards, and the contralateral upwards and outwards. This condition may occur in cerebellar tumour without operation, but it is more common after operative interference. It is usually of short duration, and has been regarded as a phenomenon of irritation by Luciani. More probably it belongs to the same category as rotation.

Another symptom almost invariably found both in cerebellar atrophy and cerebellar tumour is *nystagmus*. This is always more marked in lateral than in vertical movements of the eyeballs. The typical nystagmus of cerebellar tumour is a slow, deliberate, jerking movement to the side of lesion and a gradual swing back to the middle line. The nystagmus towards the sound side is generally more rapid and of smaller range. Spontaneous nystagmus occurs only in those cases in which there is marked tendency to conjugate deviation of the eyes, especially for a few days after the operation, and the movements are irregular and jerky towards the side of disease and gradual recession to their natural position. Cerebellar lesions seem to have no direct influence on the pupils. The condition of the pupil depends more on the degree of neuritis or atrophy that may be caused indirectly by tumours.

The majority of cases of intra- and extra-cerebellar tumours shew some degree of paresis or paralysis of the external rectus on the side of the disease more particularly, though both may be affected. This paresis is more marked in extra-cerebellar growths, and most often when the tumour invades the middle lobe and presses on the fourth ventricle. The paralysis is never complete unless the tumour actually invades the pons and involves the sixth nerve. Other cranial nerves may be compressed. The eighth is specially apt to suffer in extra-cerebellar tumour, and there is almost always some degree of deafness on the side of the lesion in such cases. The deafness may be complete both to aerial and bone conduction. Tinnitus is frequent. In intra-cerebellar tumour there is never deafness unless there is obvious compression of the auditory nerve. The seventh cranial nerve is also liable to compression, resulting in facial paresis of the peripheral type. It is specially seen in extra-cerebellar growths. In some cases the affection amounts to complete hemiplegia with the reaction of degeneration. In some cases there may be complete paralysis of the opposite side, and it is then of the usual cerebral type, and of the same character as the hemiplegia that may likewise be induced by pressure on the pyramidal tracts. The *fifth* nerve not infrequently suffers both in intra- and extra-cerebellar tumours, but especially the latter. Both roots may be affected, but as a rule the sensory more than the motor. The result is paraesthesia or anaesthesia of variable degree. In some cases a trigeminal neuralgia may be set up. Occasionally there is anaesthesia of the cornea, accompanied by so-called neuro-paralytic ophthalmia. In such case there is also loss of taste on the anterior two-thirds of the tongue. The ninth, tenth, eleventh, and twelfth cranial nerves may be implicated in extra-cerebellar growths, and as a result there may be impairment or paralysis of articulation, phonation, or deglutition. Affection of *articulation*, however, is not always due to direct pressure on the cranial nerves of the muscles concerned in articulation. The mechanism of articulation is rarely directly affected by extensive unilateral disease of the cerebellum, but it may be implicated when both sides of the cerebellum are involved, and not infrequently occurs when the whole cerebellum has been extensively injured by operative procedures for the removal of tumours. It becomes hesitating, slow, explosive, and scanning like that of disseminated sclerosis. This was observed by Nothnagel in all his cases of cerebellar atrophy, and was a marked feature in the cases of familial degeneration of the cerebellum reported by Dr. Gordon Holmes. There is reason, therefore, for believing that affection of articulation may belong to the same category as the other disturbances due to cerebellar lesion as such.

Affections of general or special sensation never occur as the direct result of cerebellar disease. On this point the evidence of clinical medicine is conclusive. If sensory impairment, general or special, ever does occur, it can always be ascribed to indirect action. Though the cerebellar cortex is the centre of convergence of afferent impressions of various kinds, such as vestibular and muscular, they are not such as rise into consciousness, and though cerebellar ataxia may be ascribed to loss of muscular afferent impressions, it is not correct to say with Lussana that it is due to loss of the muscular sense. For in cerebellar disease as such there is no impairment of the sense of position of the limbs, of appreciation of weight, or of stereognosis.

As the afferent impressions which reach the cerebellar cortex (and thence through the central nuclei act on the motor apparatus concerned in the maintenance of muscular tone) do not rise into consciousness, one cannot, strictly speaking, correlate cerebellar action with the psychical functions. Though in many cases of cerebellar atrophy there has been observed impairment of intelligence varying from mere weakness of intellect up to imbecility or even idiocy, this is more probably the result of some coincident cerebral degeneration. In tumours of the cerebellum mental hebetude or stupor is not uncommon, but this may with good reason be ascribed to increased intracranial pressure or secondary hydrocephalus. This was present in 33 per cent of Bernhardt's cases, 35 per cent of Wetzel's, and in 37 per cent of my own.

Luciani holds that the cerebellum exerts a *trophic* influence on the tissues, an opinion which he founds on the occurrence of certain cutaneous and other dystrophies after extirpation of the cerebellum in animals. Among these phenomena he describes polyuria, glycosuria, acetonuria, marasmus, and conjunctivitis. Dr. Turner and I were not able to confirm Luciani's observations, nor do they receive much support from clinical observation. It is worth noting, however, that in the cases of the three brothers, described by Dr. Gordon Holmes as examples of familial cerebellar degeneration, the sexual organs were abnormally small and evidently under-developed.

Cerebellar degeneration as such is compatible with comparatively long duration of life. In a case reported by Bond of complete sclerosis of the cerebellum the duration of life was fifty-three years, death occurring not from the cerebellar disease but from pulmonary tuberculosis. Cerebellar tumours often run a rapid course; the average duration is two years (Preston). It is noteworthy, however, that after several months of active disturbance, periods of remission may occur extending over several years, during which the patient may continue fairly well with the exception, perhaps, of impairment or loss of vision from post-neuritic atrophy. Death may occur quite suddenly, apparently from failure of the respiration. This mode of death occurs in from 20 to 25 per cent of cases of tumour.

There are certain *accessory* symptoms of cerebellar tumour which are valuable aids to diagnosis; the more important of these are vertigo, vomiting, and headache. Vertigo is specially frequent and often distressing. According to Drs. Stewart and Holmes, it occurs in two principal forms, either as a definite sense of movement of self or surrounding objects, or as an indefinable giddy feeling. Both in intra- and extra-cerebellar tumours objects seem to move from the side of lesion to the opposite side, whereas the sense of rotation of self is in intra-cerebellar tumours from the side of lesion to the sound side, and in extra-cerebellar tumours from the sound side to the side of lesion. This difference may be dependent on the fact that in extra-cerebellar tumours the auditory nerve and middle peduncle are more apt to be irritated than in tumours of the cerebellar substance proper. The other form of vertigo is a more indefinite, giddy, or swimming sensation in which the patient feels that he must fall unless he can cling to some support.

Vomiting is almost invariable at some stage in the course of cerebellar tumours. It is more frequent and more persistent than in similar disease affecting the other intracranial centres. The special frequency of vomiting under such conditions as encroach on the posterior fossa may perhaps be due to affection of the medulla oblongata and vagal nuclei.

The headache of cerebellar tumour is, like optic neuritis, an early symptom. It is often very intense. The position of the pain complained of by the patient does not, however, necessarily correspond to the seat of the disease. Though most frequently, perhaps, in the occipital region, it may be mainly or wholly frontal or temporal. In one case of long duration under my own care, the pain was frontal for eighteen years, and occipital during the last four years of life. A peculiar case has been reported by Dr. Purves Stewart. This was a cystic tumour of the right cerebellar hemisphere, with persistent boring headache localised in a small area of the left frontal region. He is of opinion that this may be due to radiation along the fronto-pontine tract, but Drs. Stewart and Holmes have found no corroboration of this hypothesis in their collection of cases.

Knapp has recorded a case in which there was such marked tenderness over the opposite temple that the patient was trephined over this region.

My own experience leads me to attach comparatively little importance to the situation of the pain spontaneously complained of in intracranial disease. It is different, however, with the pain elicited by deep pressure or percussion. In a large proportion of the cases of cerebellar tumour tenderness or decided pain may be elicited in the occiput, even though pain is not spontaneously complained of in this region, or even referred elsewhere. It is generally most marked on the side of the disease, though in some cases the whole occiput is tender, and one cannot say that one side is more so than the other. In the absence of other localising symptoms I would attach considerable importance to the seat of greatest intensity of the pain on pressure as an index of the probable position of the tumour.

Summary.—Lesions of the cerebellum may be entirely latent. This is the case most frequently when only one cerebellar hemisphere is affected.

The pathognomonic indication of cerebellar disease is an uncertain, reeling gait like that of alcoholic intoxication. With this there may coexist an unsteady, weak, and atonic condition of the limbs, especially on the side of lesion, tremor of the head and trunk, scanning speech, and nystagmus.

REFERENCES

1. Ackermann. *Deutsche med. Wchnschr.*, 1893, xix. 518.—2. Allocco. Ref. *Neurol. Centralbl.*, 1894, xiii. 223.—3. Ashby. Ref. *Lancet*, 1893, ii. 1585.—4. Ashworth. *Brit. Med. Journ.*, 1891, i. 67.—5. D'Asteos, Leon. *Rev. des mal. de l'enf.*, May 1894.—6. Atkins. *Brain*, 1878-79, i. 410.—7. Ballance. *Lancet*, 1895, i. 618.—8. Barling. *Birmingham Med. Review*, 1892, xxxii. 223.—9. Bastian. *Brit. Med. Journ.*, 1890, i. 480.—10. Batten, F. E. *Brain*, London, 1905, xxviii. 488.—11. Becker. *Virchows Arch.*, 1888, cxi. 173-186.—12. Bernhardt. *Hirngeschwülste*, 1881.—13. Bohm. Ref. *Neurol. Centralbl.*, 1892, xi. 380.—14. Bond. *Journ. Ment. Sc.*, London, 1895, xli. 409.—15. Booth. *Journ. Nerv. and Ment. Dis.*, 1890, xvii. 684.—16. Borell. *Arch. f. Pyschiat.*, Berl., 1884, xv. 268.—17. Bowlby. *Brit. Med. Journ.*, 1890, i. 1432.—18. Bradshaw. *Brit. Med. Journ.*, 1893, i. 1196.—19. Bristowe. *Brain*, 1884, vi. 185, Case 4.—20. Brown, Sanger. *Ibid.*, 1892, xv. 250.—21. Bruns. *Neurol. Centralbl.*, 1894, xiii. 47 ; *Arch. f. Psychiat.*, 1894, xxvi. 311.—22. Brydon. *Brit. Med. Journ.*, 1890, i. 709.—23. Buzzard, T. *Brain*, 1888-89, xi. 84.—24. Campbell. Ref. *Lancet*, 1894.—25. *Idem. Brit. Med. Journ.*, 1894, ii. 641.—26. Cramer. *Beitr. z. path. Anat. u. z. allg. Path.*, 1892, xi. 39.—27. Dana. *Med. Rec.*, N.Y. 1890, xxxvii. 53.—28. Dean. *Lancet*, 1892, ii. 250.—29. Dercum. *Journ. Nerv. and Ment. Dis.*, 1893, xx. 675.—30. Doursout. *Ann. méd.-psychol.*, 1891, 7. s., xiii. 345.—31. Dreschfeld. *Med. Times and Gaz.*, 1881, ii. 734.—32. Drummond. *Lancet*, 1894, ii. 190.—33. Ebstein. *Virchows Arch.*, 1870, xlix. 145-164.—34. Eskridge. *Boston Med. and Surg. Journ.*, 1895, cxxxii. 25.—35. Farquharson, *Lancet*, 1893, i. 1501.—36. Ferrier. Quain's *Dictionary of Medicine*, 1882, 226.—37. *Idem. Functions of the Brain*, 2nd ed. 1886, 174-219.—38. *Idem. Brain*, 1881, iii. 85.—39. *Idem. Ibid.*, 1894, xvii. 1.—40. Ferrier and Turner. *Phil. Trans.*, 1894, B. clxxxv. 719.—41. Fraser. *Glasgow Med. Journ.*, 1880, xiii. 199.—42. Friedeberg. *Berl. klin. Wchnschr.*, 1895, xxxii. 719.—43. Gowers. *Diseases of the Nervous System*, 2nd ed., 1893, ii.—44. Grainger Stewart and Gordon Holmes. *Brain*, 1904, xxvii. 522.—45. Grasset et Rauzier. *Traité pratique des maladies du système nerveux*, 1894, 4me éd., tome i. p. 361.—46. Gwynne. *Brit. Med. Journ.*, 1891, i. 290.—47. Hammarberg. *Nord. med. Ark.*, 1890, xxii. Nr. 23.—48. Handford. *Brain*, 1892, xv. 458.—49. Harrison. *Lancet*, 1892, ii. 763.—50. *Idem. Lancet*, 1894, ii. 795.—51. Hell, van. Ref. *Neurol. Centralbl.*, 1892, xi. 381.—52. Herringham and Andrewes. *St. Barth. Hosp. Rep.*, 1888, xxiv. 241.—53. Hitzig. *Arch. f. Psychiat.*, 1884, xv. 267.—54. Ingels. *Bull. Soc. de méd. ment. de Belgique*, 1884, Nr. 35.—55. *Idem.* Ref. *Neurol. Centralbl.*, 1885, iv. 272.—56. Holmes, Gordon. *Brain*, 1907, xxix. 545.—57. Horsley and Clarke. *Ibid.*, 1905, xxviii. 13.—58. Jackson and Russell. *Brit. Med. Journ.*, 1894, i. 393.—59. Jalland. *Lancet*, 1894, ii. 671.—60. Jones, Handfield. *Brain*, 1884-85, vii. 398.—61. Kirchhoff. *Arch. f. Psychiat.*, 1882, xii. 647.—62. Knapp. *Intracranial Growths*, 1891.—63. Krauss. *New York Med. Journ.*, 1895, lxi. 673.—64. Krohn. *Journ. Nerv. and Ment. Dis.*, 1892, xvii. 731.—65. Ladame. *Hirngeschwülste*, 1865.—66. Leimbach. *Deutsche Ztschr. f. Nervenh.*, 1891, i. 319.—67. Londe, Paul.

Hérédo-ataxie cérébelleuse, 1895.—68. Luciani. *Il Cervelletto*, 1891.—69. *Idem.* *Arch. ital. biol.*, 1894, xxi. 190.—70. Luderitz. Inaug. Diss., Strassburg, 1893.—71. M'Burney and Starr. *Trans. N. Y. Acad. of Med.*, 1893, x. 1.—72. Macdonald. *Brain*, 1890, xiii. 83.—73. Macewen. *Pyogenic Diseases*, 1893, 197, Case 1.—74. McWeeney. *Brit. Med. Journ.*, 1892, i. 1255.—75. Major. *Journ. Ment. Sc.*, 1882-83, xxviii. 532.—76. *Idem.* Ref. *Lancet*, 1895, i. 720.—77. Marie, P. *Semaine méd.*, 1893, xiii. 444.—78. Menzel. *Arch. f. Psychiat.*, Berl., 1891, xxii. 160.—79. Menzies. *Brain*, 1893, xvi. 436.—80. Milligan and Hare. *Brit. Med. Journ.*, 1890, i. 231.—81. Mills. *Journ. Nerv. and Ment. Dis.*, 1890, xvii. 43.—82. Mingazzini. *Monatssch. f. Psychiat. u. Neurol.*, 1906, xviii. 442—83. Morrow. *Journ. Am. Med. Assoc.*, 1893, xxi. 607.—84. Noel. *Bull. Soc. anat.*, 1892, 6. s., 638, 63.—85. Nonne. *Arch. f. Psychiat.*, 1891, xxii. 283.—86. Nothnagel. *Topische Diagnostik, etc.*, 1879.—87. Ogilvie. *Brain*, 1885-86, viii. 405.—88. Oliver. *Lancet*, 1885, i. 1237.—89. Oppenheim. *Neurol. Centralbl.*, 1894, xiii. 48.—90. Ormerod. *Brain*, 1895, xviii. 421.—91. Otto. *Arch. f. Psychiat.*, Berl., 1874, iv. 730, 1876, vi. 859.—92. Popoff. *Rev. neurol.*, 1895, lii. 207.—93. Prance. *Brit. Med. Journ.*, 1892, ii. 1108.—94. Preston. *Journ. Nerv. and Ment. Dis.*, 1892, xix. 263.—95. Ratcliffe. *Birmingham Med. Rev.*, 1894, xxxv. 157.—96. Ross. *Diseases of the Nervous System*, i. 57.—97. Rumpf. *Arch. f. Psychiat.*, Berl., 1897, xvi. 435.—98. Russell, J. S. R. *Brit. Med. Journ.*, 1893, ii. 680.—99. *Idem.* *Phil. Trans.*, London, 1894, B. clxxxv. 859.—100. *Idem.* *Brit. Med. Journ.*, 1895, i. 1079.—101. *Idem.* *Brain*, London, 1895, xviii. 37.—102. Schultze. *Virchows Arch.*, 1887, cviii. 331.—103. Sherrington. *Integrative Action of the Nervous System*, 1906.—104. Sommer. *Arch. f. Psychiat.*, Berl., 1884, xv. 252.—105. Stanley. *Brit. Med. Journ.*, 1895, i. 370.—106. Stewart, Purves. *Edin. Hosp. Rep.*, 1895, iii. 447.—107. Suckling. *Lancet*, 1887, ii. 656.—108. Thacker. *Med. Rec.*, New York, 1891, i. 118.—109. Thomas. *Lancet*, 1895, i. 1249.—110. *Idem.* *Rev. neurol.*, Paris, 1905, xiii. 16.—111. Wetzel. *Zur Diagnostik der Kleinhirntumoren*, Inaug. Diss., Halle, 1890.—112. Williamson. *Intern. Journ. Med. Sc.*, Edin., 1892, civ. 151.—113. Winter and Deanesly. *Lancet*, 1894, ii. 1338.—114. Wollenberg. *Arch. f. Psychiat.*, Berl., 1890, xxi. 778.—115. Wulff. *Deutsche med. Ztg.*, 1885, No. 75.—116. *Idem.* *Hérédo-ataxie cérébelleuse.*

Lesions of the Cerebellar Peduncles.—Lesions limited to the cerebellar peduncles are exceedingly rare, and in the case of the inferior peduncles practically unknown. The most exact information which we possess respecting the effects of lesions of these structures is derived from experimental physiology. The best-known experiments are those of Magendie on the middle peduncles. Magendie describes, as the chief effect of the section of the middle peduncle in the lower animals, an irresistible tendency to roll towards the side of the lesion, and a peculiar skew deviation of the optic axes, so that the eye on the side of lesion looks downwards and inwards, and the other upwards and outwards.

Curschmann has described as the effect of section of the united anterior and posterior cerebellar peduncles in rabbits, first, irregular muscular contractions, without rotation or change of position; and, secondly, a forced position to the side of lesion (Seitenzwangslage), to which the animal always returned if displaced, and which it retained till death. He obtained the same results on cutting through the middle peduncle separately, and attributes the rotatory movements and squint, described by Magendie, to lesion of the pons and tuberculum acusticum, rather than to lesion of the peduncle itself. Dr. Turner and I have found that division of any one of the cerebellar peduncles in the monkey produces symptoms similar to those of destruction of the lateral lobe (see page 137), the chief difference being the greater tendency to roll towards the side of the lesion. The most characteristic and frequent forced position was observed after section of the inferior peduncle, namely, curvation of the vertebral axis, with the concavity towards the side of the lesion; adduction and flexion of the limbs on the side of the lesion, and abduction and extension of the limbs of the opposite side. In the majority of cases also the chin was deflected towards the side opposite the lesion, and in the direction of the rotation round the vertebral axis, which caused the animal to roll to the side of the lesion. The tendency to rotation, however, is only temporary, and ultimately entirely subsides.

Superior Peduncle.—Curschmann has recorded a case of softening, from capillary haemorrhage, in the right superior peduncle. There were, however, indications also of basilar tuberculous meningitis, which somewhat complicated the case. The symptoms were a forced position of the head and body towards the right side, to which position the patient invariably returned when resistance to the movement was withdrawn. There was no motor paralysis, and no deviation of the optic axes. Bannister has recorded a case of minute haemorrhagic extravasation in the ventral aspect of the right superior cerebellar peduncle. The symptoms were sudden onset of vomiting, which was very persistent, and a reeling gait. The patient lay on the right side with her head turned strongly to this side. The left arm was rigidly flexed, the left leg extended and lax. The eyeballs were in rapid horizontal movement from side to side. The whole of the left side became ultimately paralysed, and the right side also paretic. The patient in the later stages lay on her back, but the head was still turned to the right. Death occurred twenty-two hours after the onset.

Middle Peduncle.—Lesions implicating the middle peduncle are not unknown, but they are rarely limited to the peduncle itself. Occasionally haemorrhagic extravasation occurs, more or less limited to the peduncle, or extending also into the side of the pons.

The symptoms are somewhat variable, and it is not easy to eliminate the facts due to lesion of the peduncle from those due to lesion of the pons or adjacent structures. Not infrequently paralytic symptoms of the limbs and cranial nerves complicate those strictly dependent on the peduncular lesion. The most characteristic symptoms that have been observed are forced movements, or actual rotation, associated occasionally with deviation of the optic axes. A case, observed by Serres, has been reported by Longet, in which there was a focus of softening at the point where the right cerebellar peduncle radiates into the cerebellar hemisphere. The symptoms began with rotation from right to left, followed by left hemiplegia, which ultimately disappeared, with the exception of the paralysis of the left leg. Death occurred five months after the first onset. In another case, reported by Nonat, the patient, a woman aged 60, had an apoplectic attack due to haemorrhagic effusion in the right middle peduncle. The patient assumed a forced position on the right side, with the head strongly turned to this side. The eyes were immovable, and the optic axes deviated so that the right eye looked downwards and outwards, and the left upwards and inwards. Death occurred on the following day. Another interesting case has been reported by Friedeberg of haemorrhagic effusion into the left cerebellar hemisphere followed by meningitis due to traumatic lesion. In this case the patient suffered from vertigo, with a tendency in walking to go to the left, followed by repeated attacks of rotation round the vertical axis from left to right. The eyes were in a constant state of oscillation. On cessation of the rotatory attacks vertigo continued, and a sensation as if he were falling to the right. The whole duration of the symptoms was about five weeks. A case of tumour implicating the right middle peduncle has been recorded by Bernhardt. In this case the patient when lying in bed had a tendency to turn from right to left. The eyes looked upwards and to the left, and there was some degree of nystagmus. In standing and walking there was a tendency to fall to the left.

Inferior Peduncle.—There are no clinical cases on record of lesions strictly limited to the inferior peduncle. I have already alluded to the characteristic forced movements and tendency to rotation which invariably occurred after section of the inferior peduncle in monkeys. Very commonly also anaesthesia of the cornea and face occurred on the side of lesion, owing to almost unavoidable division of the subjacent ascending root of the trigeminus. On experimental grounds I should be led to infer that forced movements or a tendency to rotation associated with anaesthesia of the cornea would be pathognomonic of lesion of the corresponding inferior peduncle of the cerebellum. I have not, however, been able to find any record of such a combination of symptoms in man. A case has been recorded by Couty of a tumour, the size of a nut, affecting the left inferior peduncle; this, however, was complicated by tuberculous meningitis of the convexity and base. During life there were motor disorders on the side of the lesion—at first pseud-ataxic, and later hemiplegic, the face being unaffected. There was no loss of sensation. Brissaud recorded a case of tumour of the left restiform body which affected also the anterior segment of the left cerebellar hemisphere. The situation of the tumour was diagnosed during life. The symptoms were slowly increasing deafness in the left ear, convulsive tic on the left side of the face, stiffness and pain in the neck, retraction of the head, severe occipital headache, and cerebellar gait. No forced movements or rotatory disturbances were observed in these two cases, nor does there appear to have been anaesthesia of the cornea or face on the side of lesion. Mendel, in reporting a case of Graves' disease in which there was atrophy of the left restiform body, attempts to establish a relation between this disease and lesion of the restiform body, and quotes the experiments of Filehne and others, from which it appears that symptoms resembling Graves' disease were induced in dogs and rabbits by section of the corpus restiforme. Dr. Turner and I, however, observed no such phenomena in monkeys; and in man integrity of the restiform body has often been established in cases of Graves' disease (*vide* Joffroy and Achard, Hezel, and Maude).

It thus appears that the most characteristic symptoms of lesion of any of the cerebellar peduncles are—rotation round the vertebral axis, or forcible impulse causing the patient to assume some fixed position. In lesions of the superior peduncle the cases of Curschmann and Bannister shew a forced position on the side of lesion; in those of the middle peduncle, the cases of Serres, Friedeberg, and Bernhardt shew rotation from the side of lesion; and that of Nonat forced position on the side of lesion—facts which are all in harmony with the experiments of Magendie, as well as with those of Dr. Turner and myself. For the rotation from the side of lesion is essentially the same phenomenon as forced position, or rolling towards the side of lesion.

REFERENCES

1. BANNISTER. *Journ. Nerv. and Ment. Dis.*, N.Y., 1890, xvii. 597.—2. BERNHARDT. *Berl. klin. Wchnschr.*, 1872, ix. 562.—3. BRISSAUD. *Progrès méd.*, Paris, 1894, xix. 41.—4. COUTY. *Gaz. méd. de Paris*, 1877, 4. s., vi. 250.—5. CURSCHMANN. *Deutsch. Arch. f. klin. Med.*, 1874, xii. 356.—6. FERRIER. Quain's *Dictionary of Medicine*, 226.—7. FERRIER and TURNER. *Phil. Trans.*, 1894, B. clxxxv. 719.—8. FRIEDEBERG. *Arch. d. Heilk.*, 1861, ii. 385.—9. GOWERS. *Diseases of the Nervous System*, London, 1893, 2nd edit. vol. ii.—10. HEZEL. *Deutsche Ztschr. f. Nervenh.*, 1893, iv. 353.—11. JOFFROY et ACHARD. *Arch. de méd. expér. et d'anat. path.*, 1893, iv. 404.—12. MAUDE. *Brain*, 1894, xvii. 246.—13. MENDEL. *Arch. f. Psychiat.*, Berl., 1892, xxiv. 282.—14. NONAT. *Gaz. des hôp.*, Paris, 1861, 3. s., xiii. 24.—15. NOTHNAGEL. *Top. Diagnose der Gehirnkrankheiten*, 1879, S. 81-98.

LESIONS OF THE MEDULLA OBLONGATA.—The pathology of the medulla oblongata is unusually complex. Not merely is it primarily liable to the same lesions as affect other nerve-centres, but, more frequently, it is implicated in diseases primarily originating in the pons or cerebellum; it is also liable to be affected indirectly by intracranial disease in general, particularly by those forms which cause an increase of the intracranial pressure. Being also a kind of isthmus between the brain and spinal cord, it is subject to ascending or descending degenerations in consequence of cerebral or spinal lesions; and, being the centre of many complicated synergic muscular acts, its nuclei are subject to special forms of degeneration similar to those which affect the oculomotor nuclei and anterior cornua of the spinal cord. With the indirect affections of the medulla oblongata in connexion with various forms of intracranial disease, ascending and descending degenerations, and the clinical history of bulbar or labio-glosso-laryngeal paralysis, it is not the purpose of this article to deal, as these subjects are discussed under their respective heads. Attention will be mainly directed to the forms of disease primarily affecting the medulla oblongata, and to the symptoms on which the regional diagnosis can be based.

Haemorrhage.—Haemorrhage limited to the medulla is comparatively rare; more often the pons and medulla are affected together. It may be the result of disease, or due to injury, such as a blow on the head or back of the neck. Duret has shewn that in animals blows on the head, especially on the forehead and vertex, may cause punctiform extravasations in the medulla oblongata and base of the brain. Schulz has recorded a case of bulbar paralysis immediately following a blow on the back of the neck, presumably due to traumatic haemorrhage. Not infrequently effusions of blood take place in the fourth ventricle, either arising from the pons, medulla, or cerebellum, or from inundations of the lateral ventricles gaining access by the aqueduct of Sylvius.

Haemorrhage into the medulla, or into the fourth ventricle, is usually fatal. Death may be instantaneous, or within a few hours, in profound coma with stertorous respiration and occasional convulsions. Very rarely bulbar haemorrhage gives rise to chronic stationary lesions with bulbar symptoms, but these are mostly due to thrombosis or embolism, or acute myelitis.

Softening.—Softening of the medulla oblongata, due to thrombosis or embolism of the vertebral arteries and their branches, is of common occurrence, and is the cause of what is termed apoplectiform bulbar paralysis. The arteries of the medulla oblongata, according to the researches of Duret, arise from the vertebrals, or from the branches of the anterior spinal and posterior inferior cerebellar. One set of these vessels, the medial, enter the medulla in the middle line, and supply the nerve nuclei. The medial branches to the eleventh and twelfth nuclei arise from the anterior spinal. Those of the tenth, ninth, and eighth come from the basilar or upper end of the vertebral artery. The other set—namely, the lateral or radicular branches—enter the medulla with the nerve-roots, and supply also the nerve nuclei. The radicular branches to the bulbar nerves arise from the vertebral, except that to the spinal accessory, which comes from the inferior cerebellar; and that to the hypoglossal, which arises from the anterior spinal artery. Those to the facial or auditory nerves may come from the vertebral, or basilar, or both.

Embolic or thrombotic occlusion of these arterioles gives rise to softening of the centres to which they are distributed, and therefore causes acute bulbar symptoms. The extent and severity of the symptoms vary as the position and extent of the softening. Generally the onset is sudden as in haemorrhage, but the course is more slow and the duration more chronic. As a rule there is no loss of consciousness, and this is sometimes considered as a point of distinction between haemorrhage and softening, but it is doubtful if it is trustworthy.

The symptoms caused by softening so induced will be considered presently.

Tumours.—Tumours may originate primarily in the medulla oblongata; but more commonly they invade the medulla from the cerebellum, pons, or base of the skull. Cases have been recorded of tumour actually in the substance of the medulla without any characteristic symptoms in life (Wilks). Sometimes tumours specially grow in the fourth ventricle; and cysticercus attached to the ependyma or free in the ventricle is not uncommon and is associated with a peculiar symptomatology (see p. 159).

Abscess. — Abscess of the medulla is very rare. Eisenlohr has recorded two cases of abscess of the medulla — the one secondary to empyema, the other to cerebrospinal meningitis. Chassel has recorded one secondary to acute infective osteomyelitis.

Acute Bulbar Myelitis.—Under this head an affection is described of the nature of bulbar or labio-glosso-laryngeal paralysis, but of rapid onset, and probably due to acute inflammation of the bulbar nuclei. The symptoms set in generally with dysphagia, followed by paresis or paralysis of the soft palate, larynx, and tongue. The extremities are occasionally paretic, and paralysis of the face and ocular muscles is not uncommon. The temperature is occasionally elevated, the pulse always rapid, and death occurs in a few days or weeks, invariably preceded by paralysis of respiration.

Microscopical examination, in the few cases so examined, has revealed signs of inflammatory change in the bulbar nuclei,—petechial haemorrhages, thrombosis, and small-celled infiltration.

Compression of the Medulla.—Sudden compression of the medulla is not uncommon from fracture or dislocation of the atlas and axis, as in falls, hanging, twisting the neck, or diseased vertebrae. Usually death is instantaneous from respiratory paralysis—the heart often continuing to beat for an appreciable interval after cessation of all respiratory movements.

Gradual compression of the medulla oblongata may be caused by chronic disease and tumours at the base of the skull, aneurysm of the basilar or vertebral arteries, and so forth.

Oppenheim and Siemerling attribute compression symptoms in some cases to dilated atheromatous vessels. They are of opinion that arteriosclerosis often leads to dilatation of the vessels without any apparent anatomical lesion, and yet the pons, medulla, and the issuing nerves may suffer in function from the pressure so exerted.

Symptomatology.—The pathognomonic symptoms of bulbar lesion are a conjoint affection of the extremities, and of one or more of the bulbar cranial nerves, with impairment of speech and deglutition, together with cardio-respiratory disturbances. In some cases of bulbar lesion the limbs only are affected, in which case no certain diagnosis can be made of the seat of the lesion. All four limbs may be paralysed ; or, in other cases, the affection of the limbs may be of the hemiplegic order. With the limbs the cranial nerve most frequently implicated is the hypoglossal, more rarely the seventh or sixth. When the hypoglossal is affected the symptoms are impaired mobility of the tongue, with a greater or less degree of dysarthria. This combination of symptoms, however, may also arise from disease of the pons, and there are no features which clearly distinguish between the one and the other. The tongue and the speech are rarely, if ever, so affected as in progressive bulbar paralysis, nor has atrophy of the tongue with diminished electrical contractility been observed. An exemplary case of bulbar lesion with paralysis of the limbs on the one side, and of the tongue on the other, has been recorded by Madame Goukovski. The necropsy revealed a focus of softening limited to the left olive and left pyramid, and destroying the greater part of the roots of the left hypoglossal. She refers to a similar case reported by Souques, which was complicated, however, with paralysis of the sixth and seventh nerves.

In some cases the affection of the limbs is of a diagonal character, that is, the arm is affected on the one side and the leg on the other. This can only occur when the motor fibres for one limb are cut before decussation, and those for the other after they have crossed; in such a case the lesion must be situated at the decussation of the pyramids. An instance of this kind, due to a minute haemorrhage in the lower part of the medulla oblongata on one side, has been recorded by Tildesley.

Dr. Turner and I have shewn that destruction of the tubercle of Rolando causes anaesthesia in the region of distribution of the fifth nerve on the side of lesion, and analgesia of the trunk and limbs of the opposite side. A case illustrating this combination of symptoms has been recorded by Senator; it was due to thrombotic softening in the outer postero-inferior aspect of the left side of the medulla oblongata. The symptoms were anaesthesia of the left fifth, and almost complete analgesia of the right side of the body.

Some cases of bulbar lesion have shewn an ataxic condition of the limbs: Goldscheider has recorded a case of lesion affecting the pyramids, upper part of the olives, inter-olivary layer, and nuclear region on both sides, but specially the left, in which the left arm was choreic but apparently normal as regards sensation; while there was loss of the sense of position in the right arm. He is of opinion that this case shews that the tracts of the muscle-sense occupy a different region from those of other forms of sensibility—the former lying mesial to the hypoglossal nerves, the latter occupying the outer portion of the formatio reticularis; and Starr considers that the absence of ataxy in anaesthetic limbs is due to the fillet not being affected.

A symptom of great importance in the regional diagnosis of bulbar lesions is the occurrence of dysphagia or paralysis of deglutition. Dysphagia may also occur to some extent from lesion of the pons, especially when the tongue is affected; but complete paralysis of deglutition is a sign of direct or indirect lesion of the medulla oblongata. This may occur when the movements of the tongue are only relatively slightly impaired.

Paralysis of the soft palate on one or both sides is a frequent symptom, and with this there may be entire loss of the palate reflex. Together with the affection of the soft palate there may be hoarseness or aphonia from paralysis of one or both vocal cords.

The association of paralysis of the palate and of the vocal cords, and especially the absence of palate reflex, serve to distinguish true bulbar lesion from the pseudo-bulbar paralysis resulting from bilateral lesion of the articulatory centres of the cortex or lenticular nuclei.

The conjoint paralysis of the palate and vocal cords indicates lesion of the nucleus of the accessorio-vagus, or of its root-fibres. In a patient under my own care there was a combination of paralysis of the right side of the face, right side of the palate, right vocal cord, right sterno-mastoid and trapezius, as well as the right glossopharyngeal. In this case there was probably a lesion of the roots of the respective cranial nerves at their emergence from the medulla oblongata. The muscles of mastication have been affected in some cases. (Dixon Mann, Tildesley.)

Trismus has been mentioned by Joffroy as a characteristic symptom in apoplectiform bulbar paralysis, but, as shewn by Nothnagel, it is not a common symptom.

Cardio-respiratory disturbances, such as irregularity of the heart, acceleration or retardation of the pulse, sighing, irregular or laboured respiration, intense dyspnoea often amounting to orthopnoea, coughing, hiccup, or vomiting have also been observed not infrequently.

In a case reported by Glynn, of a small glioma in the middle line of the medulla at the level of the calamus scriptorius, there were present intense dyspnoea, paralysis of the diaphragm, vomiting, impaired movement of the lips and tongue, dysphagia, abolition of the palate reflex, and weakness in all four extremities, especially the arms. In this case there was also a trace of sugar in the urine.

Albuminuria and glycosuria have often been observed in connexion with bulbar lesions, the latter more particularly with lesions of the floor of the fourth ventricle; but their association with acute bulbar paralysis is by no means constant.

De Jonge records a case of a small tubercle reaching from just below the left olivary body to the first cervical nerve, in which there was glycosuria, which he attributes to the lesion in question. He cites 11 cases of glycosuria with disease of the medulla, in all of which the lesion was considerably higher than in this case; and he concludes, therefore, that the diabetic centre itself was not involved, but only the fibres proceeding from it to the periphery.

Of the various symptoms above mentioned some only may be met with in a given case, and the way in which they are grouped may be of diagnostic significance as to the character of the lesion.

Cysticercus of the fourth ventricle is a not uncommon affection on the Continent, and Stern has collected 72 cases. The vesicles are attached to the ependyma, or float free in the ventricle. Occasionally a correct diagnosis has been made during life, as reported by Bruns and Osterwald. The symptoms relied on by Bruns as most characteristic are: (1) alternations of severe general symptoms, namely, headache, vomiting, and giddiness, with periods of complete intermission; (2) the association of severe attacks of vertigo with sudden movements of the head; (3) the absence or ill-defined character of other objective symptoms, optic neuritis, for instance, only occurring late in the course of the disease; and (4) tendency to sudden death. It has been assumed that the association of vertigo with sudden movements of the head (Bruns' syndrome) indicates a free cysticercus rather than a fixed one; but this has not been as yet certainly established.

Tumours of the fourth ventricle other than cysticerci are apt to be more far-reaching in their effects according to the extent they invade the cerebellum, pons, and medulla. The extraordinary point is the degree to which the pons or medulla may be flattened or displaced without marked local symptoms. The symptoms most commonly met with in tumours of the fourth ventricle are, according to the analysis of Dr. Grainger Stewart: (1) the general symptoms of cerebral tumour:—occipital headache, giddiness and vomiting are early and constant, but optic neuritis may be early or late; (2) nystagmus or lateral movements, and some weakness of the conjugate movements and of the external recti, with slight weakness also of the lower facial muscles; (3) some degree of hypertonicity of the limbs, and some instability of gait. In cysticerci and other tumours of the fourth ventricle, glycosuria has occasionally been found.

Another syndrome, to which special attention has been directed in recent years, is that caused by *thrombosis of the posterior cerebellar artery.* As the artery winds round the medulla towards the vallecula of the cerebellum it gives off small terminal arteries to the ventral and lateral aspect, and this region, therefore, is especially liable to necrosis when the main vessel is blocked. The region affected extends from the point where the inferior cerebellar peduncle begins to pass into the cerebellum to the middle of the hypoglossal nucleus below. The structures most affected are the greater part of the formatio reticularis and its nuclei; the descending root of the fifth and the cells in association; the nucleus ambiguus and glossopharyngeal nucleus and their nerves; the ventral and dorsal spino-cerebellar tracts, and other tracts (olivo-cerebellar) passing into the inferior cerebellar peduncle. The mesial fillet, supplied by twigs from the anterior spinal artery, in a pure case entirely escapes. The symptoms resulting from this lesion embrace unilateral paralysis of the palate and vocal cord, anaesthesia of the pharynx, with perhaps tinnitus, giddiness, and vomiting; central paralysis of the cervical sympathetic, and hemiataxy, with perhaps forced movements on the same side; and an alternate hemianaesthesia, the face being affected on the same side, and the trunk (sometimes also the face) on the opposite side. The sensory affection is of the dissociated type, pain- and temperature-sense being impaired, while tactile sensibility and the muscular sense are intact. Sometimes there is a further dissociation of the temperature-sense—the sense of heat or cold being affected independently of each other. Usually there is nystagmus to the side of the lesion, and frequently vasomotor disturbance of the same distribution as the anaesthesia. The case recorded by Hun is one of the best and earliest examples of this syndrome. Analgesia and thermo-anaesthesia were present on the left side of the body and right side of the face. The left limbs were ataxic, and there was hyperidrosis of the right side of the face and right arm, and other symptoms, as mentioned above. Necropsy revealed occlusion of the left posterior inferior cerebellar artery. In a case recorded by Thomas the symptoms noted were: pain on the right side of the face; tendency to fall to the right; paralysis of the right sympathetic (anidrosis); ataxia of the right upper limb; impairment of pain- and temperature-sense on the right side of the face and left side of the body. The right posterior inferior cerebellar artery, and partly also the right vertebral, were thrombosed. In several other reported cases (Breuer and Marburg, Babinski and Nageotte, Reinhold, Senator) the vertebral was also thrombosed. In two well-observed cases published by Spiller this condition also existed. In one of these there was persistent hiccup, which has also been frequently noted by others.

A valuable clinical study of this syndrome has been published by Dr. S. A. K. Wilson, though there is as yet no verification of the actual seat of the lesion. In a man aged forty-two the onset was sudden, without loss of consciousness, but with a sense of giddiness and faintness, and a feeling of being impelled to the left. The right side of the face was bathed in perspiration, while the left was perfectly dry. There were vomiting, ringing in the left ear, and painful paraesthesia of the left side of the face. There was nystagmus on lateral deviation of the eyes to the left, myosis of the left pupil, and enophthalmos of the left eye and narrowing of the left palpebral fissure; paralysis of the left half of the palate and left vocal cord, inco-ordination of the left limbs, and alternate anaesthesia of the left face and right side of the body. The anaesthesia was dissociated. Tactile sensibility, tactile localisation, and the muscular sense were intact. But on the left face and right trunk and limbs there was complete analgesia, and complete loss of the cold sense, while on the left face there was also complete loss of heat sense, and on the right side of the body all degrees of heat were felt as merely tepid. There was no affection of the pyramidal tracts, and the superficial and deep reflexes were normal. Pricking of the skin on the anaesthetic areas caused no bleeding. In addition there was some irregularity in respiratory rhythm and a degree of tachycardia.

The symptoms in this case, besides their value from a regional diagnostic point of view, have an important bearing on certain physiological problems with regard to the course of the sensory tracts through the medulla, and also on the centres of the cervical sympathetic.

Summary.—Of the various symptoms caused by lesion of the medulla oblongata the most characteristic are impairment or paralysis of articulation, phonation, and deglutition, with disturbances of the heart and respiration.

REFERENCES

1. Bruns. *Neurol. Centralbl.*, 1902, xxi. 565.—2. Chassel. *Wien. med. Wchnschr.*, 1892, xlii. 337, 380.—3. de Jonge. *Arch. f. Psychiat.*, xiii. 658; ref. *Brain*, 1883-4, vi. 418.—4. Duret. *Arch. de physiol.*, Paris, 1873, v. 97, and 1874, vi. 60, 316.—5. *Idem.* *Traumatismes cérébraux*, 1878.—6. Eisenlohr. *Deutsche med. Wchnschr.*, 1892, xviii. 111.—7. Glynn. *Liverpool Med.-Chir. Journ.*, 1887, vii. 428.—8. Goldscheider. *Charité-Ann.*, 1891, xvi. 162; and *Ztschr. f. klin. Med.*, 1890, xvii. 580.—9. Goukovski. *Nouv. icon. de la Salpêt.*, 1895, viii. 178.—10. Grainger Stewart. *Proc. Roy. Soc. Med.*, London, 1908, i. (Neurol. Sect.), 26.—11. Hun. *New York Med. Journ.*, 1897, lxv. 513, 581, 613.—12. Mann, Dixon. *Brain*, 1885, vii. 244.—13. Oppenheim und Siemerling. *Berl. klin. Wchnschr.*, 1886, xxiii. 791.—14. Osterwald. *Neurol. Centralbl.*, 1906, xxv. 265.—15. Schulz. *Neurol. Centralbl.*, 1883, ii. 99.—16. Senator. *Arch. f. Psychiat.*, 1881, xi. 713.—

17. Spiller. *Journ. Nerv. and Ment. Dis.*, 1907, xxxiv. 297.—18. Starr. *Med. Rec.*, N.Y., 1893, xliii. 168.—19. Stern. *Ztschr. f. klin. Med.*, 1907, lxi. 65.—20. Thomas. *Journ. Nerv. and Ment. Dis.*, 1907, xxxiv. 153.—21. Tildesley. *Lancet*, 1895, ii. 1288, case 1.—22. Turner. *Brain*, 1895, xviii. 231.—23. Wilks. *Diseases of the Nervous System*, 1878, 159.—24. Wilson, S. A. K. *Proc. Roy. Soc. Med.*, 1909, ii. (Neurol. Sect.), 52 (references to other reported cases).

Tumours of the Hypophysis Cerebri.—Though tumours of the hypophysis cerebri and infundibulum may not be, strictly speaking, cerebral lesions, yet as a form of intracranial disease with a remarkable general and local symptomatology, they deserve particular mention. The hypophysis consists of two lobes: an anterior—a glandular structure originally an invagination from the primitive pharynx; and a posterior, composed of ependymal and epithelial cells, an outgrowth from the primitive neural tube. On the anterior aspect of the posterior lobe and separated by a cleft from the anterior there is a layer of epithelial cells similar to those of the anterior lobe. This is termed the *pars intermedia.* From the posterior or rather the *pars intermedia*, Prof. Schäfer has separated an extract which, injected into animals, causes vasomotor constriction with rise of blood-pressure and increased excretion of urine. The action of the anterior lobe has not been definitely established.

Enlargement of the hypophysis and deepening of the sella turcica are commonly found in *acromegaly* (*vide* Vol. IV. Part I. p. 389). In a series of 25 cases of tumours of the hypophysis analysed by Dr. Purves Stewart, only 8 exhibited symptoms of acromegaly: 5 of these were cases of adenoma. There is, therefore, considerable evidence in favour of the views of Massalongo and Tamburini, that acromegaly is due to excessive functional activity of the pituitary, and is found more particularly in adenoma, but not in other forms of disease.

As regards the general and local symptoms of pituitary tumour, it is remarkable that these tumours may attain a large size without any very obvious symptoms. Headache is often absent, vomiting is rare, and optic atrophy rather than optic neuritis is the rule. A remarkable symptom is intense and paroxysmal drowsiness, the attacks lasting from hours to days, or even weeks, at a time. Another is the occurrence of amenorrhoea in the female and impotence in the male. Male patients develop a "pituitary eunuchism," and often become excessively fat. The characteristic local sign of pituitary tumour is bitemporal hemianopsy from pressure on the optic chiasma; more commonly perhaps there is complete blindness in one eye and temporal hemianopsy in the other. This sign, with the drowsiness above mentioned, constitutes the characteristic syndrome of pituitary tumour.

David Ferrier.

REFERENCES

1. Schäfer. Croonian Lecture, *Proc. Roy. Soc.*, London, 1909, B. lxxxi. 442. 2.—Stewart, Purves. "Tumours in the Region of Hypophysis Cerebri," *Rev. Neurol. and Psychiat.* Edin 1909, vii. 225. (In this paper a complete bibliography is given.)

D. F.

MENINGITIS

TUBERCULOUS MENINGITIS

MENINGITIS

By Frederick E. Batten, M.D., F.R.C.P.

Definition.—The word meningitis indicates an inflammatory condition of the membranes of the brain and spinal cord. The inflammation may attack the pia mater, the arachnoid, or the dura mater, but most commonly the pia mater and arachnoid membranes are affected. The inflammatory condition may be confined to the brain or to the spinal cord, but in the majority of cases it attacks both.

Classification.—As it is impossible to classify the various forms of meningitis satisfactorily on a purely anatomical basis, a classification based on bacteriological data, although incomplete, will prove to be the most workable. The following scheme, following the lines suggested by Prof. Osler, has advantages both from the clinical and pathological points of view.

The distinction between primary and secondary meningitis is clinical rather than pathological. For under the heading "primary" might also be grouped cases of typhoid meningitis in which meningitis is the only clinical and pathological manifestation of a typhoid infection. As the terms "serous meningitis" and "meningism" have come into common use, a description of these two conditions is given; they are, however, of clinical rather than pathological importance, and the farther pathological examination is carried the smaller will these groups become.

Classification

Primary	Meningococcic	Sporadic type, formerly known as "posterior-basic" meningitis. Epidemic cerebrospinal meningitis (*vide* Vol. I. p. 923).
	Pneumococcic	Pneumococcic infection of the meninges alone.
Secondary	Traumatic	To injuries of the scalp, skull, etc.
	Tuberculous	To tuberculous infection.
	Pneumococcic	To infection of the ear, nose, orbit.
	"	To pneumonia, empyema, endocarditis, peritonitis, etc.

Classification—*continued*

Secondary	Rheumatic . .	To the *Diplococcus rheumaticus* (?).
	Pyogenetic . .	To infection by various forms of staphylococci and streptococci, derived from infection of skin or internal organs.
	Other forms of infection	By *Bacillus typhosus*, *Bacillus enteritidis* (Gaertner), *B. influenzae*, gonococcus, actinomycosis, streptothrix, *B. dysenteriae*, leptothrix, and anthrax.
	Syphilitic.	
	Serous meningitis due to general poisons such as	Alcohol, diabetes, nephritis, lead, acute specific fevers, mumps, and certain otitic infections.
	Meningism . .	Symptoms of meningitis, without demonstrable meningitis.

All these forms of meningitis will not be dealt with here, for some of them are described in separate articles (the epidemic form in Vol. I. p. 923, the tuberculous form on p. 201), and other forms, such as the traumatic, do not enter into the scope of this article, which will be devoted principally to the consideration of the following forms. (1) Meningococcic meningitis, sporadic type formerly known as "posterior-basic" meningitis; (2) Pneumococcic meningitis; (3) Pyogenetic and other forms of infective meningitis; (4) Syphilitic meningitis; (5) Serous meningitis; (6) Meningism.

It is not easy to state with precision the proportion in which these various forms occur; but an analysis of 100 necropsies on cases of meningitis, other than tuberculous, at the Children's Hospital, shewed that 41 were meningococcic; in the majority of these the meningitis had the typical "posterior-basic" distribution, but in a few it was confined to the vertex. In 30 cases the meningitis was due to the pneumococcus. In 23 cases "suppurative meningitis" was present; in 15 of these the characters of the organism were not noted; in the remainder it was a streptococcic, a staphylococcic, or a mixed infection. In 6 cases syphilitic meningitis was present.

1. Meningococcic Meningitis (sporadic type). (Synonym: *Simple meningitis*, "*posterior-basic*" *meningitis*.)

History.—The separation of this disease from tuberculous meningitis dates from Dr. Gee and Sir Thomas Barlow's paper on "Cervical Opisthotonos in Infants" in 1878. Although during subsequent years the disease was commonly recognised clinically by these teachers and their pupils, it was not till 1897 that Dr. Walter Carr summed up, in a paper on "Non-tuberculous Posterior Basic Meningitis in Infants," the leading features of the disease, and shewed that it should be regarded as a distinct form of meningitis and separate from other forms of meningitis secondary to ear disease and other local infection. The next important step was the isolation of the organism of "posterior-basic" meningitis in 1898 by Prof. G. F. Still, who proved that it was practically the same as that isolated by Weichselbaum in the epidemic form of the disease. Then, in 1899, appeared the article of Dr. Lees and Sir T. Barlow in the first edition of this work, based on clinical and pathological observation of 110 cases of the disease. The present article is largely based on their monograph, and their observations, statistics, and illustrations have been freely utilised. I have not, however, adopted the distinction made by these authors between the "posterior-basic" and vertical types of meningococcic infection. That there is some difference in the symptoms produced is obvious, but there is no more reason for separate descriptions than there is for two forms of tuberculous meningitis, one vertical, the other basic. Although at the present time the close relation of "posterior-basic" meningitis and epidemic cerebrospinal meningitis is recognised, there are both clinical and pathological differences which render it advisable to describe these two conditions separately.

Etiology.—*Age of Onset.*—A large proportion of the cases of the sporadic form of meningitis occur in children under one year of age. The following table is given by Dr. Lees and Sir T. Barlow :—

Under three months of age	11 cases.
Over three and under six months	39 ,,
Over six and under nine months	19 ,,
Over nine and under twelve months	15 ,,
During the first year	84 cases.
,, second year	13 ,,
,, third year	6 ,,
,, fourth year	2 ,,
,, fifth year	1 ,,
,, sixth year	4 ,,
	110 ,,

Average age at onset of the 110 cases = 11 months.
,, ,, ,, 84 cases in first year = 5 months.
,, ,, ,, 26 cases older than one year = 2 years 6 months.

76 per cent of the cases occurred during the first year of life, and 88 per cent of the cases were under two years of age.

Month of Onset.—A large proportion of the cases occur during the first six months of the year. The following table is given by Dr. Lees and Sir T. Barlow :—

January	14	July	3
February	11	August	2
March	9	September	5
April	10	October	3
May	10	November	6
June	8	December	4
			85

The same seasonal variation was shewn by the cases at the Children's Hospital between 1901 and 1908, and also by Looft as regards the incidence of cerebrospinal meningitis over a long series of years in Norway, by Davis in the Massachusetts epidemic, and by Prof. Stuart M'Donald in the recent Edinburgh epidemic.

Method of Infection.—Practically nothing is known as to the method of infection. In London the cases come from widely separated districts and only rarely from the same street. Two members of the same family are seldom, if ever, affected. The disease never spreads when nursed in a general ward. Possibly the recent notification of cases in London may furnish some indication of the method of distribution.

Little is known as to the path by which infection reaches the meninges. The ears, nose, and mouth have all been regarded as possible channels of infection, but without convincing proof. The Gram-negative *Micrococcus catarrhalis* is found in the nose, but Dr. Arkwright has shewn that it differs from the meningococcus in many respects. From the faeces Wilson isolated a Gram-negative diplococcus somewhat resembling the meningococcus, but differing in its reaction to some of the sugars. Both Prof. Stuart M'Donald and Dr. Fowler argue that the infection is abdominal, and that the cord lesion is primary, on the ground that in most cases it is older than that in the brain, that the abdominal reflex is abolished early, and that the cerebral disturbance is often slight, whereas the spinal affection is well-marked. This, however, has not been established, and for the present it may be said that nothing is known as to the method of infection.

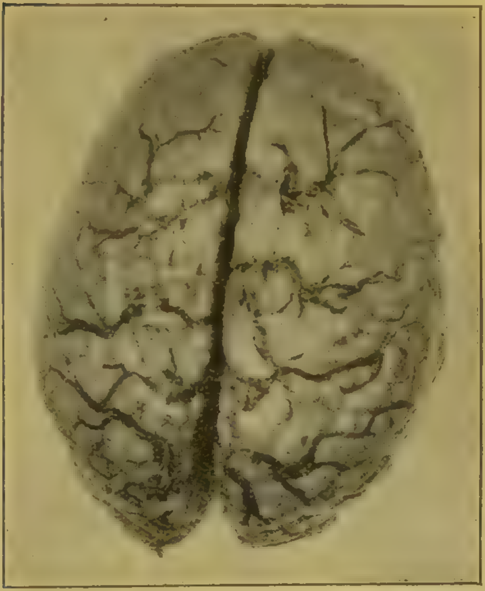

Fig. 11.—Brain from a case of meningococcic meningitis shewing exudation over the vertex with marked engorgement of the veins.

Morbid Anatomy.—Cases dying during the acute early stage of the disease shew an intense inflammatory meningitis, with exudation of lymph on the surface of the brain, sometimes at the vertex, sometimes at the base, and sometimes in both situations. On removing the skull and dura mater, the surface of the brain is seen to be covered with thick greenish lymph, on which the dark and engorged veins stand out in striking contrast. The lymph is held in the meshes of the pia-arachnoid, so that it is not readily washed away by a stream of water. In some cases the exudation is almost entirely confined to the base of the brain, and especially to the under surface of the cerebellum and pons, extending forward

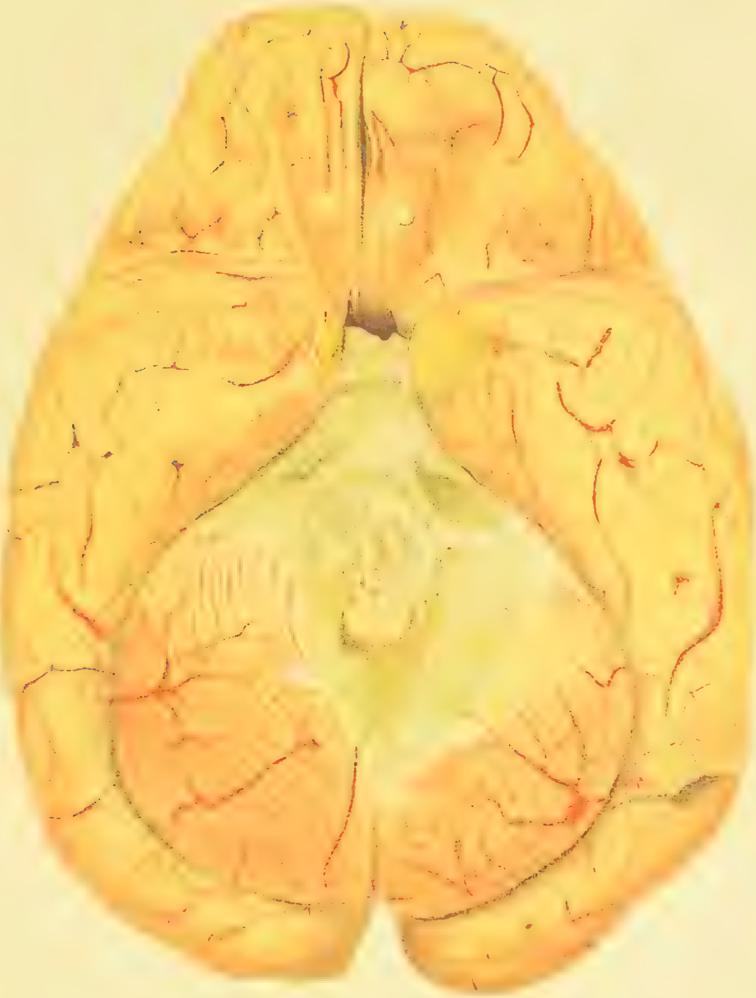

PLATE I.

Base of the brain from a case of the sporadic form of meningococcic ("posterior-basic") meningitis. The exudation extends as far forwards as the optic commissure, and involves the tips of the temporo-sphenoidal lobes. (Lees and Barlow.)

Face page 168.

as far as the optic chiasma and often affecting the tip of the temporo-sphenoidal lobes, as is well shewn in Plate I. It is this latter distribution which has given its name to the disease "posterior-basic" meningitis, for it was originally thought that this distribution was characteristic of the disease, and there is no doubt that in those cases in which head-retraction is most marked, the distribution of the meningitis is in this region. It rarely has the distribution commonly seen in tuberculous meningitis, and the Sylvian fissures are seldom matted by the exudation. When the exudation is at the base, the vertex of the brain is often pale, and the convolutions are flattened from the increase of intracranial pressure. On section of the brain it is often astonishing how little evidence there is of actual congestion; sometimes there are small areas of vascular engorgement, but usually the cerebral substance appears about normal. The condition of the lateral ventricles shews great variation; in rare instances the fluid is purulent, and sometimes the fluid in the descending horns of the lateral ventricles is turbid and contains flakes of lymph, whilst the remainder of the fluid in the ventricles is clear. Dilatation of the ventricles is seldom present in the acutest cases, but is almost invariable in the prolonged cases. The brain substance is often firm, and is not softened as it so commonly is in tuberculous meningitis; on the other hand, the brain substance may be oedematous, as shewn by the ease with which it takes the impress of the hand and by the eversion of the edges of the cut surface.

The spinal cord almost invariably shares in the meningitis of the brain, and the lymph is most thickly distributed on the dorsal surface of the cord, the ventral surface of the cord often presenting but little change. The lymph is sometimes found in the lumbar region only; whilst in other cases the cervical and lumbar regions are affected, the thoracic region being free. It is probable that the position of the patient has some influence on the distribution of the meningitis, but this can hardly be the only factor.

When the meningitis has been of long duration, the surface of the brain is pale, the convolutions flattened, and the pia-arachnoid has an opaque appearance, as if flour had been dusted on its surface. According to Dr. Lees and Sir T. Barlow these opacities in the arachnoid of young children, except when in the neighbourhood of the longitudinal sinus, are definite proof of past inflammation. On the base of the brain this form of chronic meningitis has the same distribution as that seen in the acute forms. The fourth ventricle may be obliterated by the adhesions, and the medulla may be adherent to the under surface of the cerebellum. In other cases there is distension of the "posterior cistern." The distribution of the inflammatory changes varies considerably. In one case described by Dr. Lees and Sir T. Barlow, the hydrostatic system of the brain and cord was divided into four sections—the right lateral ventricle, the left lateral ventricle, the third and fourth ventricles, and the sub-arachnoidal space of the spinal cord. The ependyma may be thickened, opaque, or granular. The choroid plexuses often shew pronounced changes; they are often thickened and matted, with disappearance of their normal fringed appearance, and small cysts are not uncommon. The middle ears, as a rule, are normal. It is true that there is often a muco-purulent secretion; but a purulent secretion is so common in the middle ears of children dying from any cause, that the incidence of this condition in meningococcic meningitis is certainly not greater than normal. The thoracic and abdominal viscera do not shew any constant lesions; but bronchopneumonia, exudation in the pleural cavity, and enlargement of the thoracic and abdominal glands may be present. In some cases pus may be found in the joints, or in the periarticular tissue.

Microscopical Examination.—The following description of the changes found is taken from a series of undoubted cases of "posterior-basic" meningitis examined by Dr. Gordon Holmes. The infiltration of the meninges is practically confined to the subarachnoidal space. There is no cellular invasion of the cortex, which, indeed, shews very little reaction except congestion of the vessels and distension of their perivascular spaces with small cells. The exudation contains: (1) cells with a relatively clear and large nucleus, often eccentric, which shews fairly coarse chromatic granules. The cell is generally round, with a large amount of finely granular protoplasm. (2) Smaller cells with very little protoplasm and deeply staining nuclei. These are most numerous round the vessels. (3) Polymorphonuclear cells, with a variable amount of poorly staining protoplasm. (4) Large, pale, and irregular nuclei, surrounded by more or less vesicular protoplasm. Many of the cells do not fit definitely into any class, and are probably degenerative forms. In sections stained by the Unna-Pappenheim method, the diplococci are very well brought out; they are most numerous when the polymorphonuclear cells form the majority of the cells present, and are often intracellular.

The nerve-cells, when stained by the Nissl method, shew an absence of tigroid, and the cytoplasm stains diffusely. A similar condition is seen in the spinal cord. From the more chronic cases the polymorphonuclear leucocytes are almost absent, and the proliferated connective-tissue cells are seen in various stages of evolution. This condition corresponds closely with that found by Speroni in epidemic cerebrospinal meningitis, and does not differ essentially from that in pneumococcic and streptococcic meningitis, except that, according to Southard and Stratton, abscesses are more liable to occur in the cerebral tissue in the two latter conditions.

The bacteriology of "posterior-basic" meningitis, which is closely connected with that of epidemic cerebrospinal meningitis, was first described by Prof. Still, who proved that the organism present in the disease was a diplococcus with certain definite characteristics. It may be intracellular or extracellular, varies in shape, but has the opposed surfaces flattened, and measures about 1·5 μ. The coccus is smaller than the pneumococcus, and has not any definite capsule. The organism stains by the ordinary methods, but not by Gram's method. This is now generally accepted, though quite recently the question has again been raised by Prof. Stuart M'Donald. It is aerobic, grows in the incubator at a temperature of

37° C., and does not grow at the ordinary room temperature. It grows on agar, glycerin- and blood-agar, and on the media known as "nasgar" and "chapasgar"; the growth is slightly raised, of a greyish-white colour, with a tendency to beading at the margin. On blood-agar its growth is very rapid and profuse, but is slower on blood-serum. It grows well in broth. It ferments glucose and maltose, but not galactose.

The diplococcus closely resembles the organism described by Weichselbaum in 1887 in cases of epidemic cerebrospinal meningitis. As pointed out by Prof. Still, its most striking difference from the *Diplococcus intracellularis* is its much greater vitality. Weichselbaum lays stress upon the remarkably short vitality of the *D. intracellularis*, and states that in no case did it live beyond the sixth day; whereas the diplococcus of "posterior-basic" meningitis almost always shews a maximum vitality of twenty-four to thirty-four days on ordinary media, and on blood-agar lives considerably longer. The diplococcus of "posterior-basic" meningitis is pathogenetic for guinea-pigs, rabbits, and mice, when inoculated intraperitoneally; subcutaneous inoculation produces little or no effect. Prof. Still concludes that, whilst the diplococcus of "posterior-basic" meningitis resembles Weichselbaum's *D. intracellularis* in many respects, it presents certain constant differences. "It seems probable that these differences are the result of natural variation, and represent rather a modification of characteristics than a distinction of kind." Netter has shewn that Weichselbaum's diplococcus grows in broth, and can be kept alive for years, so that the two chief cultural differences can no longer be insisted upon. On the other hand, further distinctions have now been established, so that the opinion expressed by Prof. Still some years ago still holds good. There is a considerable difference of opinion as to the relation which the organism of "posterior-basic" meningitis bears to that of epidemic cerebrospinal meningitis. That they are very similar in appearance, cultural reaction, and pathogenicity is certain; but there is no doubt that they do not correspond in their fermentation of certain sugars, do not give the same agglutination, and are not opsonised by the same serums to the same extent. The question of cultural characteristics, which has been fully considered by Dr. Gordon, by Drs. Shennan and Ritchie, and by Dr. Arkwright, is still under discussion. That some cases of meningitis are due to an organism of the *Micrococcus catarrhalis* class has been shewn by Drs. Arkwright and Wilson; on the other hand, Drs. Shennan and Ritchie found in every instance of 28 cases of cerebrospinal fluid examined, from cases of epidemic cerebrospinal meningitis, the characteristic Gram-negative diplococcus, and in only 3 instances found other organisms also present. Certainly in the cases of "posterior-basic" meningitis seen at the Children's Hospital between 1904 and 1909, the cultural characteristics and the fermentation-reactions have been most constant, and in but a few cases have there been other organisms present.

Opsonic Index.—It has been shewn by Miss Alice Taylor that cases of the sporadic form of meningococcic meningitis give a very high opsonic index when tested either with their own organism or one derived from a similar source; whereas, when tested with an organism from an epidemic source, a normal or low index is obtained. Drs. Houston and Rankin, working with material obtained from epidemic cases in Belfast and elsewhere, found that a very high opsonic index is obtained, and that the serum from cases of "posterior-basic" meningitis does not give this high index with the epidemic form of meningococcus. Probably this may serve as a distinguishing feature between these two varieties of organism. The index varies greatly, and during convalescence and recovery tends to assume a lower level.

Agglutination.—It has been pointed out by Dr. Gordon that there are difficulties in the use of the agglutination test for purposes of diagnosis, because it is impossible to obtain an emulsion of the meningococcus absolutely free from aggregations, and because the process of agglutination requires a long time. It has, however, been shewn, both by Taylor and by Houston and Rankin, that by testing the agglutination in conjunction with the opsonic index these difficulties are overcome, and that marked agglutination is shewn by the serum of patients tested against the organism obtained either from themselves or similar cases. These agglutination experiments are open to the objection that they are made in very low dilution, namely, 1 to 3. Agglutination experiments have, however, been carried out with the organism of epidemic forms of meningitis by Kolle and Wassermann, Kutscher, by Davis in America, and by Drs. Shennan and Ritchie in this country, and give in dilution of 1-20 to 1-50 reaction in one to two hours. The serum from a patient with the epidemic form of the disease will not agglutinate the organism obtained from a "posterior-basic" case, and, vice versa, the serum from a patient with the "posterior-basic" form of the disease will not agglutinate the organism obtained from an epidemic case. Probably, therefore, by combining the opsonic index and the agglutination test it will be possible to distinguish the organism of epidemic cerebrospinal meningitis from that of "posterior-basic" meningitis. Dr. Arkwright's recent work would seem to shew that although specific agglutination in high dilutions (1-500 to 1-2000) are obtainable, the variations in these reactions were not such as to indicate a specific difference between the epidemic and sporadic strains.

Clinical Features.—In the first place a brief account of the ordinary form of the disease will be given. A child, usually under the age of one year, well nourished and without any signs of disease, is suddenly taken ill with vomiting. The child often screams with pain, which is increased by any movement, and apparently has great hyperaesthesia of the skin. Sometimes the onset of the disease is attended by convulsions. Commonly the temperature is raised, the degree varying greatly in different cases. The child lies on its side with the arms and legs flexed and the neck stiff or even somewhat retracted. The fontanelle bulges, and sometimes presents as a prominent swelling. The upper eyelids are often retracted, and the conjunctiva is exposed above the upper edge of the cornea (Fig. 12). The eyes appear unduly prominent, but there is no true proptosis. The child, though not unconscious, appears not to take any notice of any object placed before its eyes, or even of light. Blindness is an early and frequent symptom in this disease. Squint may occur, but is not common. The pupils remain active to light, even in cases in which the blindness appears to be complete. Hearing appears to be acute; in fact, the acuity of hearing sometimes seems to be increased to such a degree as to be actually painful. Deafness is rarely observed. The child will suck and swallow well, and it is only in the late stages of the disease that there is any difficulty in this respect. The limbs in the early stages of the disease are usually flexed, and all movements can be performed; though the child resents, and does not willingly perform, any movement. The back is straight, or may be arched backwards. The abdomen is not retracted. The pulse is regular and generally rapid.

Various eruptions may appear on the skin. There may be a general redness, often occurring in patches, or there may be a vesicular rash, with the distribution of a herpes, or rarely it may be purpuric. The purpuric rash has not, however, the usual characters of a purpura, but begins by a vesicle around which haemorrhages occur, with induration of the skin and subcutaneous tissue. In some cases a joint may be swollen, or there may be periarticular swelling.

When the disease is severe, death may take place in two or three days from respiratory failure or convulsions. On the other hand, many cases pass into the chronic stage; in these the head becomes more and more retracted, the arching of the back increases, and the arms and legs become rigidly extended. The arms are rotated inward from the shoulder and the hands clenched. From this condition recovery may gradually take place, but the child is often left in an idiotic or mentally defective condition. In other cases, after a few weeks of illness, the head-retraction becomes less, and the child eventually makes a complete recovery, no sign of the disease remaining, except, perhaps, that the child is liable to attacks of temper.

We shall now consider the various symptoms in detail, and describe some of the rarer manifestations of the disease.

Vomiting is commonly the *first sign of the disease*, although convulsions also appear early. Dr. Lees and Sir T. Barlow state that out of 102 cases vomiting was the first sign in 33, convulsions in 29, and head-retraction in 24, the remaining cases being ushered in by screaming, irritability, or drowsiness. These are all early signs.

Bulging of the fontanelle is common both in the early and late stages of the disease; in the early stages it has been attributed to considerable hyperaemia of the brain. In the later stages it is often very considerable, and may be due to accumulation of fluid in the ventricles.

Head-retraction is the most characteristic symptom of the disease, both in the early and later stages, and is rarely absent, though it varies in degree. It appears always to be more marked when the meningitis affects the base of the brain rather than when the vertex is affected. Closely related to head-retraction is opisthotonos, or arching of the back. In both these conditions the muscles of the back are rigidly contracted, and any attempt to overcome this rigidity gives rise to severe pain.

Ocular Symptoms.—Retraction of the upper eyelids is commonly present, but varies in degree; it is present early in the disease, and often persists for many weeks. The type of the contraction is exactly similar to that seen on stimulation of the sympathetic; it is not, however, necessarily associated with dilatation of the pupil. Fig. 12 shews the condition in a very striking manner. Transient squints and irregular movements of the eyes are not infrequent, but a definite paralytic squint is not common in this disease. In the later stages of the disease squint may be present. A spontaneous nystagmus may be present in a few cases. It is most frequently seen in the later stages of the disease, and when there is blindness or considerable defect in vision. It is, however, a symptom to which but little importance need be attached. The pupils usually are equal, react briskly to light, and dilate to shade. In some cases the reaction is slow; in others there is considerable oscillation of the pupil. Optic neuritis is very uncommon. I have never seen a case of well-marked optic neuritis, and in most cases the margin of the discs and vessels are perfectly clear. In only 7 out of 42 cases of the disease was there any evidence of papillitis, and in only 3 was this inflammation distinct (Lees and Barlow). I have never found haemorrhages in the retina, even in cases with purpura. In 1 case there was a change about the macula very similar to albuminuric retinitis, which, after some months, cleared up entirely. This was probably due either to haemorrhage or oedema around the macular region.

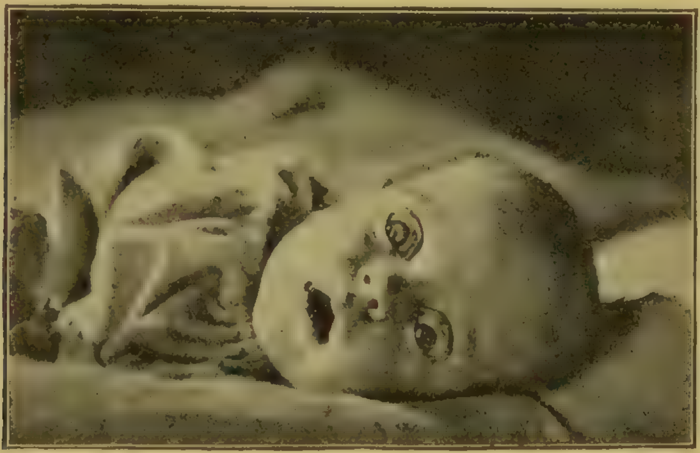

FIG. 12.—A case of meningococcic meningitis with retraction of the upper eyelids. This is an early sign, not due to hydrocephalus. (Photograph by Dr. H. Thursfield.)

Blindness is one of the most striking symptoms in some cases; it comes on suddenly, persists for several weeks, or may be permanent. It is an early symptom, and at first is commonly complete. The child is perfectly conscious, will speak and answer questions, but takes no notice of objects moved before the eyes. It is notoriously difficult in some infants to say if they see. The blinking of the eyelids is not a test on which too much reliance should be placed, either as a proof of the presence or absence of vision. In some cases the blindness rapidly passes off; in others it persists for several weeks, and then the child may regain sight completely; thus, I have seen a child recover good vision after being completely blind for four months. It has been suggested that the amaurosis may be due to inhibition of the lower visual paths; on the other hand, the ready reaction of the pupil to light with complete blindness would certainly suggest that the blindness was of cortical origin.

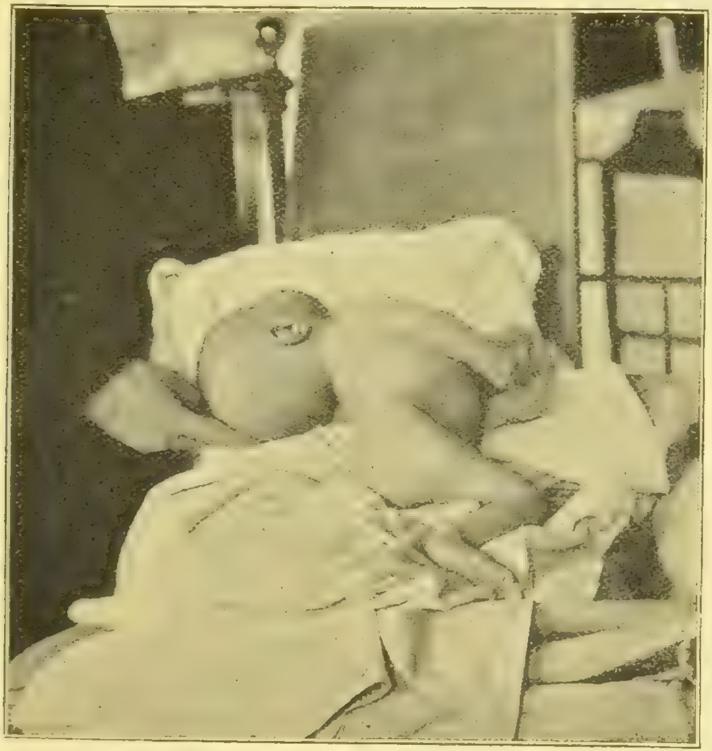

FIG. 13.—Marked head-retraction and rigid extension of right upper limb. (Lees and Barlow.)

Pseudo-glioma.—This condition has been met with in cases of meningitis. Drs. Lees and Barlow quote a case in which it was met with in a case of "posterior-basic" meningitis. I have met with this condition in children who have been said to have had meningitis, but I have never seen the condition appear in any case of meningococcic meningitis under observation.

Deafness.—In striking contrast to the frequency of deafness in cases of epidemic cerebrospinal meningitis is its absence in the sporadic form of meningococcic meningitis. The hearing is usually acute, and in many cases excessively so. Otitis media and otorrhoea are very rare.

Tonic spasm is a very characteristic feature. As affecting the muscles of the neck and giving rise to retraction of the head it is practically constant. In cases in which the inflammation is on the convexity, the retraction of the neck may not be marked, but there is usually rigidity. The spasm may involve the dorsal muscles of the back, and produce a condition of opisthotonos; but in some cases the head may be markedly retracted, whilst the trunk remains straight, or even flexed (Figs. 13, 16). Spasm of the limbs is usually present. When the spasm is of such a degree that the legs cannot be extended beyond a right angle

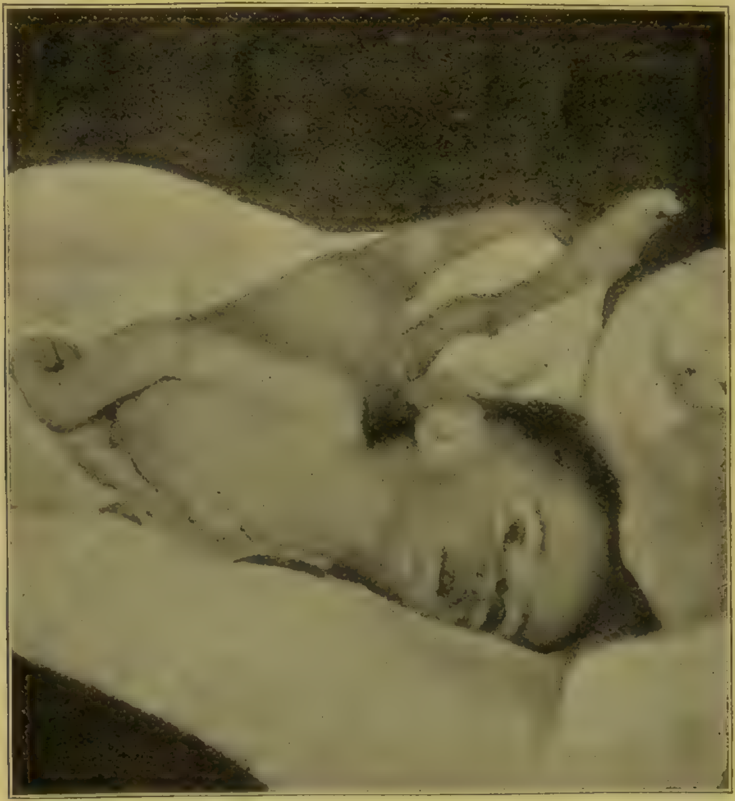

FIG. 14.—Head-retraction, marked opisthotonos, rigid extension of limbs, superpronation. (Lees and Barlow.)

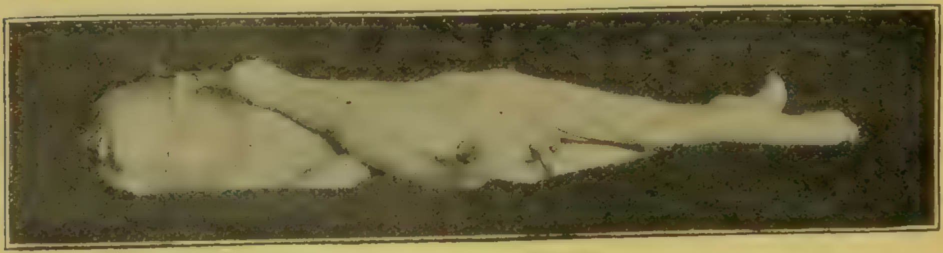

FIG. 15.—Head-retraction, rigid extension of limbs, but no opisthotonos. (Lees and Barlow.)

to the thighs flexed at right angles to the trunk, Kernig's sign is said to be present. In the earlier stages of the disease this spasm is commonly flexor, whereas in the later stages it is extensor. The upper limbs may be rigidly extended, whilst the lower are flexed, or vice versa. When the tonic spasms become extensor, the forearms are strongly adducted and rotated inward (Figs. 13, 14). The fingers are clenched and the thumbs

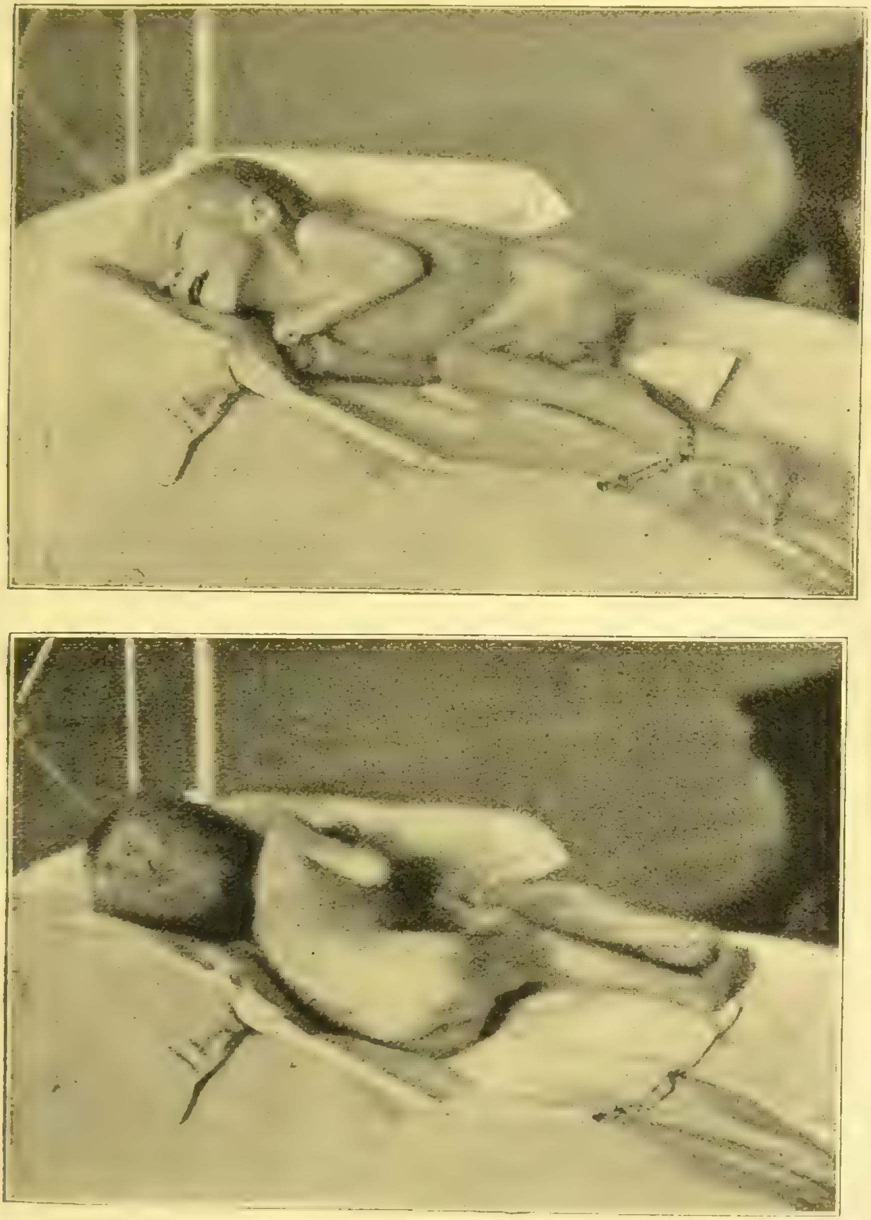

FIG. 16.—Moderate head-retraction, flexor spasm of limbs. (Lees and Barlow.)

inverted. All the joints of the lower limbs may be flexed (Figs. 15, 16), or all the joints may be extended and hyper-extended (Figs. 14, 16). The feet are in such cases generally inverted. Exacerbations of this spasm are not uncommon, the jaw and thorax muscles sharing in the contraction.

Clonic Spasms.—True convulsions with generalised clonic spasms occur, not only in the initial stages, but also during the course and as a final manifestation in the disease.

Paralysis occasionally occurs, for example, of the facial nerve, not due to disease of the petrous bone, and commonly there is weakness of the muscles of the neck and of the back during recovery. In Dr. Poynton's case of a child aged four months, the left arm was noticed to be paralysed before symptoms of meningitis appeared. There was also transient facial palsy, the condition of the left upper arm very closely resembling that in poliomyelitis. Power in the arm gradually began to return after six weeks, and was complete at the end of three months. The infant had also recovered from the meningeal symptoms during this period. Dr. Lees reports a similar case. Localised paralysis is undoubtedly a rare manifestation.

Crying and screaming are common at the onset and during the course of the disease. Occasionally the patient cries continuously, but more often he lies perfectly quiet, and only screams when touched or disturbed; sometimes the least noise is enough to cause screaming.

Respiration may be unaltered or accelerated with the rise of temperature. In some cases Cheyne-Stokes respiration may be present, whilst in others there is "cyclical breathing," that is, a series of three or four respirations of equal depth followed by a long pause.

The pulse is generally accelerated, regular, and does not shew the characteristic slowness and irregularity of tuberculous meningitis.

Temperature.—Almost any form of temperature chart may be met with; in some cases the temperature never varies more than two or three degrees, and is never above 101° F., but usually it is irregular, oscillating from 100° to 103° F. In some cases the temperature chart exactly resembles that of enteric fever, the temperature remaining persistently at 103° to 104° F., without any remissions. A form of temperature chart very characteristic of the disease shews sudden rises and falls of temperature which may occur with extraordinary regularity—it may be every twenty-four hours or at intervals of forty-eight or seventy-two hours. The rise will often occur in the morning, and the fall at night. This has been described by Dr. Box as the "Crises of posterior-basic meningitis." I have seen the temperature fall ten degrees in as many hours, from 105° to 95° F., the child's general condition altering very little with this change of temperature.

At the time of death temperatures of 107° F. and even higher often occur, or there may be a very marked fall, with a persistent low temperature for some days before death. During convalescence the temperature falls to normal.

Joints.—Periarticular abscesses occur, and yield the meningococcus in pure culture.

Nutrition often remains very good in spite of the vomiting and prolonged illness. A child, after three or four weeks of high temperature and severe cerebral symptoms, remains extraordinarily well nourished with firm and well-developed muscles. On the other hand, in the later stages of the disease, and especially if hydrocephalus be present, the most extreme wasting may take place and may persist for weeks.

Blood.—There is generally a leucocytosis of from 15,000 to 25,000 per c.mm. during the acute stage. In none of the cases under my observation has the diplococcus been cultivated from the blood during life, but it is difficult in young infants to obtain a sufficient quantity of blood for this. The organism has been found in the blood, not only in cases of meningitis, but also in cases of meningococcic infection without any evidence of meningitis.

Cerebrospinal Fluid.—In early cases the fluid obtained by lumbar puncture is always turbid, varying in degree from a slight turbidity to thick pus. It becomes turbid within forty-eight hours of the onset of acute symptoms, and may remain so from three to four weeks. Films from the cell-deposit shew a marked excess of polymorphonuclears varying from 65 to 94 per cent with intra- and extra-cellular diplococci. In later cases the fluid becomes less turbid, the number of cells diminishes, and the lymphocytic cells are increased relatively to the polymorphonuclears. In still later cases the cell-deposit becomes scanty, so that only a few degenerate cells can be found; the cultures are sterile.

Meningitis in older Children.—In older children the disease shews some variation. Dr. Langmead has pointed out that rashes are rather more frequent in older children than in infants, that the temperature is more liable to be irregularly intermittent, and that optic neuritis is more frequent.

Hydrocephalus is very often the result of the sporadic form of meningitis. The character of the fluid in the ventricles varies; in recent cases it is often turbid; in long-standing cases clear, and of a low specific gravity. That the hydrocephalus is due to the blocking of the communication between the fourth ventricle and the subarachnoid space can be proved in nearly every case. The hydrocephalus is in part due to retention of fluid normally secreted, but examination of the cerebrospinal fluid shews that the fluid has an inflammatory origin. In the sporadic form of meningitis a study of the post-mortem results seems to afford absolute proof that the accumulation of fluid is due to obstruction of the drainage channel. In some cases of meningitis there is no clinical indication of hydrocephalus, although it is sometimes revealed at the necropsy. In other cases the gradual increase in the size of the head and the prominence of the fontanelle point to hydrocephalus during life. In a series of 82 cases analysed by Dr. Lees and Sir T. Barlow hydrocephalus was manifest during life in 51; being considerable in 16, moderate in 18, and slight in 17; in the remaining 31 there was no evidence of hydrocephalus during life, though in some it was found after death.

Duration.—In acute cases, and especially in those with a purpuric rash, the disease is rapidly fatal, often running its whole course in three or four days. Less acute cases often live for several weeks and sometimes months, and finally die in extreme marasmus. A few cases recover from the acute symptoms, but die subsequently, often suddenly, from hydrocephalus.

Result.—*Mortality.*—Out of 94 cases 13 recovered completely and 2 others probably did, a total of 15 complete recoveries, or a proportion of 1 in 6. The fatal cases numbered 45, a mortality of nearly 48 per cent (Lees and Barlow).

Recovery is often incomplete; some cases remain permanently hydrocephalic; others remain blind; whilst others again are left more or less mentally defective or idiotic.

The **prognosis** as regards life is by no means unfavourable, especially when compared with that in other forms of meningitis. Of the cases admitted to hospital 50 per cent survive; of these, however, only about 15 per cent recover completely, and the rest are left with hydrocephalus, blindness, or mental defect, varying in degrees from increased irritability to absolute idiocy. In view of the favourable effects obtained in the epidemic form of the disease by the use of the Flexner-Jobling serum, it is to be expected that a similar result is to be obtained by the use of some such serum for the sporadic cases. So far the results have not been satisfactory. Recovery has followed the use of serums, but it cannot be said that the percentage mortality has fallen. The beneficial effects of lumbar puncture have been alluded to by Mr. A. Barker, but although this method has had an extended trial, it would be difficult to assert that it has had any effect in diminishing the mortality. Individual cases will sometimes make the most unexpected recovery, after the injection of serum or lumbar puncture or with the administration of iodide or mercury, but unless the total mortality over a long series of cases be shewn to be diminished, it is unsafe to attribute the cure to one or other of these methods. In connexion with this, it is of interest to compare the figures given by Robb and Watts as to the effect on the mortality produced by the use of serums in cases of epidemic cerebrospinal meningitis. In any individual case it is most difficult to assert the degree to which recovery will take place. Even after apparent complete recovery, it will be found that as the child develops there is considerable mental impairment. On the other hand, a child may lie in a semi-comatose condition for many weeks with blindness, and yet may make a complete recovery. Varying degrees of hydrocephalus and mental defect are the most common sequels of meningitis.

The **diagnosis** of a characteristic case of meningococcic meningitis presents but few difficulties. The history of an acute onset in a healthy infant, with marked retraction of the head, at once suggests meningitis. Retraction of the head is sometimes present in *otitis media*, but puncture of the membrane often rapidly relieves this symptom. In apical *pneumonia*, retraction of the head and other symptoms suggesting meningitis may be present, and it is often difficult to arrive at a correct diagnosis from the physical signs alone, as there may not be any signs of pneumonia for several days. *Retropharyngeal abscess* also causes retraction of the head, but it is only necessary to bear this in mind to avoid an error in diagnosis. Again, in *mumps* and *enlarged cervical glands* retraction of the head may be a prominent symptom, but should seldom give rise to symptoms likely to be mistaken for meningitis. In *thrombosis of the longitudinal sinus*, retraction of the head and other cerebral manifestations may give rise to considerable difficulty in diagnosis.

Difficulty in diagnosis may also arise in some cases of enteric fever with cerebral symptoms, in renal disease, sarcoma of the brain, and gastro-intestinal irritation due to worms. Cerebral sclerosis, associated with a form of syphilitic meningitis, occurs in infants and closely resembles "posterior-basic" meningitis; blindness and retraction of the head are the prominent symptoms, and hydrocephalus may also be present. There is, however, in these cases no history of acute onset, and the statement of the mother is that the disease has existed from birth or shortly after birth.

Lumbar puncture is a ready and safe means of diagnosis in cases of difficulty. With the technique of the method of lumbar puncture it is unnecessary to deal (*vide* Vol. VII. p. 275). In most cases the fluid flows through the needle in a stream under pressure.

In almost every recent case it is possible to obtain the organism characteristic of the disease, the *Diplococcus intracellularis*, either in culture or in stained specimens of the fluid. In the late cases the organism may fail to grow, and yet may be found in stained specimens.

The character of the fluid and the nature of the cells present are of considerable diagnostic value. The fluid is almost always turbid, and contains a large excess of polymorphonuclear cells. In tuberculous meningitis an increased percentage of polymorphonuclear cells is rare, the nature of the cells being usually lymphocytic, the percentage of which is often about 91; in tuberculous meningitis the fluid is commonly clear. These two points, apart from the discovery of tubercle bacilli in the small flakes of lymph, are, as a rule, sufficient to make the differential diagnosis between "posterior-basic" and tuberculous meningitis clear. From pneumococcic and other forms of infective meningitis, however, the nature of the fluid and the cell-count do not form such a ready method of diagnosis; the diagnosis depends on the identification of the organism present.

In late cases of the sporadic form of meningitis there may be an excess of lymphocytes, and the differential diagnosis between this and tuberculous meningitis, on the one hand, and a syphilitic meningitis on the other, might arise. The clinical symptoms and the history, in such cases, would in most cases afford help to the correct diagnosis.

The diagnosis from epidemic cerebrospinal meningitis may be difficult. The symptoms may be exactly similar, the cerebrospinal fluid and the organism in staining and cultural characteristics may appear exactly alike, and it is only by testing the opsonic index and the agglutinative power of the blood that any distinction can be made (see p. 172).

Treatment.—For the sake of convenience treatment may be divided into that of (1) the acute stage, and (2) the late stage.

(1) *The Acute Stage.*—The children are extremely susceptible to the least touch, movement, or noise; so long as they are left alone, they will often remain perfectly quiet, but the approach of any one to the bed, or the hand placed on the child, will at once provoke a scream. It is therefore important that those in charge of the children, who might naturally try various soothing methods, should leave them alone as far as possible. The maximum comfort and ease should be ensured by a water-bed or air-cushion. As the children usually swallow well, there is little difficulty in feeding them, but vomiting may be troublesome, and if the usual remedies fail, washing out the stomach often gives good results. For a high temperature tepid sponging will be of service, but in the large majority of cases this is unnecessary. Constipation is not, as a rule, troublesome, and yields to the usual remedies. Dr. Lees and Sir T. Barlow advocate the puncture of the membrana tympani, and state that they have seen cases rapidly recover after this procedure. It is difficult to understand how this treatment can do good in definite meningitis, although no doubt it is of great service in meningism due to otitis media.

Lumbar puncture has been advocated and tried as a method of treatment, and in some recorded cases has been followed by recovery. When employed, it may be performed every day, in order to diminish the intracranial pressure. The amount of fluid withdrawn should be regulated by the amount of pressure; on the first occasion when the needle is inserted, the fluid often spurts out in a stream, and then gradually diminishes, till it comes drop by drop; 30 to 40 c.c. may with safety be removed from an infant under such conditions. On subsequent occasions the amount of fluid obtained is very much less, sometimes a few drops only. The character of the fluid is a most excellent record of the progress of the case; at first cloudy, with a large excess of polymorphonuclear leucocytes and abundant meningococci, it gradually becomes clear, the proportion of the polymorphonuclear leucocytes diminishes, the lymphocytes increase, and the meningococci disappear from the fluid. Hohn has shewn that the amount of albumin in the cerebrospinal fluid also diminishes as improvement takes place. The fluid then becomes normal in appearance, containing a slight excess only of lymphocytes. Such a course would seem to point to recovery, and in a certain number of cases it may do so, but in others it merely indicates subsidence of the acute stage, which is then succeeded by the chronic stage. For the relief of increased intracranial pressure, headache, and convulsions, lumbar puncture may well be employed, but it has yet to be proved that it is of real value as a curative agent. If the first puncture is "dry," a second may be made at a higher level. Not infrequently, although the first and second punctures are successful in withdrawing fluid, subsequent attempts are "dry."

Closely connected with the treatment by lumbar puncture is that of intrathecal injections of chemical substances and of serums. Various salts of silver have been used, and necropsies prove that they reach the cervical portion of the cord, and even pass to the cranial cavity. No marked success has attended these injections.

Serum Treatment.—It has been shewn by Flexner and by Robb that this treatment increases the number of recoveries in epidemic cerebrospinal meningitis, but, unfortunately, this has not yet been proved to hold good for the sporadic form of meningitis. A serum has been prepared with the strain of organism derived from the sporadic form of meningitis by Dr. J. A. Arkwright at the Lister Institution, but it has not yet been shewn that this serum has any very marked effect, though success has followed its use in a few cases. No harm, however, seems to result from the injection of horse-serum into the spinal canal. The method is quite simple, for immediately after the required quantity of cerebrospinal fluid has been withdrawn, a quantity of 10 to 20 c.c. of serum can easily be injected by attaching the syringe to the needle used for lumbar puncture. The serum should be warmed before being injected. These injections may be repeated daily till improvement takes place; this is indicated by the fluid becoming clear and by the diminishing vitality and disappearance of the organism. The serums known under the name of Flexner, Kolle and Wassermann, and Ruppel have also been used for intraspinal injection, and successful results have been reported, but no series of cases in which any one serum was employed has been recorded.

Hohn suggests that after the injection of the serum the patient should be placed in a position in which the lumbar region is higher than the cervical, so that the serum may gravitate towards the cranial cavity.

Vaccine Treatment.—This form of treatment has been tried, and cases thus treated have recovered, but evidence is wanting to shew that the cure was due to the vaccin. The opsonic index in the sporadic form of meningitis is normally so high that there would seem to be no indication for raising it further. The index of the cerebrospinal fluid is low, and the introduction of a serum into the cerebrospinal canal would appear the more rational mode of treatment, and is practically attended with good results.

Drug Treatment.—During the acute stage bromide and chloral are useful in reducing the irritability of the patient. Mercury also appears to be of some service, and should be administered by inunction.

In the later stages iodides appear to be of special service. Cases in which all the symptoms seemed to point to a downward course of the disease have almost at once begun to improve on the administration of iodides. Children do not always tolerate the drug well, and in this event it should be given in the form of subcutaneous injections of 10 to 20 ♏ of iodopin. For this purpose a rather thick hypodermic needle is needed, and the syringe and the fluid should be warmed before the injection is given.

(2) *Treatment of the late stage* is concerned with the resulting hydrocephalus. Owing to the blocking of the foramina in the neighbourhood of the fourth ventricle, either by adhesion or by the formation of a "pressure cone," the ventricular fluid no longer passes freely into the arachnoid space of the spinal canal, and gradually the lateral ventricles and the iter between the third and fourth ventricles become distended.

It is in young children, in whom the fontanelle is unclosed, a simple method to puncture the distended ventricle and allow the intraventricular fluid to escape—such a procedure gives but temporary relief, and the ventricles rapidly refill. Various attempts have been made to re-establish the communication between the ventricles and the subarachnoid space by surgical methods, and although one or two cases have been successful, so far as the preservation of life is concerned, yet the mental impairment which remains is very considerable.

2. PNEUMOCOCCIC MENINGITIS.—Pneumococcic meningitis may be divided into two classes:—(1) Primary. (2) Secondary.

Primary pneumococcic meningitis includes cases in which the meninges are affected by the pneumococcus, no focus of infection being discoverable in the body. Clinically they form a definite group; pathologically they cannot be separated from other forms of pneumococcic meningitis. Out of 30 cases of pneumococcic meningitis collected from the post-mortem records of the Children's Hospital, Great Ormond Street, 7 may be regarded as primary.

Clinical Features.—The history is often indefinite; there may have been a fall some few days or weeks previously, with subsequent headache and vomiting. In the early stages of the disease the child may present very few symptoms of meningitis; the raised temperature, the vomiting, and the headache not suggesting anything more than the onset of some acute disease. Gradually, or it may be suddenly, symptoms pointing to some form of intracranial disease appear; unilateral or bilateral convulsions may occur; restlessness, irritability, and delirium follow one another in rapid succession, and the child becomes drowsy and comatose. Squint, rigidity of the neck, and localised paralysis may all be present. The symptoms do not differ from those in other forms of meningitis except in the rapidity with which they appear. The temperature, however, is generally higher than in other forms of meningitis.

The *diagnosis* is as a rule easily made by lumbar puncture, and the removal of turbid purulent fluid, which on microscopical and cultural examination shews the presence of the pneumococcus. Two points, however, must be borne in mind; the pus within the meninges may be so thick that it will not pass through a needle, so that the puncture is "dry," or in rare instances perfectly normal cerebrospinal fluid is obtained. I have seen this on one occasion only; in a child diagnosed as generalised pneumococcic infection with meningeal symptoms, the cerebrospinal fluid obtained by lumbar puncture a week before death was perfectly normal, and yet the necropsy revealed well-marked and extensive pneumococcic meningitis. This shews how rapidly infection may occur and lead to a fatal termination.

Secondary pneumococcic meningitis includes cases in which the meningitis is secondary to pneumococcic infection of other parts of the body. Pneumococcic meningitis is often a terminal event and passes almost unnoticed, or is discovered only at the necropsy. It would appear to be most common after empyema and ear diseases; out of 23 cases of secondary pneumococcic infection, 7 followed empyema and purulent pericarditis, and 7 ear disease. Other cases followed pneumonia, abdominal infections, abscesses, and joint infection.

The *clinical features* do not differ from those of other forms of meningitis, except that some cases present very few symptoms. Headache and vomiting may be present, and convulsions are not infrequent.

Morbid Anatomy.—The appearance of the surface of the brain in acute pneumococcic meningitis is very striking. After removal of the dura mater the vertex of the brain is seen to be covered with a thick, purulent, greenish exudation contained in the meshes of the pia-arachnoid, or between it and the dura mater. The vertex of the brain is commonly affected, but in some instances the base only is involved, and it is not uncommon to find both vertex and base thickly covered with exudation. The cortex of the brain may or may not shew considerable hyperaemia, and in some cases there is vascular congestion of the grey and white matter, the cut surfaces of the brain presenting numerous minute haemorrhages. On the other hand the brain substance may appear perfectly normal. Some pus may be found in the lateral ventricles, but usually the fluid is normal in appearance. There is almost invariably exudation on the posterior surface of the spinal cord. In the absence of a bacteriological examination it is often difficult to distinguish a meningitis due to the pneumococcus from one due to the meningococcus or to other micro-organisms.

Microscopically the exudation is characterised by a greater amount of fibrin than in other forms of meningitis. The cells present are chiefly polymorphonuclear neutrophils, with a certain number of medium mononuclear cells having the character of macrophages, and very few lymphocytes (Speroni).

Diagnosis.—The diagnosis rests upon obtaining evidence of the presence of the pneumococcus in the cerebrospinal fluid.

Prognosis.—It is doubtful if recovery ever takes place in any severe case of pneumococcic meningitis. There seems to be no reason why recovery should not occur as it does from other forms of meningitis; but I do not know of any evidence that recovery does take place.

Treatment.—No treatment is known to have any marked effect on the disease—symptoms may be relieved by lumbar puncture, but permanent benefit does not result.

STAPHYLOCOCCIC AND STREPTOCOCCIC MENINGITIS.—**Etiology.**—Streptococcic and staphylococcic meningitis is rarely, if ever, a primary infection of the cerebral or spinal meninges. Cases occur in infants a few days to a few weeks old in which it is sometimes difficult to detect the source of infection, but in nearly all of them the infection would seem to arise at the umbilicus. In older children the common source of infection is the ear; other cases arise as the result of infection of the bones or joints, and a few are secondary to a streptococcic empyema. In 9 cases reported by Southard and Stratton, 8 of which were adults, 5 shewed otitis media, 1 disease of the nasal sinuses, and 1 erysipelas.

Morbid Anatomy.—The meninges of the vertex and base are usually involved, and the whole surface of the brain may be covered up with a thick yellow or green exudate. The exudate spreads along the spinal cord and involves the dorsal surface to a greater extent than the ventral. The cortex of the brain is generally flattened.

Microscopically the condition does not differ from that in pneumococcic meningitis. The meshes of the pia mater are distended with cells, large mononuclear cells and polymorphonuclears being present in varying proportions. Many of the mononuclear cells are phagocytic for polymorphonuclear leucocytes and also for chains of streptococci. The walls of the cortical veins may be distended with polymorphonuclear leucocytes, and the arteries also exhibit the deepest infiltration of the intima with polymorphonuclears. The brain and spinal cord often present but little change considering the marked affection of the meninges.

The **symptoms** do not differ from those in other forms of meningitis, namely, malaise, headache, vomiting, squint, and a rise of temperature followed by restlessness, delirium, and coma. The respiration and pulse may at first be slow and irregular; then the former assumes the Cheyne-Stokes character, whilst the latter becomes rapid and feeble. Coma becomes deeper and the patient dies.

The **diagnosis** is not, as a rule, difficult; the signs of the disease are well marked. The greatest difficulty, however, arises in distinguishing cases in which the symptoms may be due to localised cerebral or cerebellar abscess from those in which the infection is more general. In abscess the symptoms are more localised, the headache, vomiting, and optic neuritis more pronounced, and the symptoms often of longer duration. The examination of the cerebrospinal fluid removed by lumbar puncture should aid the diagnosis; for in meningitis the fluid will be turbid, containing many polymorphonuclear leucocytes and streptococci; whereas in abscess the fluid is clear and contains but few cells and no organisms. In practice, however, this sharp line of distinction cannot be maintained, and from a case of cerebral abscess cerebrospinal fluid may be obtained, which resembles that in meningitis. The diagnosis of streptococcic and staphylococcic meningitis from other forms of meningitis can only be made by the bacteriological examination of the cerebrospinal fluid.

Prognosis.—Streptococcic and staphylococcic meningitis is usually, if not always, fatal. It often occurs as a terminal manifestation in a general streptococcic infection.

Treatment.—Symptoms may be relieved by lumbar puncture, but there is no effectual treatment for the disease.

INFLUENZAL MENINGITIS.—Meningitis due to the influenza bacillus, although somewhat rare, is now well recognised. Pfühl in 1892 first shewed the presence of the Pfeiffer's bacillus in the meningeal exudate. The disease occurs both in children and adults. The onset of cerebral symptoms may be preceded by an ordinary attack of influenza, or the cerebral symptoms may be a terminal manifestation in which the primary symptoms have been pulmonary or arthritic.

Morbid Anatomy.—The exudate over the vertex and base of the brain is fibrino-purulent and fills the subarachnoid space: with suitable staining numerous bacilli can be seen which are extra-cellular in position. There may be morbid changes in the other organs, from which the organism can be obtained in pure culture. Some cases of post-influenzal meningitis are due to organisms other than the influenza bacillus.

The **clinical symptoms** do not differ from those of other forms of meningitis; the two following cases recorded by Ghon may be taken as examples.

A man, aged thirty-three, was taken ill with fever, loss of appetite, cough, thirst, and diarrhoea. Five days later he had pain and swelling in the right eye, with stiffness in the neck and back, and frequent vomiting. Two days later he became unconscious, had great rigidity of the neck, and died. The fluid drawn off by lumbar puncture shewed a marked leucocytosis and red blood-corpuscles; no organisms were found in the fluid. The necropsy revealed extensive fibrino-purulent meningitis of the vertex and base of the brain, with injection of the frontal and antral sinuses. Bacteriological examination of the meningeal exudate shewed a large number of Gram-negative bacilli, which both morphologically and culturally were identical with Pfeiffer's influenza bacillus. The microscopical examination of the exudate over the brain shewed numerous bacilli both intra- and extra-cellular.

The second case was that of a breast-fed child eight months old, who was taken ill with fever, vomiting, and cough. On the fifth day after onset the child had some convulsions which recurred on several occasions. Rigidity of the neck and back was present. Five c.c. of a blood-red fluid obtained by lumbar puncture contained numerous polymorphonuclear leucocytes and a small Gram-negative bacillus, corresponding in its cultural characteristics with those of the influenza bacillus.

It is difficult to state with what frequency influenzal meningitis occurs. Out of 100 necropsies at the Children's Hospital, Great Ormond Street, on cases of meningitis other than tuberculous no known case of influenzal meningitis occurred.[1] Dr. Douglas states that Dubois collected 11 cases of meningitis in which the influenza bacillus was present; a necropsy having been made in 9 cases, whilst in the remaining 2 the organism was found during life. Dr. Douglas's case is the most complete of any in English literature. A child, aged ten months, was taken ill with symptoms of meningitis, rigidity of the neck, and prominence of the anterior fontanelle. The temperature, pulse, and respiration were all raised. The child passed into a comatose condition and developed a squint. The cerebrospinal fluid obtained by lumbar puncture contained numerous polymorphonuclear leucocytes and much albumin, and was yellow in colour. The fluid contained a feebly staining short bacillus, which culturally and microscopically corresponded to the influenza bacillus. The child passed into a convulsive state and died on the eighth day after the onset of the disease. At the necropsy an extensive purulent meningitis was found over the vertex and base of the cerebrum, the cerebellum, and along the posterior and lateral aspect of the spinal cord. Pure cultures of the influenza bacillus were obtained from the exudation.

---

[1] Since these figures were collected in 1907, 5 cases of influenzal meningitis have occurred in the hospital with 4 deaths and 1 recovery. Other cases have been recorded by Cohoe (*Amer. Journ. Med. Sc.*, Phila., 1909, cxxxvii. 74).

An infant with a swelling around the elbow-joint due to creamy-yellow pus and yielding a pure growth of the influenza bacillus developed meningeal symptoms and died. Purulent meningitis was found over the vertex and base, and the intra-ventricular fluid was also purulent. A pure growth of the influenza bacillus was also obtained from the cerebrospinal fluid (Dudgeon and Adams). Similar cases are reported by Pfühl, Judson Bury, Saundby, and others. Cohen has recently again called in question many of the reported cases of influenzal meningitis. He states that the organism found is fatal to guinea-pigs and rabbits, and that it is possible to vaccinate an animal against the infection, and the serum from such an animal possesses great antitoxic powers, whilst the serum of animals vaccinated against the Pfeiffer bacillus does not possess any antitoxic powers.

The **diagnosis** can only be established by obtaining the organism from the cerebrospinal fluid.

The **prognosis** is extremely grave, but even in cases in which the Pfeiffer bacillus is present in the cerebrospinal fluid removed by lumbar puncture recovery may take place (Thomesco and Gracoski). Recovery is reported in many other cases in which the diagnosis has not been made conclusive by the finding of the organism.

Treatment.—No special line of treatment can be advocated. Repeated lumbar puncture may be practised for the relief of intracranial pressure, but it is doubtful if this has any curative effect.

RHEUMATIC MENINGITIS.—It is doubtful whether we are yet in a position to describe rheumatic meningitis, rather than meningitis in the course of rheumatic fever. That meningitis occurs in association with rheumatism is admitted, but it is argued that such meningitis is due to some secondary affection of the meninges.

Drs. Poynton and Paine record a case of a boy, aged thirteen years, who had an attack of arthritis of the ankle and knees in August. It appeared to be an ordinary attack of rheumatic fever, a mitral systolic murmur being present. Under suitable treatment the boy rapidly got well and steadily convalesced till

Sept. 17, when, without apparent cause, his temperature rose to 102·4° F.; he was sick and complained of headache. On the 19th he became unconscious with fixed and dilated pupils and general muscular rigidity alternating with flaccidity, and he died on that day, with a rectal temperature of 106° F. At the necropsy there was meningitis almost entirely basal in distribution. A good deal of turbid fluid was present together with flakes of exudation over the medulla, pons, and cerebellum. There was also a general spinal meningitis. Cultures from the cerebrospinal fluid contained a pure growth of a diplococcus which had an unusually high virulence and was capsulated, but otherwise corresponded to the *Diplococcus rheumaticus.* The authors believe that the case was a true rheumatic meningitis, in spite of the fact that capsulation was observed in the organisms obtained from the blood of the rabbit experimentally inoculated.

Other observers have shewn the diplococcus to be present in cases in which there were marked microscopical changes, but no gross meningitis. Drs. Poynton and Paine consider that rheumatic meningitis corresponds to severe rheumatic pericarditis.

MENINGITIS DUE TO BACILLUS ANTHRACIS.—Meningitis due to infection by the *Bacillus anthracis* is rare; Schultze states that in spite of the bacillus being abundant in the blood, meningitis occurs but seldom, and is then due to some other infection. Symmer and Wilson, however, report the case of a woman, aged nineteen, in whom the pustule appeared on the forehead—six days later she developed symptoms of meningitis and died. The brain after removal of the dura mater was of a brilliant red colour, as if it had been smoothly and thickly covered over with red paint. The cerebrospinal fluid was abundant, turbid, and furnished on culture a pure growth of *Bacillus anthracis.* Microscopically the meninges shewed an enormous accumulation of polymorphonuclear leucocytes, red cells, and fibrin, and a large number of anthrax bacilli. The meninges alone were affected and no change was found in the brain substance.

MENINGITIS IN MUMPS.—It is well established that in a small proportion of cases of mumps meningeal symptoms occur. Elsaesser in an epidemic of 300 cases observed 3 cases which died with symptoms of meningitis. There are fairly numerous cases of this form on record, but a satisfactory pathological examination is wanting.

In one case reported by Maximowitch, a hyperaemia of the membranes and a sero-fibrinous exudation was found.

Until further and more exact information is obtained these cases should be classed under the title serous meningitis.

MENINGITIS DUE TO STREPTOTHRIX INFECTION.—Streptothrix infection of the meninges is a rare condition. It is most commonly found as an extension from a focus in the skull or soft parts of the face or throat, but some cases of primary affection of the membrane have been reported. Sometimes true metastatic foci have been found in the meninges secondary to infection in other parts of the body.

In many cases the infection is not limited to the membranes but spreads into the brain substance. The following case published by Drs. Beevor and F. Buzzard is one of the few recorded in English literature :—

A woman, aged thirty-seven, had had good health except for occasional epileptic fits. Three weeks before admission to the hospital she had severe attacks of vomiting followed by headache, pain in the back of neck, and failing sight. A week later she had difficulty in swallowing and her speech became husky. On admission she had severe headache with tonic contraction of the neck muscles causing her to rotate her head to the left. There was no retraction of the head and no optic neuritis. There was weakness of the ocular muscles but no paralysis of the limbs. The temperature was irregular. Three days after admission she became worse and passed into a condition of stupor with retraction of the head and arching of the spine. The case was diagnosed as one of basal meningitis. The patient died fifteen days after admission to the hospital.

The vertebral column and spinal meninges appeared normal, but on removing the brain a well-marked suppurative meningitis was found at the left base extending from the optic chiasma in front to the upper end of the first cervical segment behind, and limited sharply internally by the mesial line of the pons and medulla. There was caries of the body of the sphenoid which extended in the direction of the posterior nares, but the source of infection could not be found. All the internal viscera were examined with a negative result. Smears of pus taken from the base of the brain stained by Gram's method shewed the presence of an abundant streptotricial organism in the form of mycelial filaments. As the organism failed to grow in any medium, the cultural characteristics were not obtained.

Howard, writing in 1902, states that he has been able to find only 5 cases in which actinomycosis was primary in the central nervous system, and 13 in which it was secondary. In none of the 5 primary cases was there definite evidence of infection of the meninges. Of the 13 cases in which there was secondary infection of the nervous system, the membranes were found to be affected in 7 (Ponfick, Naunyn, Keller, Eppinger, Delore, and two of Chiari's cases). The most complete case is that reported by Eppinger, of which the following are the details.

The patient, aged fifty-two, developed what was apparently joint rheumatism. Eight days later he was unable to work. Later there was paralysis of the left side, tenderness of the right side of the head, delirium, and coma. Death occurred after an illness of twelve days. At the necropsy there was well-marked cortical, basal, and spinal meningitis, and on examination a branching, thread-like organism was obtained which produced a pseudo-tuberculosis in guinea-pigs and rabbits.

Prof. Stuart M'Donald has described a case of general streptothrix infection in which although the brain contained numerous abscesses, and the pia-arachnoid was congested, there was no evidence of any streptotricial infection of the meninges. (For the "Pathology of Streptothrix Infections," see Vol. II. Part I. p. 302.)

The Diagnosis can only be made by finding the streptotricial organism in the fluid removed by lumbar puncture.

Treatment.—In the event of the organism being found in the cerebrospinal fluid, large doses of iodide of potassium should be tried. The possibility of treating the condition by a vaccin has been raised by the success which followed such treatment in a case of actinomycosis of the lungs published by Dr. Wynn.

Gonococcic Meningitis.—A considerable number of cases of cerebrospinal meningitis occurring in the course of a gonorrhoeal infection have been reported. In 1894 Barrie collected some 30 cases, in 1898 Kahane added to this number, and the subject has been fully dealt with by Drs. Henderson and Ritchie (1909). Weichselbaum in 1903, although many instances of supposed gonococcic infection had been published, asserted that the etiological relation of the gonococcus to meningitis has not been definitely established in a single case. Since that time cases have been published by Block, Wynn, and Prochaska; but in none of these can it be said that the organism obtained from the cerebrospinal fluid was proved to be the gonococcus. De Jong reported a case in 1908, presumably the same as that recorded by Rombeck in 1907, in which he seems to have definitely proved that the organism was the gonococcus. In 1909 Drs. Henderson and Ritchie published a further case of undoubted gonococcic infection.

Treatment.—Removal of the cerebrospinal fluid by lumbar puncture relieves the headache. Cases of recovery are reported, but these have not followed any one line of treatment. The cases reported by Drs. Henderson and Ritchie and by de Jong recovered completely.

Typhoid Meningitis.—Many cases of enteric fever present cerebral symptoms, but in very few can meningitis be shewn to be present; thus, Hoffmann found 4 out of some 250 cases of enteric fever. Typhoid meningitis may be divided into two main groups: (1) In which the meninges are affected without any other lesion; (2) in which meningitis occurs in the course of enteric fever.

The *first group* is small; out of the few cases reported, Lavenson's may be quoted.

A woman, aged thirty-six, had for some weeks before the onset of her illness nursed her brother through a severe attack of enteric fever, which she had had many years previously. Her illness began with severe headache, vomiting, photophobia, and great muscular soreness; there was suppression of urine for thirty-six hours. The temperature was raised and the patient slightly stuporous. When first examined nothing abnormal could be detected. Kernig's sign, at first absent, appeared after a few days, the neck became rigid and the patient delirious. There was a slight leucocytosis, and the agglutination reaction, negative on the fifth day, was positive on the eighth day. Lumbar puncture was performed and a pure culture of undoubted *Bacillus typhosus* was obtained. Necropsy revealed a large patch of purulent exudate over the right cerebral hemisphere. There was no intestinal lesion and no enlargement of the spleen.

In a similar case, published by Henry and Rosenberger, the fluid obtained by lumbar puncture was purulent and gave a pure culture of the typhoid bacillus.

Group 2.—The second and by far the larger group is that in which meningitis occurs in the course of enteric fever. These cases may, for convenience, be divided into two main categories: (*a*) serous meningitis; (*b*) purulent meningitis. These two groups pass from the one into the other. A third group is described in which symptoms of meningitis are present, but in which no lesion exists; these cases are known under the title meningism (*vide* p. 199).

Morbid Anatomy.—The following description is given by MacCallum. The meninges of the whole length of the cord are covered with yellowish turbid fluid containing numerous mononuclear cells and some polymorphonuclear leucocytes. The brain is also covered with this yellowish exudate, which is not so opaque as in ordinary meningitis. The ventricles contain turbid fluid but no exudate, and there is very little exudate on the surface of the meninges, the bulk of it infiltrating the tissue-elements between the meninges and the brain. The exudate contains a considerable quantity of fibrin, but it is especially rich in cells of remarkably various forms. In certain areas there are abundant accumulations of polymorphonuclear leucocytes, in other parts the exudate is composed chiefly of mononuclear cells. There are all transitional forms, from small cells in every respect resembling the lymphocytes of the blood to very large cells. Plasma-cells of typical form and staining reactions are present in considerable numbers, especially in the neighbourhood of the veins. In most places, however, the predominant cell is the much larger and more irregular mononuclear phagocyte; its protoplasm is frequently stretched out into the most fantastic contour, and its diameter is three to five times that of a polymorphonuclear leucocyte, and its nucleus is large, vesicular, and has an extremely abundant, hyaline, pink-staining protoplasm. The veins are distended with blood, and their walls infiltrated with cells of various forms. In the arteries the endothelium is often raised off the basement membrane by masses of cells. The typhoid bacillus can be demonstrated in the exudate. Nothing else in the anatomical picture is absolutely peculiar to typhoid meningitis.

The serous form of meningitis shews slight clouding and oedema of the pia-arachnoid, which on microscopical examination is seen to depend on infiltration with mononuclear and polymorphonuclear cells. In the brain there is small-celled infiltration of the sheath of the vessels of small and moderate size.

Silberberg has shewn the presence of organisms resembling typhoid bacilli in the cerebrospinal fluid of seven out of nine cases of enteric fever, but in only one of these did cultures give a positive result. Eight cases of typhoid meningitis in children followed by recovery are recorded by Rocas and Carles, but it is doubtful if in all of these there was true infection of the meninges.

Wentworth describes meningitis, due to the typhoid bacillus, in a child aged four. In the cerebrospinal fluid obtained fourteen days before death no organism could be found, but from a specimen taken immediately before death an abundant culture was obtained. The agglutination reaction was negative.

Symptoms.—The symptoms do not differ from those in other forms of meningitis. Headache, drowsiness, coma, restlessness, and delirium are the constant features. Convulsions, stiffness of the neck, retraction of the head, abdominal rigidity, and incontinence of urine and faeces occur in some cases. Vomiting is a variable symptom. Strabismus is present in some cases, but optic neuritis is rare. Facial paralysis has been recorded. Kernig's sign is present in some cases. In the first form the cerebrospinal fluid obtained by lumbar puncture is clear and contains only a few polymorphonuclear leucocytes. The typhoid bacillus cannot always be demonstrated microscopically, but cultivations always shew a pure culture. In the purulent typhoid meningitis the symptoms and onset do not differ from those found in the cases of serous meningitis. The main distinction would seem to be that in the serous meningitis a fatal result or recovery takes place before the cerebrospinal fluid becomes purulent. The cerebrospinal fluid in these cases is turbid, and the turbidity has been shewn to depend on the large number of bacteria present. Leucocytes are present, but not in large numbers.

Prognosis.—Although most cases of typhoid meningitis are fatal, cases in which the typhoid bacillus has been found in the cerebrospinal fluid have recovered.

Meningitis due to Bacillus enteritidis (Gaertner) is closely related to that due to the typhoid bacillus. Cases of infection of the intestine by *B. enteritidis* are by no means common, and the rarity of infection of the cerebral membranes must be great. Prof. Symmers and Dr. Wilson published a case of this nature in an infant four months old, who developed characteristic symptoms of meningitis. The cerebrospinal fluid obtained by lumbar puncture twice during life, and once after death, gave a Gram-negative actively motile bacillus, with all the cultural and fermentative reactions of the *Bacillus enteritidis.* The necropsy did not shew any evidence of intestinal or glandular infection, but the organism was cultivated from the spleen and glands. The base of the brain was covered with a fibrinous exudate which extended over the anterior surface of the pons.

Syphilitic Meningitis.—The syphilitic affections of the cerebral membranes are so intimately connected with syphilitic disease of the skull and of the nervous system that the following description will be limited to meningitis in babies and young children due to congenital syphilis. Even in the older children the symptoms so closely resemble those of juvenile general paralysis that it is doubtful if any useful distinction can be made between the two groups. In infants, however, the clinical picture is sufficiently distinct. In a series of 100 cases of fatal meningitis in children under twelve years of age, other than tuberculous, collected at the Children's Hospital, Great Ormond Street, 6 were described as syphilitic. It is doubtful if this represents the true proportion, for these cases run a prolonged and often chronic course, and hence might be discharged from the hospital as incurable, whereas the other forms of meningitis die rapidly. Five out of the six children were under two years of age.

Morbid Anatomy.—The condition of the membranes and brain is very characteristic. The pia-arachnoid over the vertex is opaque and greatly thickened. In some places it is closely adherent to the cortex, and any attempt to remove it tears off the surface of the cortex; in other parts it is fairly easily removed, leaving shrunken convolutions and wide sulci. The meningitis is not, as a rule, so marked over the base of the brain as over the vertex, and the membranes over the cerebellum are often normal. The cerebral convolutions are wasted and in parts resemble wash-leather (Fig. 17). On vertical section through the brain the ventricles are found to be dilated, in some cases to a very considerable degree. In parts the grey and white matter appears relatively normal; in other parts there is a space between the upper and lower layers of the cortex (Fig. 18). The cavity thus formed in the grey matter is filled by a milky fluid. This wash-leather condition may affect any part of the cerebral cortex, but not the cerebellum, which, however, may be sclerotic.

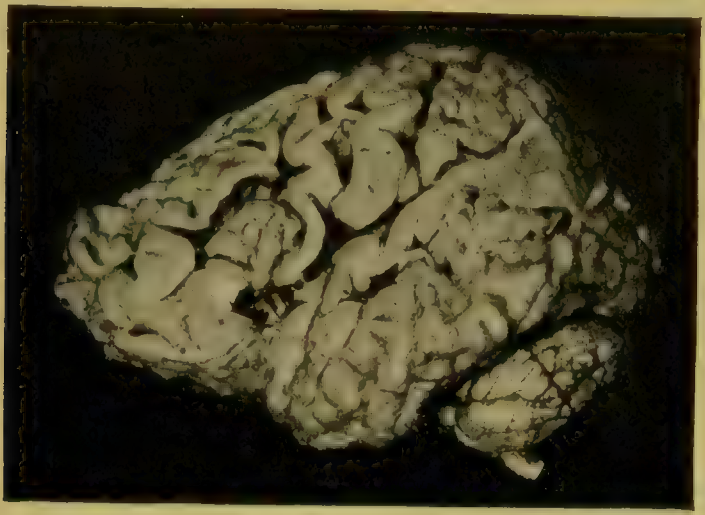

Fig. 17.—Syphilitic meningitis. Brain of child shewing thickening of the membranes and atrophy of the cortex. The membranes have been in part detached, but owing to the adhesions between the membrane and cortex, the latter has been in parts pulled away.

Microscopically the pia-arachnoid is thickened and infiltrated with small round cells which form a layer on the surface of the cortex. The superficial layers of the cortex are relatively little disturbed. Below this there is a layer of feebly staining loose tissue in which small round cells are comparatively abundant. The nerve-elements in this region are almost completely absent. Below this layer there is another layer of brain tissue which has a more or less normal appearance, but even here the nerve-elements are lacking, and many of the large nerve-cells which still remain have undergone calcareous change (Fig. 19). In those portions of the brain in which the disease is most advanced, it is this feebly staining loose tissue which forms the subcortical cyst. This area is always vascular and contains numerous new-formed vessels. The vessels are engorged with blood, and those with definite walls are surrounded by small cells. Both the smaller and larger arteries shew syphilitic endarteritis.

Ranke found the *Treponema pallidum* in nine out of fifteen cases examined by the Levaditi method. He found haemorrhage frequently, and in one case external hydrocephalus. Microscopically the pia was in all cases very cellular. The meshes were formed of fibroblasts; the meshes contained large round cells with a pale nucleus and often a vacuolated cell-body, and also plasma-cells. Lymphocytes were rare in the pia in spite of their presence in the cerebrospinal fluid. The cerebral cortex was infiltrated with plasma-cells, and changes are described in the glia-cells and in the nerve-cells. Weyl described the infiltration of the pia with lymphocytes and plasma-cells, and notes the presence of foci of encephalitis in the cortex.

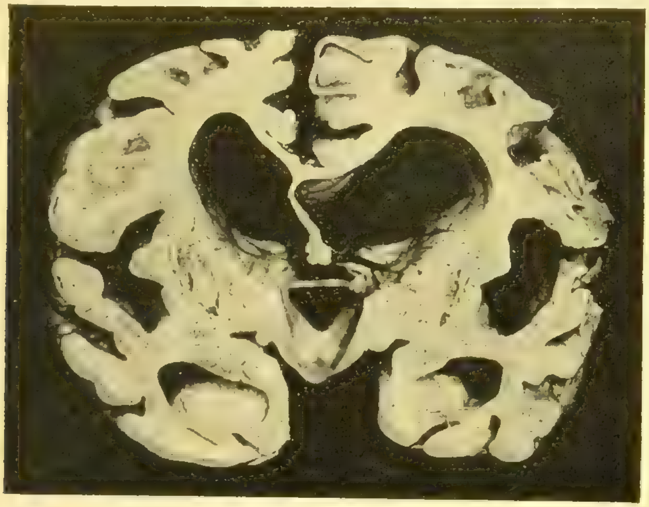

FIG. 18.—Syphilitic meningitis. Section through cerebral hemispheres, shewing (1) atrophy of cortex, (2) formation of subcortical space, and (3) dilatation of ventricles.

Sir T. Barlow described a case of syphilitic meningitis, arteritis, and choroiditis in a child the subject of congenital syphilis. The child had no meningeal symptom, except possibly laryngismus and some carpo-pedal contraction, but the examination of the fundus shewed a condition of choroiditis. The child died of marasmus, and at the necropsy the pia mater was extremely thickened. In many places, both on the vertex and on the under surface of the temporosphenoidal lobes, the small vessels of the cortex could be traced for some distance and were quite natural, then becoming white in colour, and resembling white thread. The changes in the choroid consisted in the presence of small isolated collections of corpuscles in the choroidal capillaries. Gummas on the cranial nerves are also described in another case in an infant aged fifteen months.

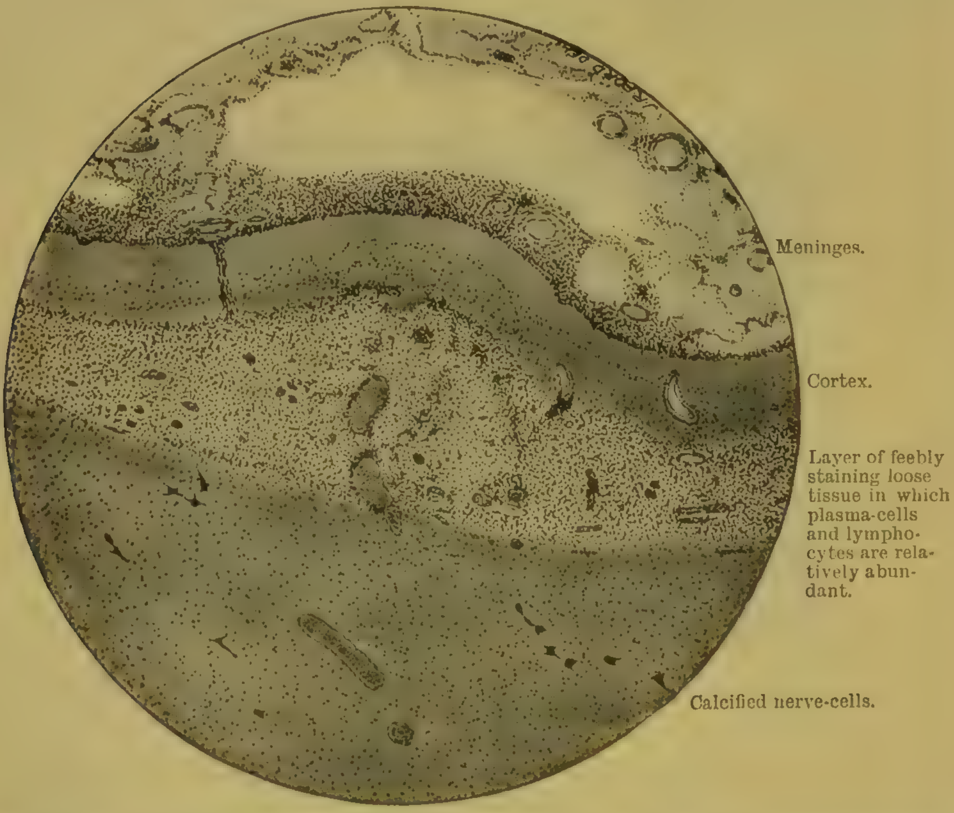
Meninges.
Cortex.
Layer of feebly staining loose tissue in which plasma-cells and lymphocytes are relatively abundant.
Calcified nerve-cells.

Fig. 19.—Section through the cerebral cortex of a case of syphilitic meningitis shewing the thickening and infiltration of the pia. The superficial layers of the cortex are relatively normal; between the lower layers, however, is interposed a layer of feebly-staining loose tissue, containing cells of various kinds, plasma-cells, lymphocytic, and glial cells. Numerous vessels are seen in this tissue. Below this, the deeper layer of the cortex again assumes a more normal aspect, but the large nerve-cells are few in number, and several of them have undergone calcareous change.

The Clinical Features of syphilitic meningitis are those of chronic meningitis, not infrequently associated with some hydrocephalus. The following case gives most of the leading features:—An infant aged eighteen months was said by the mother to have been healthy until three months previously, when the head began to enlarge. On questioning the mother it was found that the child had never been able to hold its head up or to sit up. The child took but little notice of its surroundings, and its vision was obviously defective. The head was large, and the anterior fontanelle open. The arms were rigid and the child made but few movements with them. The legs were also rigid, and there was but little power of movement. All the reflexes were increased, and the plantars gave an extensor response. There was some atrophy of the optic discs. By lumbar puncture a slightly turbid fluid was obtained which gave a high lymphocytic count = 92 per cent. The case ran a very chronic course, wasting becoming more and more pronounced, and the child died.

In some cases the onset of the disease is ushered in by fits which in the first instance may produce a right or left hemiplegia. Fits may then become general and recur with increasing frequency. The fundus of the eye may shew considerable degeneration of the choroid.

Although the symptoms may vary somewhat in individual cases, the course of the disease is very similar, viz. one of slow degeneration.

The diagnosis from the more acute forms of meningitis presents but little difficulty, though the symptoms in some cases bear a close resemblance to those of chronic cases of "posterior-basic" meningitis—the retraction of the head and blindness being the points of greatest similarity. A careful consideration of the history of the case and of the acute onset will aid one to a correct view of the case.

The presence of other syphilitic stigmata will aid the diagnosis, but their absence does not exclude the possibility of syphilitic meningitis. The examination of the cerebrospinal fluid affords some help, for a lymphocytosis is constant in syphilitic meningitis, but this may also be seen in a late case of the sporadic form of meningitis and in tuberculous meningitis.

Prognosis.—When syphilitic meningitis has been diagnosed the prognosis as to recovery and cure is hopeless, but the child may live on for many months, gradually becoming more and more feeble.

Treatment.—Mercurial inunction and large doses of iodide have been tried, but I have never seen any benefit ensue. It is possible that in early cases some arrest of the disease might take place.

SEROUS MENINGITIS.—The name serous meningitis is applied to a class of case in which there are symptoms of meningitis, and in which, on lumbar puncture, the cerebrospinal fluid flows out under increased pressure, and is clear, containing an excess of albumin and a few leucocytes, but no bacteria. It is a clinical rather than a pathological group, and will diminish in proportion as knowledge increases. Some forms of meningitis, such as the tuberculous, the syphilitic, and the late stages of "posterior-basic" meningitis, might be designated as serous, but it is far more accurate to retain their ordinary nomenclature.

The meningitis met with in mumps, in rheumatism, and other acute specific fevers, in some cases of otitis, and as the result of general poisons such as alcohol and lead, must for the present be regarded as serous meningitis, although at a future period they may be relegated to their own class.

Quincke's original description of serous meningitis was confirmed by the observations of Boenninghaus and Hansemann, who collected a series of cases, and more recently Riebold has published further cases. Cases have also been published by Dana of acute serous meningitis occurring in chronic alcoholism.

Morbid Anatomy and Pathology.—Riebold describes a round-celled infiltration and exudation of the pia mater and ecchymoses, which may thus account for blood in the cerebrospinal fluid.

According to Riebold meningitis may be due to measles, enteric fever, pneumonia, influenza, and is especially frequent in otitis media. Rheumatic infection, toxic influences, constipation, and menstruation also play a part in serous meningitis.

The Symptoms of acute serous meningitis do not differ from those of other forms of meningitis. The onset may be sudden and attended with convulsions; the symptoms may pass off in a few days or persist for weeks. Increasing drowsiness, fever, rigidity of the neck, headache, vomiting, convulsions, optic neuritis, and strabismus may all be present. The pulse may be slow and respiration irregular. The fluid obtained by lumbar puncture is clear, and contains albumin and a few leucocytes, but no bacteria. It comes out under high pressure, from 100-300 mm. of water.

Symptoms either local or general often occur in association with otitis either with or without operation. The local symptoms may be paralysis of one or more of the ocular muscles, or there may be the more general symptoms of headache, vomiting, and optic neuritis. Recovery often takes place in serous meningitis. The following case may, I think, be regarded as one of serous meningitis in a child, although finally a streptococcic infection supervened:—

A child, J. P., aged five months, was taken ill suddenly on 20th July. He was fretful and feverish. On 25th July a rash was noticed over the abdomen, and the throat seemed sore. The fever continued, and on the following day the neck was noticed to be stiff, the pulse rapid, and the respiration slow. The legs were rigid, Kernig's sign was present, and there was some twitching of the hands. Nothing abnormal was noted in the eyes. Some greyish exudation was present on the uvula and was shewn to be due to streptococcic infection. The child vomited frequently. Lumbar puncture was performed twice during life and once after death; the fluid was albuminous, shewed a slight polymorphonuclear leucocytosis, but did not contain any organisms. The child became comatose, and died twenty days after the onset of the disease. At the necropsy there was no meningitis of the brain or cord; there was a general streptococcic infection, and streptococci were recovered from the cerebrospinal fluid, although on the previous occasions the fluid had been sterile. On microscopical examination the brain and membranes appeared perfectly normal.

The Diagnosis of serous meningitis depends upon the examination of the cerebrospinal fluid. A clear fluid which comes out under pressure and contains albumin, a few leucocytes, but no bacteria, must be regarded as characteristic of serous meningitis.

The Prognosis in serous meningitis is relatively good; the majority of reported cases recovered.

Treatment.—The symptoms are often rapidly relieved by the removal of some of the cerebrospinal fluid by lumbar puncture.

MENINGISM.—This name was first applied by E. Dupré in 1894 to a group of symptoms indicating meningitis in cases which either recovered, or, if they proved fatal, did not present the morbid changes of meningitis. Although unendowed with a name this condition has been long recognised as occurring, especially in children, during the specific fevers or almost any febrile condition. Before the time when lumbar puncture was frequently performed it would be difficult to say that the cases were not meningitis, and probably many of the earlier recorded cases of meningitis with recovery belonged to this class.

Morbid Anatomy.—The condition may be said not to have any morbid anatomy, as the brain, spinal cord, and their membranes appear perfectly normal. The nature of the toxin responsible for the symptoms is unknown; but since the symptoms commonly arise in association with some acute febrile disease, it is probable that the toxin belongs to the class of poisons present in these fevers.

The Clinical Features are those of meningitis, namely, headache, vomiting, twitching, and convulsions, retraction of the head, squint, and rigidity of the trunk and limbs. The temperature may be raised and there is marked restlessness and irritability. The symptoms may come on rapidly, and after lasting a few days may clear up rapidly, leaving the child in perfect health. All the concomitant symptoms of meningitis—slow pulse, irregular respiration, tache—may be present. The distinctive feature lies in the result of the lumbar puncture. The fluid obtained by lumbar puncture may flow out under pressure, but is perfectly normal, and free from all cells and organisms.

Diagnosis of meningism from meningitis depends upon the examination of the cerebrospinal fluid. Dr. Tylecote gives the following points of distinction:—(1) In the acute infections meningism is often early in its occurrence and of short duration; (2) Kernig's sign is usually absent in meningism; (3) fever is more often absent in meningism than in meningitis; (4) opisthotonos is in favour of meningism; (5) lumbar puncture is more often beneficial in meningism than in meningitis; (6) frequent recurrence of the symptoms following lumbar puncture favours an organic lesion; (7) marked implication of cranial nerves favours meningitis; (8) the onset of the symptoms in question for the first time during the defervescent or convalescent stage of an acute infection is much against meningism; (9) the onset of meningism is acute, whereas in meningitis the symptoms often come on gradually; (10) the symptoms once developed usually remain of the same intensity till they begin to decline—there is not the ebb and flow, or the gradual increase in intensity often seen in meningitis; (11) hyperaesthesia and *tache cérébrale* are more frequent in meningitis; (12) absence of marked progressive wasting during the period when the symptoms are present favours meningism; (13) in fevers the absence of a disposing cause to

MEDICAL SCHOOL.

meningeal infection, such as injury or otitis, or of a family history of tuberculosis, or of its presence in the patient, favours meningism; (14) slowing of the pulse and irregular respiration are common in meningitis, rare in meningism.

No one of these points can be considered of great value in making the diagnosis between meningism and meningitis, although taken together they may aid diagnosis. The greatest difficulty arises in the diagnosis between serous meningitis and meningism, and without an examination of the cerebrospinal fluid this decision is impossible. It is not improbable that many cases described as meningism really belong to the class serous meningitis.

Prognosis in meningism is usually good; recovery may take place rapidly and completely.

Treatment.—With or without treatment these cases commonly recover. Rapid improvement has been recorded after lumbar puncture, which appears to be the most rational method of treatment.

FREDERICK E. BATTEN.

REFERENCES

Meningococcic Meningitis: *Clinical.*—1. BARKER. *Proc. Roy. Soc. Med.*, 1908, i. (Surg. Sect.), 143.—2. BAUMANN. *Brit. Journ. Child. Dis.*, 1905, ii. 49.—3. BOX. *Trans. Clin. Soc.*, London, 1905, xxxviii. 30.—4. CARR, J. W. *Med.-Chir. Trans.*, London, 1897, lxxx. 303.—4*a*. ELSER and HUNTOON. *Journ. Med. Res.*, 1909, xx. 377 (References).—5. FORBES. *Quart. Journ. Med.*, Oxford, 1908, i. 109.—6. FOWLER. *Rev. Neurol. and Psychiat.*, Edin., 1907, v. 249.—7. *Idem. Ibid.*, 1909, vii. 8.—8. GEE and BARLOW. *St. Barth. Hosp. Rep.*, London, 1878, xiv. 23.—9. LANGMEAD. *Practitioner*, London, 1907, lxxviii. 485.—10. LEES and BARLOW. This *System*, 1st edit., 1899, vii. 492.—11. OSLER. *Brit. Med. Journ.*, 1899, i. 1517.—12. POYNTON. *Clin. Journ.*, London, 1899, xiv. 594.—13. POYNTON and JEFFREYS. *Proc. Roy. Soc. Med.*, 1909, ii. (Child. Sect.), 1.—14. ROBB. *Brit. Med. Journ.*, 1908, i. 382.—15. WATTS. *Lancet*, London, 1908, ii. 524. *Pathological and Bacteriological*,—16. ALLEN. *Vaccine Therapy*, 1908, p. 210.—17. ARKWRIGHT. *Journ. Hyg.*, Cambridge, 1907, vii. 193; 1909, ix. 104.—18. DAVIS. *Journ. Infect. Dis.*, 1905, ii. 602.—19. GORDON. *Rep. Loc. Gov. Board*, 1907.—20. HOHN. *Klin. Jahrb.*, Berlin, 1908. xx. 357.—21. HOUSTON and RANKIN. *Lancet*, London, 1907, i. 1213.—22. *Idem. Brit. Med. Journ.*, 1907, ii. 1414.—23. LOOFT. *Nord. med. Ark.*, Stockholm, 1901, ii. 4.—24. M'DONALD, S. *Journ. Path. and Bacteriol.*, Cambridge, 1908, xii. 442.—25. *Idem. Rev. Neurol. and Psychiat.*, Edin., 1907, v. 592.—26. SHENNAN and RITCHIE. *Journ. Path. and Bacteriol.*, Cambridge, 1908, xii. 456.—27. SOUTHARD and STRATTON *Journ. Amer. Med. Assoc.*, Chicago, 1906, xlvii. 1271.—28. TAYLOR. *Lancet*, London, 1907, ii. 16.—29. WEICHSELBAUM. *Fortschr. der Med.*, Berlin, 1887, v. 573.—30. *Idem. Centralbl. f. Bakt. u. Parasitenk.*, 1903, xxxiii. 510.—31. WILSON. *Lancet*, London, 1908, i. 1686. **Pneumococcic Meningitis**: 31*a*. SOUTHARD and KEENE. *Journ. Amer. Med. Assoc.*, Chicago, 1905, xlvi. 13.—31*b*. SPERONI. *Arb. a. d. path. Inst. zu Berlin*, 1906, 160. **Influenzal Meningitis**: 32. BURY. *Brit. Med. Journ.*, 1900, ii. 877.—32*a*. COHEN. *Ann. de l'Inst. Pasteur*, Paris, 1909, xxiii. 273.—33. DOUGLAS. *Lancet*, London, 1907, i. 87.—34. DUDGEON and ADAMS. *Ibid.*, 1907, ii. 684.—35. FRÄNKEL. *Ztschr. f. Hyg.*, 1898, xxvii. 315.—36. GHON. *Wien. klin. Wchnschr.*, 1902, xv. 700.—37. NAUWERCK. *Deutsche med. Wchnschr.*, 1895, xxi. 393.—38. PFÜHL und WALTHER. *Ibid.*, 1896, xxii. 82.—39. PFÜHL. *Berlin. klin. Wchnschr*, 1892, xxix. 979.—40. SAUNDBY. *Brit. Med. Journ.*, 1908, i. 1341.—41. SIMON. *Rev. mens. d. mal. de l'enf.*, Paris, 1905, xxiii. 271.—42. THOMESCO et GRACOSKI. *Rev. neurol.*, Paris. 1905, xiii. 44. **Staphylococcic and Streptococcic Meningitis**: 43. SOUTHARD and STRATTON. *Journ. Amer. Med. Assoc.*, Chicago, 1906, xlvii. 1271.—44. WILSON. *Lancet*, London, 1907, ii. 1816. **Syphilitic Meningitis**: 45. BARLOW, T. *Trans. Path. Soc.*, 1877, xxviii. 287.—46. BARLOW and

Berry. *Dictionary of Psychology* (H. Tuke), 1892.—47. Ranke. *Neurol. Centralbl.*, Leipz., 1908, xxvii. 1082. **Typhoid Meningitis**: 48. Cole. *Johns Hopkins Hosp. Rep.*, Balt., 1904, xii. 379.—49. Lavenson. *Univ. Penna. Med. Bull.*, Phila., 1909, xxi. 55. — 50. Henry and Rosenberger. *Amer. Journ. Med. Sc.*, Phila., 1908, cxxxv. 240.—51. Hoffmann. *Deutsche med. Wchnschr.*, 1900, xxvi. 448.—52. Kamen. *Centralbl. f. Bakteriol.*, 1897, xxi. 440.—53. MacCallum. *Johns Hopkins Hosp. Rep.*, Balt., 1904, xii. 411.—54. Nieter. *München. med. Wchnschr.*, 1908, lv. 1009. —55. Rocaz and Carles. *Rev. Neurol. and Psychiat.*, Edin., 1908, vi. 557.—56. Silberberg. *Berlin. klin. Wchnschr.*, 1908, xlv. 1354. — 57. Southard and Richards. *Journ. Med. Research*, 1908, xix. 513. — 58. Symmers and Wilson. *Journ. Path. and Bacteriol.*, Cambridge, 1909, xiii. 251. — 59. Tictine. *Arch. de méd. expér. et d'anat. path.*, Paris, 1894, vi. 3.—60. Wentworth. *Arch. Pediat.*, 1899, xvi. 824. **Gonococcic Meningitis**: 61. Fürbringer. *Deutsche med. Wchnschr.*, 1896, xxii. 424.—62. Henderson and Ritchie. *Rev. Neurol. and Psychiat.*, Edin., 1909, vii. 75.—63. de Jong. *Centralbl. f. Bakt.* (*Orig.*), 1908, xlv. 501.—64. Schultze. *Spec. Path. u. Therap.*, Nothnagel, Bd. ix. Th. iii. Abt. 1, p. 65; *Krankheit. d. Hirnhaut*, 1901. **Rheumatic Meningitis**: 65. Poynton and Paine. *Lancet*, London, 1905, ii. 1760. **Anthrax Meningitis**: 66. Schultze. *Spec. Path. u. Therap.*, Nothnagel, *Die Krank. die Hirnhaut*, 1901, Bd. ix. S. 66.—67. Symmers and Wilson. *Journ. Path. and Bacteriol.*, 1909, xiii. 252. **Meningitis in Mumps**: 68. Roger et Margarot. *Rev. de méd.*, Paris, 1909, xxix. 826. 68*a*. Schottmüller. *Spec. Path. u. Therap.*, Nothnagel, 1904, iii. 62. **Streptothrix Infection of Meninges**: 69. Beevor and Buzzard. *Trans. Path. Soc.*, London, 1903, liv. 320.—70. Bollinger. *München. med. Wchnschr.*, 1887, xxxiv. 789.—71. Chiari. *Ztschr. f. Heilk.*, 1900, xxi. 351.—72. Delore. *Gaz. hebdom.*, 1896, i. 496.—73. Eppinger. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1890, ix. 287.—74. Howard. *Trans. Assoc. Amer. Physic.*, 1903, xviii. 427.—75. Keller. *Brit. Med. Journ.*, 1890, i. 709. —76. M‘Donald. *Scot. Med. and Surg. Journ.*, 1904, xiv. 305.—77. Nikitin. *Deutsche med. Wchnschr.*, 1900, xxvi. 612.—78. Wynn. *Brit. Med. Journ.*, 1908, i. 554. Further references will be found in Flatau, Jacobsohn, and Minor's *Handbuch der pathologischen Anatomie des Nervensystems*, Bd. i. S. 387. **Serous Meningitis**: 79. Beck. *Neurol. Centralbl.*, Leipz., 1907, xxvi. 1170.—80. Blumenthal. *Arch. f. Kinderh.*, 1904, xxxviii. 18. — 81. Dana. *Med. Rec.*, N.Y., 1897, lii. 801. — 82. Hansemann. *Verhandl. der XV. Kong. für innere Medicin.* — 83. Jackson. *Journ. Amer. Med. Assoc.*, Chicago, 1907, xlviii. 1078.—84. Oppenheim. *Monatsschr. f. Psychiat. u. Neurol.*, 1905, xviii. 135.—85. Riebold. *Deutsche med. Wchnschr.*, 1906, xxxii. 1859. **Meningism**: 86. Garrod, A. E. *Lancet*, London, 1909, i. 835. —87. Jackson. *Journ. Amer. Med. Assoc.*, Chicago, 1907, xlviii. 1078.—88. Tylecote. *Med. Chron.*, Manchester, 1907, xlvi. 157.

F. E. B.

TUBERCULOUS MENINGITIS

By Sir Thomas Barlow, Bt., K.C.V.O., M.D., F.R.S.

Definition.—Tuberculous meningitis is an acute disease depending on the invasion of the cerebral pia mater by the tubercle bacillus. The results of this invasion are the following morbid changes (which vary much in relative proportion):—the formation of miliary granulations along the sheaths of the vessels of the pia mater, the deposition of inflammatory lymph in the subarachnoid space, softening of the brain, acute hydrocephalus, fluid exudation into the spinal theca, and the formation of miliary granulations in the spinal membranes. These lesions of the nervous system are likewise accompanied by tuberculous deposit in one or more of the other organs of the body; some of this deposit may be contemporaneous with the lesions of the nervous system, but some of it is antecedent thereto.

History.—Our knowledge of the different anatomical characters of the disease, as set forth in the above definition, has been slowly won by successive workers. Robert Whytt (1768) in his observations on the "Dropsy in the Brain," although he had not recognised the specific nature of the affection, gave an excellent clinical picture of tuberculous meningitis; he insisted on its prodromes, and divided the illness into the three stages which have been accepted ever since. He first pointed out the important change in the pulse from rapidity and regularity in the first stage to slowness and irregularity in the second; and that in the third stage the pulse rose to "feverish quickness and once more became regular." The anatomical feature which arrested his chief attention was the acute hydrocephalus, and by this title tuberculous meningitis was long known. In 1825 Senn made the important addition that the primary inflammation is at the base of the brain, and that the pia mater is the membrane invaded. The tuberculous nature of the disease was discovered by Papavoine in 1830. He described the tubercles as "plaques" and granulations, and indicated their distribution in the meninges, and their identity and association with tubercles in other parts of the body. Papavoine also observed that granulations might exist in the pia mater without giving rise to obvious inflammatory reaction. Barthez and Rilliet advanced the knowledge of the disease enormously, especially on the tuberculous side. They asserted that the prodromes are the expression of the tuberculous diathesis; they regarded the existence of tubercle in organs other than the brain as the most essential antecedent condition; they even went so far as to claim as tuberculous meningitis cases of fatal brain affection in children in whom no tubercle was detectable in the brain, but in whom tuberculosis existed elsewhere; and in the typical manifestation of the disease they insisted on the multiple pathology which has been indicated in the above definition.

By degrees the partial convulsions of the second stage of the disease were referred to cortical disturbance consecutive to the meningeal inflammation. Also the distinction was drawn, by Rendu and others, between the temporary paralyses, the pathology of which is not explained, and the permanent paralyses of which many examples have been recorded, and which appear to depend on more or less circumscribed softening.

It was also maintained by Hayem that, in addition to the softening from necrobiosis after vascular obstruction, there is a true diffuse inflammatory encephalitis.

There has been much discussion on the nature of the hydrocephalus and the part played by it in the production of the pupillary changes, the paralysis, and the coma. Some, at least, of the pressure symptoms, which originally were referred exclusively to the ventricular effusion, are now attributed to a general encephalitis consecutive to the meningeal affection. Furthermore, the softening of the ependyma and of the walls of the ventricles is now considered to be largely inflammatory, and not a mere result of imbibition.

The final step, arising out of Koch's discovery, was that the disease is in all cases caused by the invasion of the tubercle bacillus; either by local infection through lymphatic paths, of which the subarachnoid space may be regarded as one, or, more commonly, through the blood-stream in embolic fashion along the distribution of the cerebral arteries.

Etiology.—*Age.*—Tuberculous meningitis is a disease of childhood rather than of adult life. It may occur, however, at any age, though with rapidly lessening incidence as years advance. Children at the period of the first dentition seem to be particularly liable to it. Of 100 cases, collected and analysed by Prof. Still, occurring in children under twelve years of age, in which the disease was verified by necropsy, 48 were under two years old: 83 of the 100 cases occurred during the first five years. During the first twelve months of life non-tuberculous forms of meningitis are more frequent than the tuberculous, but in the accompanying table it will be seen that 10 cases of the latter disease were recorded. The youngest patient was three months old; but some cases have been reported even in younger patients than this.

Sex.—It is generally held that after puberty many more females are attacked by tubercle than males; but, so far as tuberculous meningitis in adults is concerned, the careful statistics of Seitz indicate that more men are attacked than women.

Incidence according to Ages in 100 Cases under 12 Years of Age. (Still.)

Under 6 Months.	6 Months–1 Year.	1–2 Years.	2–3 Years.	3–4 Years.	4–5 Years.	5–6 Years.	6–7 Years.	7–8 Years.	8–9 Years.	9–10 Years.	10–12 Years.
3	10	35	13	13	9	5	4	4	2	1	1

Incidence according to Age in 126 Cases between 16 and 60 Years of Age. (Seitz.)

16–20 Years.	21–25 Years.	26–30 Years.	31–35 Years.	36–40 Years.	41–45 Years.	46–50 Years.	51–55 Years.	56–60 Years.
28	29	23	14	13	11	4	3	1

Tuberculous meningitis has been observed repeatedly in two or more children of the same family, and likewise in families in which other members have suffered from divers manifestations of tuberculosis. But the absence of a tuberculous family history is a fatally delusive argument against the recognition of early symptoms of tuberculous meningitis. In a large number of cases a single child out of a large family may die from tuberculous meningitis, and no other member of the family shew any indication of tuberculosis whatever. A family history of tuberculous disease was obtained by Prof. Still in 47 out of 100 cases (that is, nearly 50 per cent); but this proportion has no special significance, for almost exactly the same proportion can be obtained in the family history of persons not suffering from any tuberculous disease.

Insanity and hypochondriasis have been recorded in the family history of some cases of tuberculous meningitis, especially of the adult cases; and it is well known that a large number of insane patients die of pulmonary tuberculosis.

There is no build of body peculiar to tuberculous meningitis. The disease may attack either an adult or a child who is the subject of pulmonary tuberculosis and presents therewith any degree of emaciation. On the other hand, children apparently well nourished, with a fair amount of subcutaneous fat, are frequently attacked. Fair bodily nutrition is quite consistent with latent tuberculosis.

The common opinion that a large head and especially a prominent forehead make a child prone to tuberculous meningitis is quite absurd. This was pointed out by Whytt long ago. The reason for the popular belief is probably that the large forehead indicates rickets, and that a rickety child is more liable to convulsions than a non-rickety one.

For a full account of the *paths of infection* in tuberculosis the reader should consult Dr. Bulloch's article in Vol. V. p. 299. Putting aside (*a*) the cases of actual inoculation of the skin; (*b*) the cases of tuberculosis introduced by the genito-urinary tract; (*c*) the exceedingly rare and almost negligible cases of congenital tuberculosis, in the human subject the common modes of introduction of this virus may be divided into (1) inhalation through the respiratory tract; and (2) deglutition and ingestion through the alimentary canal. In adults inhalation is still generally accepted as the principal mode of introduction of the virus, but von Behring contended that even in adults an early ingestion-tuberculosis may be latent for years, and ultimately by dissemination through the lymphatics and blood-vessels give rise to pulmonary tuberculosis. In children anatomical and clinical investigation seems to shew that the introduction by inhalation is also the more common mode; but that deglutition and ingestion are probably more important in this respect than in adults for a certain indeterminate number of cases of tuberculosis. Calmette and other followers of von Behring go farther and maintain that the common portal of invasion in children is the alimentary canal, and that from the mesenteric glands the transmission of tubercle bacilli to the bronchial glands by the lymphatics and blood-vessels is common.

This is really a matter of inference and not of actual knowledge so far as the human subject is concerned. The experiments of the second Tuberculosis Commission (*vide* Interim Reports, 1907-1908) performed on bovine and other animals shew that tuberculous virus, both bovine and human, may by ingestion give rise to mesenteric tuberculosis and subsequent dissemination without any necessary primary tuberculous lesion of the intestinal mucosa. By analogy it seems probable that a similar invasion may occur in young children; and as tuberculous milk has been proved an efficient vehicle in animals, it is assumed with considerable probability that it is also an efficient vehicle in children. But for obvious reasons this cannot be investigated experimentally. Without disputing the probability of the hypothesis, it ought to be remembered that mesenteric and intestinal tuberculosis are much more common in children above twelve months old when milk is not the dominant diet than under twelve months old when milk in one form or other is the dominant diet. In my experience the most strongly suggestive instances of probable infection by tuberculous cow's milk have not been those in which mesenteric tuberculosis was proved, but cases of severe cervical gland tuberculosis in infants from four to six months old in which surgical operation and bacteriological examination have proved the existence of tubercle bacilli alone or accompanied by some other infective organisms. I believe it is aggravated cases of this type that afford the most promising field for inquiry respecting tuberculous milk, but they are not common. Furthermore, it is to be remembered that tubercle bacilli are widely disseminated, especially in towns, and that infants have many opportunities for the deglutition as well as the inhalation of dust more or less associated with tuberculous sputum. According to Emmett Holt, American experience points strongly to family contagion in the large majority of cases of tuberculous meningitis. There are well-authenticated cases of tuberculous meningitis in breast-fed infants who have never tasted cow's milk.

The causation of tuberculous meningitis is, strictly speaking, the causation of general or local tuberculosis plus the special invasion. It is rarely, if ever, primary: almost invariably some tuberculous focus of earlier date than the meningitis can be found. The focus may be a localised caseous process in any part of the body, as, for example, in the lungs, glands, bones, joints, skin, or generative organs. It is a perfectly legitimate criticism of the recorded cases of tuberculous meningitis in which no other tuberculous focus was found in the body, that the examination was not sufficiently exhaustive. In many cases tuberculosis of glands following simple enlargement from some peripheral irritation—such as catarrh of mucous membranes, especially of the throat, skin irritation, or carious teeth—forms the starting-point of a meningeal tuberculosis.

In other cases the disease forms part of a generalised acute miliary tuberculosis; but here also a primary focus generally exists; and in a large number of cases a breaking down of caseous glands seems to be the source of general infection. Prof. Sims Woodhead lays stress on cases which supervene on tuberculosis of the retroperitoneal glands and of the other glands in front of the vertebral column and in the posterior mediastinum.

A few cases arise by direct extension from a tuberculous mass in the cortex of the brain; and a few are associated with a caseous (tuberculous) process in the middle ear, or with some tuberculous caries of other cranial bones, or with Pott's disease, or with tuberculous dactylitis. In a case of Demme's tuberculous meningitis followed tuberculous ozaena. A curious relation, noted in two cases by Henoch, was the occurrence of tuberculous meningitis simultaneously with the abrupt subsidence of some enlarged cervical glands: with this may be compared a case, long under my own observation, in which the sudden retrocession of an extensive eruption of phlegmonous scrofulides coincided with the outbreak of fatal tuberculous meningitis. There are certain facts which seem to shew that occasionally surgical interference with tuberculous lesions, by the stirring up of latent deposits, may lead to discharge of tuberculous material into the general circulation, and so induce a general tuberculosis of which meningitis is a part. Two examples may be given: the onset of tuberculous meningitis was observed within a few days after forcible straightening of a hip-joint affected by subacute tuberculous disease; and, in the other case, within a few days after the removal of tuberculous glands from the neck. It is held by some surgeons that the scraping operations on tuberculous foci are attended with greater risk of entry of tuberculous material into the blood-stream than are complete excisions.

Tuberculous meningitis has been said to follow erythema nodosum: but from the reports it may fairly be asked if, in some of these at any rate, the initial lesions were not tuberculous periostitis or tuberculous disease of the skin (phlegmonous scrofulides).

Of remote causes, the acute specific fevers are the most important, especially measles, whooping-cough, and influenza: the former precedes tuberculous meningitis, as also other forms of tuberculosis, with remarkable frequency; histological examination of the bronchopneumonia of measles often reveals tubercle bacilli. It is probable that the exanthems not only lay the foundation for tuberculosis, but also reawaken any latent deposit; it is noteworthy that many of the exanthems are associated with glandular enlargement; and if the glandular enlargement left after an exanthem does not subside after a few weeks tuberculosis ought to be suspected. Measles, cervical glandular enlargement, tuberculous meningitis form a very frequent sequence.

Starvation and adverse circumstances probably play some part in the causation of this as of other forms of tuberculosis. Injury, especially a fall on the head, has often been assigned as a cause; it may act as a determinant.

Overpressure in education has also been alleged as a factor in the causation, but the evidence adduced is not satisfactory.

The immediate determining causes of the onset of tuberculous meningitis are very mysterious; it seems likely that any general lowering of nutrition, or some local irritation in a peripheral area which has lymphatic relation to previously caseating glands, by calling tuberculous foci into fresh activity, may determine an attack of the disease.

Morbid Anatomy.—Tuberculous meningitis is usually a leptomeningitis; but occasionally a few lentil-shaped white granulations are found on the dura mater, and larger tuberculous caseous masses sometimes implicate the dura secondarily. The arachnoid of the convexity feels slightly greasy, but does not present any other sign of inflammation. The superior longitudinal sinus usually contains a scanty pale clot; the veins over the surface of the hemispheres are not generally distended.

The convolutions are flattened, and, as the result of the ventricular effusion, the sulci are less marked than normal. It is in the subarachnoid space at the base and in the pia mater that the most marked changes take place. In the interpeduncular space, over the chiasma, over the anterior inferior portions of the temporo-sphenoidal lobes, and over the anterior portion of the superior vermiform process of the cerebellum, there is opacity due to inflammatory exudation. This is in some places green in colour, and of tough consistency. The Sylvian fissures are firmly glued. In these adhesions some greenish lymph may be found; but on separating the adhesions they are seen to consist for the most part of masses of grey or greyish-white tuberculous nodules, which have become partly confluent. Wherever the pia mater extends, such nodules may be traced; predominantly in the Sylvian fissures, but also along the fissures of Rolando, in diminishing numbers upwards to the vertex. There are often small deposits on the two surfaces of the superior longitudinal fissure; and likewise on the adjacent contiguous surfaces of the cerebrum and cerebellum. If the pia mater be traced inwards, granulations may be found in the choroid plexuses of the lateral ventricles; the velum interpositum is often packed with them, and so firmly matted that it is difficult to strip it from the subjacent structures. Such granulations vary in size from the finest "writer's sand" ("tuberculous dust") to confluent groups of an eighth of an inch in diameter; and, except at the base, they are seldom accompanied by inflammatory lymph. Dr. Higgs has recorded a case in which no miliary granulations were visible to the naked eye, and yet collections of tubercle bacilli were identified microscopically. This observation seems to justify the contention of Barthez and Rilliet that some cases of fatal brain affection in children are tuberculous meningitis, although tubercles cannot be seen in the membranes with the naked eye, whilst definite tuberculous lesions are found in other parts of the body (*vide* p. 202). Microscopically, the formation of the miliary tubercles begins in the small arteries and capillaries and spreads inwards into the lumen and outwards into the perivascular spaces in which tubercle bacilli can be detected. The exudation consists of polymorphonuclear leucocytes, epithelioid cells, and cells of various forms and sizes from small cells to large actively phagocytic cells. Definite giant-cells are rare. The mass thus formed readily undergoes caseation.

The brain substance occasionally shews small areas of capillary haemorrhage in the grey matter. Definite tracts of yellow softening are sometimes to be found also in the areas supplied by branches of the middle cerebral arteries; this softening is induced by tuberculous infiltration of the vessel-walls and by the thrombosis which completes their obstruction. Besides this localised cortical and subcortical vascular softening, there is a widespread alteration of brain substance. The cerebrum as a whole, in a well-marked case, is soft, and the corpus callosum and white matter above the lateral ventricles may be almost diffluent, even when the ependyma is intact, and there is little or no hydrocephalus; so that the softening cannot be the result of maceration. Microscopical investigation throws some light upon this condition: the deposition of miliary tubercles in the subarachnoid space, and along the ingrowing processes of pia mater, not only interferes with the vascular nutrition of the brain substance, but actually invades it, giving rise to extensive cellular infiltration, and causing degeneration of both axis-cylinders and ganglion-cells. This seems to justify the modern designation of the disease as a tuberculous *meningo-encephalitis.* Small circumscribed deposits of tubercle are not uncommonly also found embedded in the cortex; these are transition forms to the caseous tuberculous masses. Such large caseous masses are found in a fair proportion of cases of tuberculous meningitis. Thus, in his series of 100 examples Prof. Still found 15 in which such tumours were present. Around these masses there is not infrequently a local eruption of miliary tubercles in the pia mater, comparable with the miliary tubercles over a caseous mesenteric gland in the peritoneum. In typical tuberculous meningitis there is dilatation of the lateral ventricles, with a considerable excess of fluid which is rarely purulent in the true sense, but is turbid from the partial breaking down of the roof or sides of the ventricles. The third and fourth ventricles may present slight dilatation; but there is no closure of the cerebrospinal foramen, or adhesion of the membranes in the neighbourhood of the posterior arachnoid cistern: in this respect tuberculous meningitis presents a marked contrast with non-tuberculous "posterior-basic" meningitis, in which, if life be sufficiently prolonged, complete occlusion often takes place, and considerable hydrocephalus supervenes, but with clear fluid and firm walls.

There has been much difference of opinion as to the cause of the hydrocephalus in tuberculous meningitis; some regard it as a dropsy, others as the result of an inflammation. Dr. Bastian is inclined to lay stress on the occasional thrombosis of the veins of Galen, and on the general and extensive venous obstruction. It must be conceded that the absence in this disease of adhesions in the neighbourhood of the foramen of Majendie seems to negative the simple mechanical explanation of interference with the drainage system of the brain. Nevertheless, the fairly constant tuberculous infiltration and matting down of the velum interpositum may play some part in lessening the channel of the iter, and thus diminishing the outflow of the lateral ventricles. It seems possible that the anatomical conditions may be the joint result of inflammation and of dropsy; and that the special softening of the walls may be the outcome of the widespread cerebritis above described.

Circumscribed Tuberculous Meningitis.—It is important to note that there are cases of restricted tuberculous deposit in the membranes unaccompanied by the other (as I believe) more important lesions of the substance of the brain before described.

(i.) Cases of rapidly fatal miliary tuberculosis of the convexity. The duration of the illness may have been but a few days, and in the light of the necropsy it is difficult to see why, with such meagre changes, the case was fatal. The amount of miliary deposit on the convexity may be very small; there is little or no accompanying lymph or hydrocephalus; there is no important naked-eye alteration in the substance of the brain, and no basal change.

(ii.) Occasionally in necropsies on cases of generalised tuberculosis, circumscribed miliary deposit may be found in the pia mater, and small cortical and subcortical caseous tubercles without any lymph accompaniment, and likewise without subjacent softening or hydrocephalus. In some of these cases there has been no clinical indication whatever of brain affection. It is important to note, however, that the patients have generally been bedridden, and therefore exempted from many of the occupations and trials by which brain symptoms might have been evoked.

(iii.) In rare cases of old-standing tuberculosis, or of tuberculous meningitis (in its generalised form), there may likewise be found at the necropsy a circumscribed area on the convexity, presenting fibrous cicatricial material associated with some small caseous deposit and some scanty granulations — evidently the result of a healed tuberculosis. Finally, with respect to circumscribed areas of tuberculous deposit in the meninges, as compared with the generalised disease which we call tuberculous meningitis, it may be observed that the former is relatively more common in adults, and the latter in children.

Associated Lesions of Generalised Tuberculous Meningitis.—In a large number of cases the spinal cord is affected. Of the cases of tuberculous meningitis in adults, analysed by Seitz, 12 out of 20 shewed miliary tubercle of the spinal membranes also. In 22 cases of children who died of tuberculous meningitis, Prof. Still found tubercle 15 times, either in the theca or on the surface of the cord. The commonest condition observed was fine miliary tubercle, in scanty amount, on the inner surface of the theca, or on the cord in the lumbar region.

Tuberculosis, in various stages, coexists in many organs of the body. It appears in some situations to be contemporaneous with that in the membranes of the brain—as, for example, in the choroid, on the peritoneal surface of the liver, in the spleen, and even in the lungs and pleurae, where either the finest grey or opaque white recent granulations may be sometimes found. Tuberculosis of the parathyroids has been found in a few necropsies, and it has been suggested that this had a causal relation to the tetany-like spasm of the hands ("obstetric hand") which sometimes occurred during life. Winternitz has reviewed the scanty records concerned with this alleged association as well as the observations which have been made on the increase of calcium output in the urine and its diminution in the blood and the brain of animals suffering from tetany. In tuberculosis also the calcium output of the urine is increased, and there appears to be often a demineralisation of the tissues. The hypothesis is offered that given a deficiency of calcium in the circulating fluid an injury to the parathyroids by increasing the disturbance of calcium metabolism might induce tetany. The objection to any true causal relation between tuberculous change in the parathyroids and tetany-like spasm in tuberculous meningitis is the very transitory character of this spasm. It is one only of many forms of spasm which rarely continue more than a few hours, and are replaced either by paresis or by other forms of contraction. Tuberculosis of older date is to be found in some part of the body, especially caseous lymphatic glands in the neck, at the root of the lungs, at the bifurcation of the trachea, in the mesentery, and in front of the spine; and some of these older lesions are often softening, with evidence of renewed activity and dissemination of tubercle in their immediate neighbourhood, suggestive of invasion of the blood-current.

Purulent collections in conjunction with what has been called surgical tuberculosis—that is, psoas abscess from caries of the spine, hip-joint disease, caries of the temporal bone, or tuberculous salpingitis—may in certain cases be fairly regarded as foci of infection.

The amount of chronic tuberculous deposit may be exceedingly small, and a few examples have been recorded in which no tuberculous focus was found in the body; but it is fair to ask in such cases if the examination was sufficiently exhaustive.

A concomitant lesion sometimes found in the lungs of children dying from tuberculous meningitis is multiple haemorrhagic foci symmetrically distributed on the posterior surface of both lungs, larger than the ordinary asphyxial ecchymoses, sometimes even as big as an almond.

Symptoms.—The insidious *onset* is the more common of the two, and constitutes what is called the prodromal stage. In children this may last from two weeks to three months, and is ill-defined throughout. The child seems out of sorts, he lies about instead of playing with his toys, he sleeps badly, he grinds his teeth and may have night-terrors. His appetite fails, he loses flesh, and occasionally he vomits, especially at night and without apparent cause. At this stage there may be a very fetid smell of the breath, as if from gastric catarrh. The bowels are generally constipated. There is nothing uniform about the temperature, but erratic unexplained pyrexia is common. The dominant features are alternate languor and irritability, the latter being manifested when the child is disturbed. In children of the school age change of temper is sometimes observed: a child previously obedient and amiable becomes irritable or wilful. He sometimes complains of headache, often frowns, and occasionally flushes. In a child over five years of age mistakes in speech may be noticed: the speech may be slow and very deliberate, as of laboured production; a marked reluctance to answer questions may be observed, and sometimes even a taciturnity amounting to speechlessness for days and weeks; although when questions are asked, the child may answer them. In adults, speech alterations often occur. There may be true aphasia, or some delay in answering questions which suggests cerebral inertia. A boy, aged about eight years, in the prodromal stage of what proved to be tuberculous meningitis, was sent to a shop to buy something. He found when he got to the shop that he was unable to ask for what he wanted. A very young child does not generally make mistakes of speech, but is often speechless for a day or two at a time, takes no notice of mother or nurse, asks for nothing, drinks only when liquid is presented, and sometimes silently passes urine in its bed, contrary to its wont.

The patient often stares and frowns at the doctor, but presents no other oddity of manner. It is most important to remember that in adults the onset of tuberculous meningitis is sometimes indistinguishable from that of hysteria; and the suppression of such initial symptoms by douching and electrical applications may give rise to delusive hopes. Incontinence of urine and faeces, again, associated with strange behaviour, occurring in the prodromal stage in adults, may likewise be set down to hysteria; many cases of tuberculous meningitis in adults have at the onset been mistaken for early mental disease.

Delirium is rare in children, but not uncommon in cases of adults; and in some instances of early tuberculous meningitis even delirium tremens is simulated. The early headache is much more severe in adults than in children. Lethargy, like that in the early days of a severe exanthem such as typhus, has been recorded; in such cases there may be prolonged retention of urine. Children are more somnolent than adults. When the disease supervenes on some chronic tuberculous illness which has confined the patient to bed, the onset of tuberculous meningitis may be unsuspected, until a squint suddenly appears, or a complaint of double vision, or a slight facial or upper limb palsy.

Another mode of sudden onset in tuberculous meningitis in bedridden patients is capricious vomiting, apart from cough.

Finally it is to be noted that the supervention of tuberculous meningitis on severe or extensive tuberculous disease elsewhere is sometimes quite latent, especially in bedridden patients, and not discovered till after death.

In considering the prodromal manifestations generally it will be seen that although no one of these symptoms singly is pathognomonic, yet, in reviewing a case from beginning to end, their cumulative value is very great. The prodromes probably correspond with some dissemination of tubercle; and it is a matter of experience that during this vague period some slight chronic enlargement of glands in the neck or under the jaw, or some impaired resonance near the manubrium, although unattended with active symptoms, may give a cue to the imminent disease. The invasion of tuberculous meningitis Dr. Gee defines as the period at which such new symptoms occur as enable us to pass from the uncertainties of the prodromal stage, and to declare positively that, from this date at all events, the child has sickened with hydrocephalus. But it is fair to say that these new symptoms are often no more than exaggerations of one or more of the prodromes—such as vomiting, headache, or lethargy associated with constipation. Of definite new manifestations the most striking invasion-symptom, though not the most frequent, is convulsion. Such invasion-convulsion is in most cases general or bilateral; it is either a generalised rigidity with pallor and loss of consciousness, or it is divided into tonic and clonic stages affecting both sides of the body alike. From this period the disease has been divided into three stages, namely (*a*) the stage of irritation—referred to the meninges and cortex; (*b*) the stage of pressure—referred to the ventricular effusion; (*c*) the stage of relaxation and paralysis—referred to extension to the medulla oblongata (Traube).

But the symptoms of the first stage overlap those of the second, and the most satisfactory method is to consider each symptom separately.

Facies.—In the earliest stage the face shews nothing more than the frowning; but after the invasion there is often at times a vacant stare, with pupils dilated, and the eyes fixed on some distant object—"the far-off look," as it has been called. The flush of the cheeks, which in the prodromal stage was occasional, becomes more persistent, and is sometimes accompanied by a scarlet suffusion over the rest of the body, and a blotchy erythema of the palms. The tache cérébrale, or red flush elicited by stroking the skin, though by no means peculiar to this disease, is very frequently obtainable. In some cases a white streak appears before the flush. In the late stage any part of the body flushes if subjected to pressure.

During the prodromal and early stage of the disease the *skin* is dry and harsh; but in the last forty-eight hours it is often bathed with sweat.

Decubitus is very characteristic: quite early in the disease the child lies persistently on one side; if turned on its back or face it turns back on its side at once and lies with its knees flexed on the abdomen, and usually with the elbows flexed and drawn to the sides. It resents any interference; often, if disturbed, it will cry out peevishly, "Go away"; and if the bed-clothes be pulled back it will often clutch at them to draw them back (Stocker's sign).

In the later stage, when paralysis and coma have supervened, the child often lies on its back, low down in the bed, with the arms and legs extended, and the hands lying one over the other just above the pubes.

Photophobia is more common in adults than in children. The *hydrocephalic cry*—a sudden shrill and apparently causeless scream—is characteristic of intracranial disease, but not especially of tuberculous meningitis; the majority of cases run their course without its occurrence. Usually as long as the child is undisturbed it lies quite quiet in the position described; but occasionally there is great restlessness, the child rolling itself from side to side with such violence as to require a padded bed. In rare cases there is delirium, which may be quiet and muttering or wild and noisy. The occasional simulation of delirium tremens in adults has been mentioned already.

Headache is generally present in adults; but children often make no complaint of it, though the raising of the hands to the head, or the boring of the head into the pillow, may betray its presence: in some cases, however, the child cries out frequently, "My head, my head," and some of the sudden screams may be due to paroxysms of headache. Headache in adults is more commonly in the frontal region; but generally in children is not localised.

Pain is sometimes referred to other parts of the body, as to the abdomen, to the soft parts of the limbs, to the joints, and to the spine. Complaint of pain in the knees was so severe in the early stage of one case as to suggest rheumatism and the application of cotton-wool round the joints. In a girl, aged ten years, complaint of the spine and marked local hyperaesthesia, in the absence of any other symptoms except pyrexia, were set down to hysteria; but within a few hours coma supervened, and at the necropsy tuberculous meningitis was found.

Vomiting, although one of the most frequent invasion-symptoms, is singularly variable in the subsequent stages of the disease. In rare cases it is incessant for days together; more commonly it recurs once or twice in the twenty-four hours. It is of the cerebral type; the contents of the stomach seem to regurgitate without retching or effort. Often no exciting cause is apparent, but movement and disturbance are likely to induce it. It is remarkable how often persistent recumbency seems to favour its subsidence. Certainly it often occurs after taking food, and it is influenced to some degree by diet. Ophthalmoscopic examination sometimes excites it.

Constipation is a marked symptom, and usually persists throughout the illness. As a rule it is easily overcome by simple aperients or enemas. Occasionally it is so obstinate as to secure exclusive attention, and to mask the diagnosis. Thus, in two cases of this kind the combination of obstinate constipation, vomiting, and a slight visible peristalsis raised the suspicion of intestinal obstruction, and the proposal of laparotomy was for a short time entertained. But in both patients there was marked lethargy, although no other definite cerebral symptom had appeared.

Rarely there is diarrhoea, which may or may not be associated with tuberculous ulceration of the bowel. In either case the abdomen generally becomes flattened or carinate; the few exceptions being mostly cases of antecedent tuberculous peritonitis. Associated with this carination there is often marked flaccidity of the abdominal wall.

Even at this early stage there is often some inertia of micturition, leading to retention of urine; this can generally be overcome by getting the patient either to stand or sit on the side of the bed.

The *pulse* early shews irregularity, a symptom present with such frequency as to have considerable diagnostic value; the irregularity may affect rate, rhythm, or force. The rate per minute alters distinctly during the course of the disease; there may be a little quickening during the prodromal stage, then slowing takes place during the irritative stage, and with the slowing the artery gives a sensation to the touch as if tightly contracted. Marked pulsation of the carotids in the neck may be noticed at the same time; and if the fontanelle be open it is tense and its pulsation manifest. The slowing of the pulse is much less marked in young children than in children of five years old and upwards. Finally in the paralytic stage the pulse becomes softer, more and more rapid, and may reach 220 per minute or become uncountable; generally at this stage the irregularity is lost; and in the stage of relaxation a few hours before death the fontanelle often becomes flattened again.

Respiration shews a corresponding irregularity quite early; even sometimes in the prodromal stage an occasional deep inspiration is followed by a deep and long expiration—the so-called "sighing respiration." As the disease advances this becomes more frequent, and towards the end Cheyne-Stokes breathing is common. Finally the respiration becomes simply shallow and rapid.

A symptom often present, but indicative rather of meningeal or cortical mischief in general than of tuberculous meningitis, is *quasi-purposive movements* of the mouth; such as sucking, pursing or biting the lips, champing of the jaw, and grinding of the teeth. Quasi-purposive movements of both upper and lower limbs are by no means uncommon. The repetition of these movements, with slow alternations, may continue for a considerable time, even against resistance. Plucking at the lips, nipples, and genitals is often present quite early in the disease.

Slight *stiffness of the neck* and even slight *retraction of the head* may be present occasionally, but are very rarely pronounced or continuous. In the absence of permanent head-retraction, tuberculous meningitis usually shews a marked contrast to simple "posterior-basic" meningitis. There are, however, very rare cases of tuberculous meningitis, in which head-retraction is pronounced and prolonged, and in which after death an excessive amount of miliary deposit is found at the posterior arachnoid bridge.

The *knee-jerk* is extremely variable. It may be normal or excessive on one side and normal on the other, or difficult to obtain, or paradoxical. It often varies on successive days. Kernig's sign is most conveniently obtained in dorsal decubitus by flexing the thigh at right angles to the abdomen, it will then be found if the sign is present that the leg cannot be extended into the same straight line as the thigh. Morse of Boston has made an elaborate investigation as to its occurrence in infancy, and he finds that it is almost never present in very young children except in meningitis. It is never present in many cases, and in many others it is present only intermittently. It has no apparent connexion with the degree of intracranial pressure. It is more often present when the knee-jerk is increased than when it is diminished. Morse considers, in opposition to the more commonly accepted view, that it is of no value in diagnosis between the tuberculous and cerebrospinal forms.

Tremor of the head and limbs on movement, associated with rigidity of the neck, is almost always present when the disease is established; and even in an early stage it can generally be elicited if the observer place his hand behind the child's shoulder and raise him slightly from the bed. It is also well seen if the patient attempt to stand, which he does in an unstable fashion with his legs widely separated; his head, arms, and lips shewing meanwhile a coarse tremor.

The *convulsions* of tuberculous meningitis may be roughly divided into:—(*a*) The convulsion of invasion mentioned on p. 212, which may be general and solitary, and removed by a considerable interval from those which follow; (*b*) the convulsions of the middle period, which tend to be more or less limited; and (*c*) those of the later period, which are frequently generalised. Those of the middle period are the most important and distinctive, because they point to localised cortical disturbance. A varying tonic stage is often succeeded by clonic spasm, limited either to one side of the face and body, or sometimes to one limb. But, although the spasms may on one day be confined to a single limb, on successive days other limbs generally become affected. This is the rule in children, and it points to bilateral distribution of the lesions. More or less continuous tonic spasm affecting one wrist and the fingers, and simulating tetany, is not rare in the second stage. Such spasm may last for a day and be replaced by spasm in some other region, but so far as I have seen does not continue beyond a day. Occasionally in adults the spasm is tolerably constant in its limitation, and this is in harmony with the anatomical fact that in adults the meningeal deposit is often more circumscribed than in children.

Sooner or later some *rigidity* of the limbs appears; most commonly it persists after a convulsion, and it often varies on successive days. All four limbs may be rigidly extended, or an arm or a leg may be rigidly extended or flexed. Attempts to overcome the rigidity may elicit expressions of pain, and sometimes may set up rhythmical contractions of other limbs. Convulsions are sometimes followed by *paralysis* rather than by rigidity; and spastic contraction of the limbs of one side may coexist for varying periods with flaccidity from paralysis of the other side. In most cases there is some paralysis of cranial nerves, as might be expected in a disease chiefly affecting the base of the brain. Oculomotor and facial palsy are seen more frequently than palsy of any other cranial nerve. Squint may be one of the earliest symptoms of the disease; and the patient's first complaint, especially if an adult, may be that he sees double. These cranial nerve palsies, like those of the limbs, are often temporary.

Permanent *palsies*, though much less frequent, arise sometimes with, sometimes without, preceding convulsions; and of these hemiplegia, brachial monoplegia, facial and oculomotor paralysis are the most common.

The *alterations of speech* in the prodromal stage have been already mentioned (p. 211); but these by no means persist throughout the illness. For the most part they vary like the alternating convulsions and temporary paralyses. Delay in response, or actual speechlessness, is more common in children than aphasia properly so-called.

In the later stages there is great *difficulty in swallowing*; but this varies from day to day in a remarkable manner.

Other disorders of movement are seen at times. Mechanically associated movements may be present; for example, on forcibly flexing one leg on the abdomen the arm of the same side or the leg of the opposite side becomes flexed; and probably analogous to this is the adductor spasm in the opposite thigh produced sometimes in tuberculous meningitis when obtaining the knee-jerks, but it is not peculiar to this disease.

In the early stage there is excessive *hyperaesthesia*, though, on account of the general irritability of the patient, it is difficult to gauge. Nevertheless, in some cases there seem to be areas of increased intensity of the hyperaesthesia; as for example over the vertebral spines. In the later stage it is alleged that some *local anaesthesia* can be made out. It is difficult to prove; but at the end of the disease, when the patient is becoming comatose, the loss of conjunctival reflex and the easily abraded cornea would seem to favour the opinion that sensibility is lowered.

During the early stage the *superficial reflexes* are not altered; later Henoch specially notes the disappearance of the cremasteric reflex. Babinski's sign (extensor response of the great toe on tickling the sole) is sometimes obtained on one side, sometimes on both. It may be present one day and absent on the next.

Symptoms referable to the *eyes* may be considered in a group. As to the external muscles, varying squint and ptosis are very common during the irritative and pressure stages. If facial palsy occur, either temporary or permanent, the corresponding eye may remain half open for a time. Independent movements of the eyeballs are often seen, especially slow movements of one or both eyes from side to side, but rarely definite nystagmus. Jerky movements of the eyeballs, either with or without other clonic spasms, and constituting minor convulsions, are sometimes confounded with nystagmus.

The pupils, which are often slightly contracted in the prodromal and early irritative stages, gradually become dilated and shew a curious oscillation when exposed to a bright light. The pupil contracts, but immediately dilates again; and then alternately contracts and dilates. Archambault laid great stress on this oscillation of the pupil in tuberculous meningitis; but it is also found in other morbid brain states. In the last stage the reaction to light is lost. The dilatation becomes more marked and persistent as the ventricular effusion increases. In tuberculous meningitis of the convexity, in which case there is not usually any associated hydrocephalus, the dilatation of the pupils does not occur, or is much less marked than in the common form. There is also during the pressure stage some irregularity of the pupils. It has been observed that in some cases the pupil on the side on which the patient lies is more dilated than the opposite one; and in a case recorded by Sir Samuel Wilks, in which there was much hydrocephalus, the dilatation was reversed by reversing the position of the head.

Anaesthesia of the cornea and of the conjunctiva occurs during the last stage. There is much vascularity, and muco-pus collects on the conjunctiva: and on the lower margin of the cornea there is frequently a crescent of opacity followed quickly by superficial erosion.

Ophthalmoscopically the two changes which require consideration in this disease are optic neuritis and choroidal tubercle, to which, respectively, attention was first directed by Sir Clifford Allbutt and Cohnheim.

With regard to *optic neuritis*, its indications in the early stage of the disease are often ambiguous; but late in the disease it can generally be detected on both sides, though unequally. Even then it is rarely severe or comparable with that associated with cerebral tumour; but the increase of blurring of the edge and of curving of the vessels is conclusive to one who has observed the progressive change in a given case. In judging of the progress of the neuritis, Garlick lays great stress in these cases on the alterations which take place in the apparent lumen of the arteries and veins, and on their altered relation to one another.

Tubercles of the choroid can be observed occasionally in successive development at various parts of the fundus, beginning as minute round dots which become gradually opaque in their centres, and sometimes confluent at their margins. Tubercles of the choroid thus seen to develop during the course of a brain illness are so far in favour of that illness being tuberculous meningitis. I found that out of 16 consecutive cases of choroidal tubercle 13 shewed post-mortem evidence of tuberculous meningitis; whilst 3 presented tubercle in other organs, but no tubercle in the pia mater. Thus, apart from any other signs, tubercle of the choroid cannot be taken as indicating that there is necessarily a deposit also in the pia mater, but it bears witness probably to a widespread tuberculosis.

The course of the *temperature* is difficult to distinguish from that of generalised tuberculosis; but in general terms it may be stated that the advent of the meningeal affection in a case of marked tuberculosis is associated with a lowering of the previous temperature, and with some departure from the hectic type. This lowering of temperature is sometimes of diagnostic value in the first and second stages of the disease; but it is more noteworthy in children of five years and upwards, and in adults than in young children. During the later stage of tuberculous meningitis the temperature rises in the majority of cases. For a day or two before death the rise may reach 108° F.; especially when repeated convulsions have occurred. Less commonly the temperature remains low throughout; it may even become subnormal, especially as death approaches.

The *urine* is generally normal in appearance, though said to be deficient in amount. Not infrequently it contains a trace of albumin, and, rarely, a trace of sugar may be found in a late stage of the disease. Both urine and faeces may be passed under the patient from the time that the disease first declares itself.

The *blood* usually shews a leucocytosis of from 15,000 to 30,000; Cabot noted a leucocytosis in 32 out of 43 cases. As far as can be gathered from the available differential counts, there is not any relative increase of lymphocytes.

Lumbar Puncture.—The cerebrospinal fluid is clear or turbid, shews a lymphocytosis, and contains tubercle bacilli. In a series of 102 cases mononuclear cells predominated in all but 3, and tubercle bacilli were present in 100 or 98 per cent (Bernstein).

Tuberculin Tests.—Von Pirquet's and Calmette's tests give fairly trustworthy results in the early stages, but the reactions are usually negative in the later stages (Holt).

Consciousness is seldom completely lost until quite late: the earlier condition is rather one of drowsy irritability; the patient can be roused and is sufficiently conscious to resist attempts to feed him, but when left alone speedily relapses into his semi-conscious condition. Occasionally the condition is one of semi-consciousness to the end; the patient can be roused and may answer questions even an hour or two before death. Rarely he remains quite conscious and takes notice of his surroundings until within a few hours of death, when he rapidly becomes unconscious: such cases are usually those in which tuberculous meningitis has supervened on some previous chronic disease. There are a few examples in every man's experience of the patient's waking up from a coma, recognising his parents, taking food, and even playing with toys, only to relapse again into fatal unconsciousness.

Prognosis. — The morbid anatomy of this disease gives the only reasonable basis on which to discuss the prognosis. If the tuberculous deposition be limited to a part of the surface of the brain, there is a possibility of recovery; but if the disease become generalised, so that besides the invasion of the pia mater there is an extensive meningo-encephalitis with or without hydrocephalus, the chance of recovery is almost nil.

There are several cases on record in which there was a history of brain symptoms from which the child recovered, but after an interval of months or years succumbed, either to a fresh outbreak of tuberculosis in some other part of the body, or to an attack of generalised tuberculous meningitis, when after death the evidence of old cicatrised limited tuberculous deposit in the pia-arachnoid was found. An example of this group was under my observation for a considerable time and the sequence of events was as described. From a careful investigation of the recorded cases of alleged recovery Dr. A. Martin finds that 20 undoubted cases can be accepted since 1894. The criteria upon which Dr. Martin lays stress are the proof of tubercle bacilli obtained by lumbar puncture and the ultimate verification post-mortem of cicatricial changes in the pia mater with evidence of tuberculosis in other parts of the body. Dr. Martin also rightly insists on the value of remissions in the course of tuberculous meningitis as additional indications of the possibility of partial subsidence in the evolution of tubercle in the brain. It is also relevant here to draw attention to the clinical indications of the occasional recession of choroidal tubercles as a parallel to the obsolescence of tubercles on the pia mater.

The duration of the generalised disease has very wide limits. The difficulty of separating the prodromal stage from the onset of the pronounced disease has already been mentioned. The widest variation is observed between the so-called primary and secondary cases. In the primary cases (that is, in those in which tuberculous meningitis supervenes on a state of apparently fair general nutrition) Dr. Gee's estimate of twenty-three days, reckoning from the time that the disease becomes decided to the day of death, certainly covers a very large number; yet there are a goodly number of longer duration. I have notes of cases in which the disease was definite for thirty-seven days, for forty-two days, and for sixty-three days respectively.

In the secondary cases (that is, in those in which brain symptoms supervene upon some chronic form of tuberculous disease) the duration is short, and may be not longer than three days.

Vertex Cases.—Though it is amongst this group that we must look for the very rare examples of recovery from a circumscribed deposit, yet as a rule the duration of the cases is much shorter than that of the common basic generalised type. In these vertex cases the patients often pass rather suddenly into coma, and die within a week.

Diagnosis.—Tuberculous meningitis presents such wide differences in its early and late stages that the difficulties of diagnosis also vary according to the period at which an opinion is sought. The disease is much more difficult to detect in an early than in a late stage. Speaking generally, the difficulties at *the outset* in children are concerned with either (*a*) the distinction of the constitutional symptoms from those of some form of *gastro-intestinal catarrh*, or (*b*) from those of one of the *exanthems* or of *pneumonia*.

In both (*a*) and (*b*) there may be headache, vomiting, sleepiness, restlessness, or convulsion. A decision is often impossible for three or four days; but by that time the characteristic eruption of an exanthem or some pulmonary sign of pneumonia should have appeared. In general terms, however, it may be said that at this early period the temperature becomes suddenly and persistently higher, and the pulse and respiration more frequent in (*a*) and (*b*) than in the prodromal stage of tuberculous meningitis. The vomiting, though possibly more violent during one or two days in (*a*) or (*b*), does not recur day after day with so little distress as is the case in tuberculous meningitis.

Fulness and pulsation of the fontanelle in young children are warnings of incipient meningitis, though they are not conclusive. In the state of exhaustion with semi-coma following severe infantile diarrhoea, to which Marshall Hall gave the name of *hydrocephaloid disease*, the fontanelle is depressed, whilst the abdomen is commonly distended.

Enteric fever in the past was the condition most difficult to distinguish from tuberculosis in an early stage. Enteric fever can now be recognised comparatively early by the agglutination reaction, whilst lumbar puncture is a great assistance in the recognition of tuberculous meningitis. During the second stage of tuberculous meningitis the pressure of the cerebrospinal fluid is notably increased. The fluid may be clear or turbid, and characteristically shews a lymphocytosis, though in some reported cases polymorphonuclear cells have predominated. The presence of tubercle bacilli clinches the diagnosis. Turning now to the purely clinical differences it may be pointed out that, even if constipated, the typhoid patient has some tympanites as a rule, whereas in tuberculous meningitis the abdomen is retracted. And in tuberculous meningitis the temperature is much lower, and the irritability is greater than in enteric fever. It is interesting to recall Stocker's sign as a means of differentiating the two diseases: in enteric fever, if the bed-clothes be pulled down the patient takes no notice, or at most shews but a languid interest in what has been done; but in tuberculous meningitis the patient resents the interference, and immediately draws the clothes up again. Lateral decubitus is the rule in tuberculous meningitis, dorsal decubitus in typhoid; and the general attitude in the enteric disease is one of relaxation, whilst in tuberculous meningitis there is a proneness to the curled-up position. The facies of typhoid is generally one of relaxation, whilst frowning is common both in the early and middle stages of tuberculous meningitis. The "far-off" look with dilated pupils in tuberculous meningitis is very characteristic. Even within the prodromal stage of tuberculous meningitis, if the child be raised from the lying posture, there is often an indication of slight stiffness about the neck; but in enteric fever this does not appear. The pupils in early tuberculous meningitis are contracted; in enteric fever they are commonly dilated. In the second stage of tuberculous meningitis the pupils are generally dilated. Early ptosis or squint is always an important suggestion. An occasional sigh, and an intermission in a slow pulse, are most important suggestions, even in the prodromal stage of tuberculous meningitis; although suspirious breathing sometimes occurs in enteric fever, this is rare in the first week. Headache is sometimes very severe in early enteric fever, but, as pointed out by Sir W. Jenner, when delirium appears in this disease complaint of the head ceases. In meningitis, on the other hand, even after the delirium has appeared, complaint of the head continues. This distinction is more valuable in adults than in children.

In adult life, in adolescence, and even occasionally before puberty there arises not very rarely the difficulty of discriminating tuberculous meningitis from *hysteria*. The most reasonable view seems to be that these symptoms of defective control, when manifested in tuberculous meningitis, are as truly hysterical as when associated with some phases of disseminated sclerosis, and even with cerebral tumour. Emotional manifestation in early tuberculous meningitis may unquestionably be arrested by cold douches and by electrical stimuli, and thus a shallow observer may be led to give the assurance that there is no cause for further anxiety.

The occasional mistake of confounding early tuberculous meningitis in adults with *delirium tremens* or *mental disease* has already been mentioned. Every such case must be judged on its own characters; but the undue complaint of head pain and the occurrence of vomiting rarely fail us as aids to diagnosis. It is important to bear in mind that these patients also, like the pseudo-hysterical patients, are very prone to pass suddenly into a dazed condition and thence into coma.

The other group of diseases from which it is necessary to discriminate tuberculous meningitis comprises many different forms of intracanial organic mischief; and, although for the most part the members of this group are simulated in the middle and late stages of tuberculous meningitis, some of them have to be considered in the early stage.

The various brain complications of *ear disease* require careful exclusion. No doubt in the majority of cases, if the ear have been the primary offender, there will be indications of severe local trouble about the mastoid, tympanum, or meatus, with high fever and sometimes rigors; but sometimes tuberculous meningitis coexists with otorrhoea, or even with tuberculous caries of the petrous bone. In more than one case, trephining of the skull and exploration of the brain for supposed abscess consequent upon ear disease have been performed in cases which have proved to be tuberculous meningitis. The help which the ophthalmoscope yields is not conclusive, for it has been found that thrombosis of the sinuses consecutive to ear disease may be accompanied by optic neuritis. It is also important to note that although we may fail to identify tuberculous meningitis, ascribing its symptoms to the results of ear trouble, conversely one form of ear trouble is often wrongly diagnosed as tuberculous meningitis. This is *double otitis media*, as it is apt to occur in very young children. It often supervenes on general or pulmonary catarrh, pneumonia, and the like, without any preliminary otorrhoea; and the signs of local ear-distress may be nil. The child may be fretful and may scream frequently; it vomits now and then; it has an irregular pyrexia; sometimes a tense fontanelle, and, if not actual head-retraction, that early rigidity of the neck, seen on lifting the head from the pillow, which has been mentioned amongst the prodromes of tuberculous meningitis. Definite convulsions may occur, either of rigid tonic or bilateral clonic form. These are amongst the multiform results ascribed to dentition. But it is in the experience of many practitioners that such patients may continue sometimes for three weeks presenting great intolerance of food and very marked emaciation, and that recovery may begin after the appearance of a slight purulent discharge from one or both ears. I have been indebted to my surgical colleagues for early incision of the tympanic membrane on both sides in several of these cases; a little thin pus has escaped, and in several of them recovery was complete. In others the improvement, if any, was but temporary, and ultimately the necropsy shewed non-tuberculous meningitis either of the convexity or, more commonly, of the "posterior-basic" or cerebrospinal forms. The more detailed discussion of these cases is given in the article on "Meningococcic Meningitis" (p. 166). But it may be pointed out here that, as between otitis media and "posterior-basic" and cerebrospinal meningitis, the latter may be diagnosed when the head-retraction is very pronounced, and still more certainly when opisthotonos occurs. Marked head-retraction, as a persistent condition, is very rare in tuberculous meningitis, whereas in "posterior-basic" meningitis it is the rule, begins early, and may last for weeks unaccompanied by any other active sign except vomiting. In the rare cases of tuberculous meningitis which present severe and persistent head-retraction, an unusual and excessive deposit of tubercle at or near the posterior base is generally found at the necropsy. Retraction of the abdomen, on the other hand, such as occurs in tuberculous meningitis, is not characteristic of the cases of otitis media or "posterior-basic" or cerebrospinal meningitis; and the same statement may be made with respect to constipation. The whole series of limited clonic convulsions and temporary paralysis which are characteristic of the second period of tuberculous meningitis are conspicuously absent in the "posterior-basic" and cerebrospinal non-tuberculous forms. Kernig's sign was first described in connexion with cerebrospinal meningitis. The pathognomonic value of the sign has been shaken, because it has sometimes been found in other diseases, including uraemia. Morse (*vide* p. 214) does not admit its diagnostic value in infants between the cerebrospinal and the tuberculous forms of meningitis. But I believe that in the "posterior-basic" and cerebrospinal forms Kernig's sign, when present, will prove to be more constant day after day than in the tuberculous form in which the variations on consecutive days are marked.

Meningitis of the convexity, as a rule, is more rapidly fatal, presents earlier convulsions, and is less prone to dilatation of the pupils than ordinary tuberculous meningitis; but, as pointed out by Dr. Gee, the clinical discrimination of non-tuberculous from tuberculous meningitis of the convexity is impossible. Of the forms of meningitis due to syphilis, whether acquired or congenital, it may be observed (*a*) that they are exceedingly chronic, and (*b*) that they are apt to be associated with choroiditis disseminata, and with coexisting gummas or vascular disease, which are suggested respectively by persistent definite paralysis of cranial nerves and by persistent hemiplegia (*vide* also p. 197).

As between meningitis due to tuberculosis or to other causes, it is important to note Emmett Holt's observations with respect to the detection of tubercle bacilli in the sputum of children. He found that they were present in 52 per cent of the cases of tuberculous meningitis, although in many cases there were either no pulmonary symptoms or the symptoms and signs were very slight. The method adopted was to excite a cough by irritating the pharynx, and to catch the sputum on a bit of muslin held in an artery clamp.

Thrombosis of cerebral sinuses, whether infective or in association with marasmus, is, in some cases, exceedingly difficult to distinguish from tuberculous meningitis. Headache may be very severe, and if there be extensive small cortical haemorrhages into the grey matter (which are not uncommon in the marasmic variety), short repeated violent screams may simulate the hydrocephalic cry. Extensive haemorrhages in the fundus of the eye are occasionally a very important aid to the diagnosis of sinus thrombosis, as are indications of local venous engorgement about the eyes and temples. In the infective form the existence of a possible cause, such as disease of the bones of the ear or nose, or phlegmonous inflammation about the face, is of value; whilst the marasmic form would be suggested by debility due to prolonged diarrhoea or other exhausting diseases. (Cf. art. on "Occlusion of Cerebral Vessels," p. 303.)

Abscess of the brain is equally difficult to distinguish from tuberculous meningitis, especially in adults. The existence of a suppurative focus as a possible source and the occurrence of rigors are aids when present. But the close parallelism of the symptoms of tuberculous meningitis suddenly supervening upon pulmonary tuberculosis, with those of abscess or septic meningitis supervening on bronchiectasis or chronic empyema, is well recognised. The diagnosis hinges more on the pathological antecedents of the patient than on the evolution of the brain symptoms.

Intracranial tumour, especially a tuberculous mass, is frequently mistaken during one of its acute or advancing phases for tuberculous meningitis. This is not surprising, for multiple tuberculous tumours often have areas of circumscribed tuberculous meningitis in their neighbourhood. But as between tumour and tuberculous meningitis it may be observed that in the first the symptoms are more prolonged than in the second; and even in the active phases they do not shew the changes from day to day which are so characteristic of the latter disease. Moreover, as pointed out by Sir W. Gowers, the early occurrence of limb symptoms and the gradual loss of power indicate tumour rather than tuberculous meningitis. Ophthalmoscopically the optic neuritis of tumour is much more profound than that of tuberculous meningitis, and, besides shewing considerable swelling and sometimes haemorrhage, is often severe enough to end in atrophy.

The conclusive proof of some other coexisting tuberculous lesion in the body, especially if it be reawakened or active, is an important factor in the diagnosis of tuberculous meningitis. Tileston has lately drawn attention to the diagnostic value of certain lesions of the skin, which he states are, strictly speaking, tuberculous eruptions (tuberculides). Some of his cases seem to correspond with the acne scrofulosorum of Fox, and others with the varicella gangraenosa and ulcerosa of Sir J. Hutchinson. In several examples of the latter I found, some years ago, on post-mortem examination, more or less tuberculous disease of the viscera, and others also observed this, but the exact bearing of this relation was left open. The demonstration by Tileston of tubercle bacilli in some of these necrotic lesions is an important addition to knowledge. But, besides these necrotic lesions, which average the size of a pea, and may be much larger, Tileston lays special stress on the occurrence of small lesions 2 to 3 millimetres in diameter, each consisting of a papule of dull-red colour, with an umbilication covered by a crust. The spots appear in crops, so that all the different stages may be present at the same time. These spots occur predominantly on the buttocks, genitalia, and adjacent portions of the thighs, but a few may occur on the trunk, face, and limbs. This form Tileston considers especially common in cases of infantile miliary tuberculosis, and he maintains that it is pathognomonic. The essential feature is that he finds tubercle bacilli and giant-cells in these lesions.

Treatment.—*The preventive treatment* of tuberculous meningitis includes everything which is comprised in good hygiene. Some of the points to which recent investigators have drawn attention have been mentioned in the section on etiology; namely, a good milk-supply derived from cows proved by the tuberculin test to be free from tuberculosis; the milk up to the time of consumption being kept cool and protected from contamination. If such pure milk cannot be obtained, then the safest plan is to bring it up to ebullition. To this should be added, at the proper age, abundance of good food, especially of assimilable fat; and fresh air and sunshine should always be secured. In those children and adolescents in whom lymphatic gland activity is strongly marked it is most important to remove as soon as possible every peripheral irritation of skin and mucous membranes, lest chronic glandular enlargement be brought about. Timely operation on enlarged tonsils, adenoids, and carious teeth is very important; for these lesions afford opportunity for the entrance of tubercle bacilli into the glands, with which they are in lymphatic relation, and thus make subsequent haemic infection possible from the glands which have become tuberculous. Pulmonary and abdominal tuberculosis, and some of the tuberculous diseases of bones and joints, must inevitably leave behind them residual deposits, in glands and elsewhere, which cannot be dealt with surgically, and are apt to give rise to fresh infection. In many such cases all that can be done is to invigorate the patient by securing for him good food and favourable circumstances, so that the tuberculous material may become encapsulated. But with regard to glands in the neck, where in many cases the tuberculous material is circumscribed, there is much to be said for the removal of such potential foci of dissemination. If the active inflammatory process in such glands has taken on an autonomy of its own, the proper surgical treatment of it becomes tolerably simple. But in the ambiguous subacute cases the most assiduous efforts should be made, by change to sea or mountain air, and by the use of both local and constitutional remedies, to reduce the peripheral irritation which originally set up the mischief in the glands. When this has been accomplished, if the glands remain permanently enlarged, and still more if they shew indications of disintegration, the safest step is to remove them. Sir A. E. Wright's researches would seem to shew that before removal of tuberculous glands is performed the value of the opsonic index against tubercle should be ascertained, and, if necessary, the resisting power of the patient should be raised by minimal injections of tuberculin.

Treatment of the Disease.—We have seen that it is doubtful if any case of generalised tuberculous meningitis can end in recovery; but, from circumscribed patches of it, in which there is no encephalitis, it would seem that recovery is possible. Whether any drug can exert a definite influence upon such local outbreaks is difficult to prove; but mercury is a powerful germicide, which appears to modify certain inflammatory processes. Experience on the whole favours its employment. It may be given either as perchloride (ʒj. doses of liq. hydrarg. perchlor.), or combined with iodide of potassium; or as calomel or grey powder; or best of all as an inunction (ʒj. ung. hydrarg. to be applied on a binder daily).

Blisters, setons, and irritating ointments to the scalp are useless and give needless distress. An ice-bag to the head sometimes relieves irritability in the early stage, but often annoys the patient; in cases of hyperpyrexia it may be useful in lowering the temperature. If there be much pain in the head, hot sponging sometimes gives relief, and the application of one or two leeches is occasionally helpful.

Bromides and chloral are indicated in the convulsive stage, and there is something to be said for the cautious employment of opium.

The constipation can generally be relieved by grey powder, small doses of castor oil, and enemas.

Feeding is often difficult throughout. In the early stages there may be active resistance; in the middle stage it may be difficult to rouse the sensorium. As with other features of the disease, swallowing is curiously variable. One day there may be great difficulty in the act, on the next no trouble whatever. In the third stage, however, the swallowing reflex seems to be lost, and there is a risk of liquids passing into the larynx. Nasal feeding and nutrient enemas are useful at intervals throughout the disease.

Tuberculin Treatment.—Koch's original method of tuberculin injection (T.O.) can only be mentioned for its pathological interest. It was proved that after the injection there was great increase of hyperaemia around the tuberculous deposits, and that signs of compression of the brain became decidedly more evident, in consequence of the inflammatory reaction. Of the value of the later and more benign preparation (T.R. and the Tub. Bac. Emulsion) introduced by Koch we have as yet inadequate evidence in the treatment of tuberculous meningitis.

Dr. N. Raw reports 3 cases of what he believed clinically to be tuberculous meningitis in which Koch's new tuberculin was repeatedly injected. Two of them completely recovered, and one died uninfluenced by the treatment. In these cases he says it was impossible to isolate the tubercle bacillus. This must be held to invalidate the diagnosis if the cerebrospinal fluid removed by lumbar puncture was adequately examined, because there seems to be no doubt that in tuberculous meningitis tubercle bacilli in this fluid are practically constant (*vide* p. 218).

Surgical Treatment.—Lannelongue in one case trephined in four places (occiput and frontal), and attempted to irrigate the base of the brain with a through stream of weak solution of perchloride of mercury, in the hope of thus dealing effectively with the chief sites of tuberculous activity in the Sylvian fissures. There was much haemorrhage from the occiput, and the result was fatal.

Less heroic attempts have aimed simply at the withdrawal of the fluid, either directly from the lateral ventricle or from the subarachnoid space. The lateral ventricles have been attacked through the frontal lobe by Bergmann; through the temporo-sphenoidal lobe by Poirier, Keen, and Kendal Franks; through the arm centre by Mayo Robson. In many of these operations there has been temporary benefit so far as compression signs were concerned; and sometimes the benefit has lasted for four or five days. But the ultimate result has been discouraging. Robson's case was the most satisfactory. It was that of a girl, aged ten years, who had hemiplegia with aphasia and optic neuritis supervening on pain and deafness referred to the left ear with otorrhoea. Trephining was performed over the arm centre, and six drams of serous fluid were withdrawn. At the time of the record considerable recovery had taken place, but there were still occasional spasms of the right arm. In this case the diagnosis of tuberculous meningitis was admitted to be doubtful.

The withdrawal of fluid from the subarachnoid space has been attempted both in the region of the lumbar spine and at the occiput. The non-closure of the cerebrospinal foramen, and the absence or scantiness of inflammatory deposit in that situation in tuberculous meningitis, render the free evacuation through puncture of the spinal theca comparatively simple. Four of the early cases were reported by Dr. Wynter. Quincke, Ziemssen, Fürbringer, and others have made extensive trials of this method. Considerable quantities of fluid can be evacuated, there is no difficulty in repeating the operation several times, and the risks are very small. The most complete and detailed investigation of this measure recently recorded by Connall only claims that some amelioration of the headache and restlessness in tuberculous meningitis can be obtained by lumbar puncture, and that this is most manifest in the adult cases.

The occipital operation has for its object the drainage of the ventricles by means of the cerebrospinal foramen, and by the posterior subarachnoid reservoir which is bridged over by the arachnoid extending from the cerebellum to the medulla oblongata. Of the two recorded cases, the first, by Mr. Parkin, was proved after death, which followed sixteen hours after the operation, to be an undoubted example of tuberculous meningitis. The other case recorded by Dr. W. W. Ord and Mr. Waterhouse was not absolutely proved to be tuberculous. Recovery followed trephining the occipital bone and the drainage thus provided. About ten years later the patient came under Mr. Waterhouse's observation with tuberculous disease of the left elbow-joint. She gave a positive reaction with tuberculin. If the tuberculous nature of the brain attack in this case is conceded its importance cannot be gainsaid. Previous writers who have urged the desirability of surgical interference in tuberculous meningitis, either by draining the lateral ventricles or the subarachnoid space, have for the most part based their arguments on the advantage of lessening intracranial pressure. Dr. Ord and Mr. Waterhouse, drawing an analogy from the benefit which sometimes occurs from simple laparotomy for tuberculous peritonitis with effusion, urge that the removal of fluid in tuberculous meningitis may be advantageous in lessening the activity of growth of the tuberculous granulations. My own belief is, that in tuberculous meningitis the part played by the hydrocephalus has been exaggerated, and that the deleterious effect of the increased fluid pressure is not more important than the widespread cerebritis and its attendant softening which are brought about by the spread of the tuberculous invasion from the pia mater to the brain tissue. Experience seems to shew that the drainage of a moderate effusion in the lateral ventricles may be followed by excellent results if there be no widespread and severe damage to the brain tissue; and again the results of surgery have proved that, with proper methods of evacuation, the brain may recover from a local necrosis of moderate extent. But the widespread cerebritis and softening which, together with hydrocephalus and an extensive plugging of small vessels, sum up the conditions before us in unmistakable tuberculous meningitis, would appear too formidable a combination for any surgical procedure to remove.

Thomas Barlow.

REFERENCES

1. Allbutt, C. *On the Use of the Ophthalmoscope in Diseases of the Nervous System and of the Kidneys, also in certain other General Disorders*, 1871.—2. Barthez et Rilliet. *Traité clinique et pratique des maladies des enfants*, 2nd edition, 1854, iii. 445.—3. Bastian. "Tubercular Meningitis," Quain's *Dictionary of Medicine*, 2nd edition, 1894, ii. 48.—4. von Bergmann. *Die chirurgische Behandlung von Hirnkrankheiten*, 1889.—5. Bernard, Gustave. *Tentatives chirurgicales dans le traitement de la méningite tuberculeuse*, 1895.—6. Bernstein. "The Value of Lumbar Puncture with special Reference to the Diagnosis of Tuberculous Meningitis," *Mt. Sinai Hosp. Rep.*, N.Y., 1907, v. 491.—7. Cabot. *Clinical Examination of the Blood*, 1904, 303.—8. Connall. "A Study of the Cerebrospinal Fluid in Infective Diseases of the Meninges," *Quart. Journ. Med.*, Oxford, 1910, iii. 152.—9. Epstein. "Über Tuberculose im Saüglingsalter," *Prager Vierteljahrsschrift*, 1879.—10. Forbes, "The Pathology of the Cerebrospinal Fluid derived from Lumbar Puncture," *Quart. Journ. Med.*, Oxford, 1908, i. 109.—11. Franks, Kendal. "Tapping and Draining the lateral Ventricles in a Case of Tubercular Meningitis," *Brit. Med. Journ.*, 1890, ii. 1294.—12. Fürbringer. *Deutsche med. Wchnschr.*, 1895, xxi. 739.—13. Garlick. "Observations on the Ophthalmoscopic Appearances in the Tubercular Meningitis of Children," *Med.-Chir. Trans.*, 1879, lxii. 442.—14. Gee, S. "Tubercular Meningitis," Reynolds's *System of Medicine*, 1872, ii.—15. Gowers. *Diseases of Nervous System*, 1893, ii.—16. Greenfield. "Cases of Tubercular Meningitis," *Lancet*, 1874, i. 266, 297, 367, 584, 833.—17. Hall, Marshall. "The Hydrocephaloid Disease," *Medical Essays*, 1825; also *Diseases and Derangements of the Nervous System*, p. 153; 1841.—18. Hayem. *De l'encéphalite*, Thèse, 1868, 70.—19. Henoch. *Lectures on Children's Diseases*, translated by John Thomson, New Sydenham Society, 1889, i.

317.—20. Higgs. *Brit. Med. Journ.*, 1909, i. 1170.—21. Holt. *Diseases of Infancy and Childhood*, 1909, 770.—22. Keen. "Surgery of the Lateral Ventricles," *Verhandl. des X. internat. medicin. Congresses*, Berlin, 1890, Band III. *Chirurgie*, S. 108.—23. Landowski et Claret. "Polynucléose rachidienne dans trois cas de méningite tuberculeuse," *Arch. gén. de méd.*, Paris, 1907, cxcviii. 584.—24. Martin, A. "The Occurrence of Remissions and Recovery in Tuberculous Meningitis: a Critical Review," *Brain*, London, 1909, xxxii. 209.—25. Morse, J. L. "Kernig's Sign in Infancy—Study of Two Thousand Cases," *Arch. Pediat.*, N.Y., 1908, xxv. 167.—26. Ord, W. W., and Waterhouse. "Case diagnosed as Tubercular Meningitis treated by Trephining and Drainage of the Subarachnoid Space: Recovery," *Lancet*, 1894, i. 597.—27. Papavoine. *Propositions sur les tubercules considérés spécialement chez les enfants*, 1830.—28. Parkin. "The Relief of Intracranial Pressure by the withdrawal of Cerebrospinal Fluid from the Basal Subarachnoid Cavity," *Lancet*, 1893, i. 21, 1244.—29. Poirier. *Topographie cranio-encéphalique trépanation*, 1891.—30. Quincke. *Berlin. klin. Wchnschr.*, 1891, xxviii. 929, 965.—31. Rendu. *Recherches cliniques et anatomiques sur les paralysies liées à la méningite tuberculeuse*, 1873.—32. *Reports of Royal Commission on Tuberculosis*, 1895, 1907-8.—33. Robson, Mayo. "Tapping and Draining the Ventricles in Certain Cases of Brain Disease," *Brit. Med. Journ.*, 1890, ii. 1292.—34. Seitz. *Die Meningitis tuberculosa der Erwachsenen*, 1874.—35. Senn. *Recherches anatomico-pathologiques sur la méningite aiguë des enfants et ses principales complications: hydrocéphale aiguë des auteurs*, 1825.—35*a*. Tileston. "Disseminated Miliary Tuberculosis of the Skin," *Arch. intern. Med.*, Chicago, 1909, iv. 21.—35*b*. *Tuberculosis in Infancy*, edited by T. N. Kelynack.—36. Whytt, Robert. "Observations on the Dropsy in the Brain," *Works of Robert Whytt*, p. 723; 1768.—37. Wilks, S. "On some Diseases of Children," *Guy's Hosp. Rep.*, 1860, 97.—38. *Idem.* "Cases of Tubercular Meningitis in Adults which have occurred lately in Guy's Hospital, with Remarks on the Obscurity of the Early Symptoms," *Med. Times and Gaz.*, 1863, ii. 274.—39. Winternitz. "Tuberculosis of the Parathyroid and Tetany in Tuberculous Meningitis," *Bull. Johns Hopkins Hosp.*, Balt., 1909, xx. 269.—40. Wright, A. E. *Med.-Chir. Trans.*, London, 1906, lxxxix. 1.—41. Wynter. "Four Cases of Tubercular Meningitis in which Paracentesis of the Theca Vertebralis was performed for the Relief of Fluid Pressure," *Lancet*, London, 1891, i. 981.

T. B.

DISEASES OF THE BRAIN

Some milder Forms of Concussion.
Acute Polioencephalitis.
Abscess.
Intracranial Tumours.
Occlusion of the Cerebral Vessels.
Haemorrhage.
Intracranial Syphilis.
General Paralysis of the Insane.

Aphasia and other Speech Defects.
Apraxia and Agnosia.

Cerebral Palsies of Children.
Amaurotic Family Idiocy.

Paralysis Agitans.
Epilepsy.
Chorea.
Migraine.
Impediments of Speech.
Tetany.
Recurrent Paralysis.
Tics.
Facial Spasm.
Paramyoclonus Multiplex.
Eclampsia Nutans.
Head-Nodding.
Occupation-Neuroses.
Hysteria.
Neurasthenia.
Disorders of Sleep.
Night-Terrors.

SOME MILDER FORMS OF CONCUSSION OF THE BRAIN

By Sir WILLIAM BENNETT, K.C.V.O.

ALTHOUGH the more severe cases of concussion of the brain come immediately under surgical treatment, a large number of the slighter cases, escaping treatment altogether at first, in the course of a few days after the accident fall into the hands of the physician in consequence of symptoms which are set down by the patient to causes unconnected with the injury, to which, however, they are really due. Indeed, my own experience tends to shew that even now medical men are not always alive either to the importance of these symptoms, or to the relation which they bear to an injury which in some instances may have appeared to be trivial. This want of vigilance appears to be due mainly to the teaching of most of the surgical textbooks, which still insists upon pronounced loss of consciousness as a necessary symptom of concussion of the brain. Without ignoring such points as may be usefully discussed in relation to concussion of the brain generally, it is my intention to deal more especially with the minor cases. For this no apology is offered, since it is certain that the apparently trivial nature of some of these cases leads finally, for reasons which will become manifest, to their proving of more importance than many of those cases which are attended immediately upon the occurrence of the injury by much graver symptoms, as, for example, prolonged unconsciousness.

The consideration of the subject must obviously be from a purely clinical standpoint, seeing that in the conditions dealt with in the present article opportunities for pathological investigation are rarely if ever forthcoming.

For practical purposes cases of concussion of the brain may be classified as follows :—

1. Those in which unconsciousness, following immediately upon injury, lasts for a period of one hour and upwards.
2. Those in which unconsciousness persists for periods varying from a few minutes to an hour.
3. Those in which the loss of consciousness occupies a few seconds only.
4. Those in which no loss of consciousness occurs at all.

Classes 1 and 2 represent the severe forms of concussion as described in the various textbooks, and, by reason of their grave and unmistakable symptoms, are sure of immediate treatment. Classes 3 and 4 are the milder forms which, in consequence of the slightness of the symptoms, frequently escape treatment altogether in the first instance; especially as unconsciousness is very transient or entirely absent. Hence cases of this class, merely from primary neglect on the part of the patient, or from failure on the part of the practitioner to appreciate the possible importance of the condition, may in the end have far graver results than the severe cases in which treatment as a matter of course is rigidly enforced from the beginning.

Loss of consciousness, as such, is no necessary concomitant of concussion of the brain. Injury, if sufficiently severe, will cause complete unconsciousness for periods varying from minutes to hours or days. Many cases, however, occur that may be accurately described as examples of concussion, in which, the injury being slight, the loss of consciousness may either be so transient as to escape notice entirely, or may not occur at all—a feeling of being "dazed" or confused for a few moments being all that the patient notices, excepting perhaps a temporary sensation of giddiness.

In these milder cases the symptoms are at times mistaken for indications of "biliousness," or of other ailments with which they are not really connected; their true cause being ignored or overlooked. Such cases are therefore of much interest, and, considering the serious results to which they sometimes lead, have not received the general attention they merit.

The degree of loss of consciousness or brain confusion in concussion depends mainly upon the severity of the injury; and in severe cases the whole cranial contents are of necessity more or less uniformly shaken and jarred. In the slighter cases, however, not only is the loss of consciousness merely partial or altogether wanting, but, as it appears from my own experience, the concussion does not necessarily affect the whole of the intracranial nervous apparatus, but may be limited either to the cerebrum proper, or to the medulla and pons with their connexions lying in immediate relation to the base of the skull, especially to the basilar process. It also appears that the symptoms arising in concussion of these two portions of the brain in cases of slight injury differ sufficiently to justify their separate consideration under the heads of concussion (*a*) of the brain proper (cerebrum), and (*b*) of the parts extending from the mesencephalon above to the cerebrospinal junction below.

The situation of the intracranial damage seems to be determined by the kind of injury received. Direct blows on the skull—as, for instance, from a hammer, or a projecting branch of a tree—are followed by concussion of the brain proper; whilst indirect injury, such as may occur in falls on the buttocks—the trunk being at the same time vertical or nearly so—may be followed only by damage to the parts immediately above the cerebrospinal junction, the latter kind of damage being consequent on the jamming of the parts against the bony surface, with which they lie in contact, by the sudden impact of the heavy structures above, at the moment when the body is arrested in its fall. I am aware that exception may be taken to the possibility of differential concussion of portions of the intracranial contents in the manner suggested; but it is, I believe, a fact, which admits of no denial, that the two kinds of injury (direct and indirect) produce symptoms which are so far distinct from each other as to be easily recognisable if a little care be used in their investigation.

It will be found further, in many instances of severe concussion with profound unconsciousness, that the symptoms following upon the reactionary stage will by degrees conform to the type characteristic of damage by direct or indirect injury, as the case may be. The appreciation of the difference in the symptoms of these two conditions is of small importance in relation to treatment, since all cases of concussion must be managed upon the same principles; but in the matter of prognosis it is of considerable moment, as will presently be shewn.

CONCUSSION FROM DIRECT BLOWS ON THE CRANIUM (Concussion of the Cerebrum).—After a direct injury to the skull, if the violence be sufficiently great, the ordinary symptoms of concussion with profound unconsciousness must follow immediately. In the slighter cases, when the loss of consciousness is very transitory or altogether wanting, no symptom may follow directly upon the injury sufficient to attract the patient's attention, or at all events to lead him to seek treatment at the hands of a medical man. Sooner or later, however, after the lapse of a few hours or perhaps of days, symptoms almost always arise which lead to the summoning of advice, although they may not then have any connexion in the mind of the patient with the apparently trivial accident met with a short time before.

Symptoms.—The first symptom to attract attention is usually pain, with or without giddiness, nearly always associated with restlessness and constipation; a combination which leads the patient to suppose that the trouble is due to "biliousness." Careful examination at this stage elicits the following symptoms:—Pain, giddiness, a general feeling of sensitiveness about the cranium, an unnatural aspect of face, a distinctive character of pulse, and a peculiar mental mood. The pain is more or less acute, extending over the vertex and across the brow; it is increased by movement, and is worse after taking solid food. The giddiness bears a direct ratio to the amount of pain, and is influenced by the same conditions. The peculiar facial expression, although not always sufficiently well marked to attract attention upon superficial observation, is nearly invariably present; it is difficult to describe it better than by saying that the patient has a dazed and sometimes half-frightened look. The pupils as a rule are irritable; but they may be sluggish, and then are often unequal: photophobia, in a minor degree, is not infrequent. The symptom which never fails in constancy and quality is a peculiar character of the pulse which is common to every case of concussion of the brain, severe or slight; and is never absent, excepting temporarily during the reactionary stage in very severe cases with profound unconsciousness. This pulse, when felt by the finger in the ordinary way, seems sufficiently good, and at first perhaps suggests nothing worthy of note; it comes up quickly under the finger and seems sufficiently strong; but the slightest pressure reduces it to nothing; it is extraordinarily compressible—the phrase "action without power" fairly describing its character. In cases of the class under discussion this peculiarity of the pulse may be the first symptom to attract attention, and the last to disappear; the rate varies from 70 to 100 per minute, and the tendency is to rapidity rather than the reverse. The skin is warm to the touch and inclines to dryness; very rarely is there any unnatural moisture. Intra-ocular changes (choked disc) occasionally occur, but are temporary; the reflexes (knee-jerk, etc.) are generally unaffected.

Wasting of the muscles occurs, especially of those of the extremities, and is much more manifest if the patient is kept to bed; but it does not, as a rule, exceed in degree that which may naturally be produced by disuse.

The mental mood inclines to irritability and antagonism; the patient resents the restrictions necessary for his proper treatment; tends to become over-critical, and is sometimes suspicious of those about him.

Concussion from Indirect Injury (Concussion of the parts immediately above the cerebrospinal junction).—Indirect injury, if sufficiently violent, as in the case of direct blows, will cause general concussion with profound unconsciousness, varying in degree with the extent of the damage.

In the slighter cases the immediate result of the injury is frequently no more than a very temporary mental confusion without loss of consciousness. These apparently trivial cases are again, as in the milder forms of those resulting from direct injury, of great importance, as the insignificance of the initial symptoms sometimes leads to neglect.

After the passing away of the mental confusion at the moment of the injury the patient so far recovers that he is not usually led to seek advice until some hours, days, possibly a week or even a fortnight later; when pain and feelings of general depression call attention to an unsatisfactory condition of health, which is commonly attributed to gout by the patient himself, and sometimes even by the medical practitioner.

Symptoms.—The main symptoms elicited at this stage are—pain, a general feeling of heaviness about the head, restlessness at night, sweating after slight exertion, the peculiar pulse, evidence of rapid muscle wasting, and a peculiar mental mood.

The pain is dull and limited to the parts about the occiput and nape of the neck; very rarely does it extend above the occipital region; with the pain and limited to the same area is a feeling of heat, which is often so much in excess of the pain that it is the only discomfort referred to. The head is held somewhat stiffly, and is as a rule bent a little forwards. The parts below the occipital protuberance and about the nape of the neck are frequently too sensitive to the touch. The expression, beyond suggesting fatigue, is natural. Photophobia is rarely present, but muscae are often complained of, and "singing" in the ears frequently causes annoyance.

The feeling of heaviness is general and amounts at times to an uncontrollable lethargy. With this is a peculiar mood of mental apathy; the patient feels ill, but does not resent his illness, and shews no great desire to escape from it; subsequently there is a strong inclination to lapse into contented invalidism. The pulse is of the character already described, and inclines to infrequency, sometimes slowing down to 30 beats to the minute. In a cabman under my care in St. George's Hospital the pulse-beat fell to 28 in a minute. In some cases this slowness of pulse becomes practically permanent. The temperature remains nearly normal, as a rule; if any change occur, it is usually to fall below the normal. The urine sometimes contains sugar. The sweatings are fairly constant and characteristic; they follow any exertion, however slight, and are always ushered in by the feeling of heat about the back of the head already referred to; at night they occur spontaneously and are profuse, coming on generally as the patient awakes from sleep—especially if a change from the lying-down to the sitting-up position is rapidly made. General muscular wasting is remarkable, and is sometimes so rapid as to become very marked in a few days; it occurs whether the patient be confined to bed or not, and in the former case is altogether in excess of that which may come of mere disuse. As the appetite is usually good, and sometimes voracious, adiposity often rapidly follows the muscular wasting.

The reflexes (knee-jerk, etc.) are not affected, as a rule; the knee-jerk, however, sometimes inclines to slight exaggeration, and in rare cases may be very excessive.

Comparison of the symptoms described above, as resulting from direct and indirect injury of the milder sort, indicates distinct differences which can hardly be mistaken in exemplary cases. It is clear, however, considering the great difference in the degree of violence and in the conditions under which it is inflicted in different cases, that in many instances (indeed, in the majority) the symptoms must at first so much overlap one another as to lose their distinctive characteristics; although, as has already been stated, they tend strongly, as the condition of the patient improves, to conform to one type or the other. The importance of this fact in prognosis is considerable. The resemblance of the symptoms, in the milder class of cases, to certain forms of illness from liver disturbance, gout, and the like, requires no analysis here. It may, however, be excusable to suggest the wisdom of paying more attention to the possibility of some slight injury of the head as the cause of illness associated with vague, ill-defined pain and discomfort about that part, rather than to assume at once that the symptoms have some gouty or dyspeptic origin. I have less hesitation in suggesting this somewhat obvious precaution, as cases of the kind have come under my personal observation in which symptoms clearly due to an injury, too trivial to attract the patient's immediate attention, were considerably aggravated by the treatment for gout—including in more than one case a visit to Aix-les-Bains and other European "watering-places"—which could hardly have been advised had the recent injury been known to those concerned in treating the patients. The association of the peculiarity of pulse described, with a history of injury to the head, however slight it may appear to have been, should leave no room for a misapprehension about the nature of the case.

Prognosis.—The prognosis, with regard to the life of the patient, in severe concussion of the brain depends, in the first place, upon the gravity of the injury and the degree of shock; and, secondly, upon the efficacy, or the reverse, of the treatment carried out. Comparatively few cases of concussion alone prove immediately fatal (from shock); but gross organic brain lesions, such as laceration or haemorrhage, may cause death, either immediately or very shortly after the accident. If a rapid rise in temperature and pulse-rate occur, danger to life may also arise in severe cases during the stage of reaction, the cause of death in these circumstances being generally acute meningitis; but acute meningitis is rare in concussion without fracture of the skull, or some gross lesion of the brain or membranes. In young and delicate subjects tuberculous meningitis may supervene, and cause death in cases in which the temperature has been high in the reactionary stage: this result, however, rarely happens unless the tuberculous tendency is strong.

In simple concussion the gravity of the case increases step by step with the period during which profound unconsciousness lasts; and it is undeniable that recovery from profound insensibility is more rapid, and the stage of reaction less severe, in cases complicated with scalp wounds which give rise to free bleeding; a fact which has an important bearing upon treatment. The vomiting which ushers in the stage of reaction is usually salutary, but in very severe concussion in feeble subjects it may be followed by syncope and fatal collapse. The progress after the stage of reaction depends almost entirely upon the efficacy of the treatment. However severe the case, complete recovery may be reasonably anticipated under proper management; provided, of course, that no gross organic lesion exists. On the other hand, if the treatment be inadequately carried out, the recovery will be partial; the defect in recovery depending upon the want of proportion between the treatment and the gravity of the case. A very severe case treated adequately will be more likely to end in complete recovery than a comparatively mild one imperfectly managed; the commonest cause of imperfect recovery being the return of the patient too soon to the ordinary habits of life.

In the milder cases no immediate danger to life arises, but it is common for the recovery to be only partial, and this on two accounts:—(i.) the neglect of all treatment at first; and (ii.) the failure to continue the proper restrictions long enough. The first of these causes is the more important, because treatment is sometimes neglected until the patient is beyond the stage in which complete recovery in the full sense is possible. Complete recovery means the return of the patient to the normal condition of health in all respects; partial recovery includes every degree, ranging from helpless chronic invalidism, or mental derangement, to a state so near the normal condition of health that the only symptom detectable may be the persistence of the peculiarity of pulse described earlier in this article. Cases of slight concussion, if treated properly at once, invariably end in complete recovery; but neglected cases of this class afford the majority of imperfect cures. A certain number of slight cases do undoubtedly end in recovery without any treatment at all, but this number is very small indeed; in nearly all the apparent recoveries after neglect of treatment some trace of the damage shews itself sooner or later. The patient is rarely quite restored to the same condition as before the accident; age advances prematurely; energy is to some extent lost; tastes change, and peculiarities, sometimes of an objectionable kind, appear which are quite foreign to the original disposition of the individual.

Speaking generally, it may be considered that in cases in which irritability and antagonism are amongst the early symptoms (that is, in the type of case described under the head of concussion from direct injury), the results of imperfect recovery tend to the manifestation of vicious peculiarities, such as ill-temper, criminal inclinations, insanity, and proneness to suicide. On the other hand, in cases like those described under the head of concussion from indirect injury, in the earlier stages of which all symptoms of resentment or annoyance are conspicuous by their absence, the patients are prone, when recovery is incomplete, to sink into contented invalidism with a melancholy tendency, to become grey early and prematurely aged, or to assume entire indifference to calls of business or duty. These patients display no vicious inclination, nor any sign of lunacy or suicidal tendency; but they seem incapable of applying their minds to any useful end. A curious form of partial recovery, which may occur in any case of concussion, is that in which, while the health is generally good, the patient completely changes in disposition, sometimes in a most striking manner. It is not unlikely that amongst certain classes neglected cases of slight concussion are more responsible for indifferent health, lassitude, and apparent indolence than is commonly suspected. The occurrence of temporary glycosuria in some of these cases is a symptom which does not appear to affect prognosis; it would, however, be a matter of great interest to determine if injuries of the brain, in which glycosuria follows immediately, dispose to the occurrence of diabetes at a later date.

Treatment.—The treatment of all cases of concussion of the brain, whether they be severe or slight, may conveniently be dealt with in three stages: (i.) immediately after the injury; (ii.) during the period of reaction; (iii.) during the period subsequent to the reaction stage.

In a general sense, it may be held that incompleteness of recovery in slight cases depends for the most part upon inadequate treatment immediately after the injury; and, in severe cases, upon imperfect management during the period subsequent to the reaction stage.

(i.) *Treatment immediately after the Receipt of Injury.*—Absolute rest in bed is the first essential indication; and a mercurial purge should be given at once, 5 grains of calomel (in adults) sprinkled on the tongue being the most effectual, and, indeed, in profound unconsciousness, almost the only practicable means of attaining the desired result. The nourishment should be sustaining and of a fluid kind; alcohol is inadmissible unless indicated by special symptoms. In severe cases no difficulty, of course, arises in the treatment of the early stage; but in the slight cases, in which the loss of consciousness or mental confusion is very transient, leaving the patient for the time being apparently well, great and sometimes insuperable difficulty is met with in inducing the injured person to submit to the restraint and inconvenience which adequate rest entails. Nevertheless, it is impossible to exaggerate the importance of immediate rest, even in the slightest cases; for it is upon this immediate quiet that the ultimate issue of the case almost always depends. Although sometimes it may be impossible to induce the patient to pursue the proper course, the practitioner has at all events no further responsibility if his advice is ignored, or followed imperfectly.

Obvious as the indication may appear, I make no apology for urgently insisting upon the necessity in these mild cases for complete rest at once, or as soon as possible; from my own experience it is clear that there is a conviction amongst not a few practitioners that in such apparently trivial cases nothing is necessary beyond free purgation, a low diet for a few days, and a temporary abstention from alcohol, with perhaps some very short and incomplete rest—a line of treatment which directly favours incomplete recovery in its various degrees.

(ii.) *Treatment during the Period of Reaction.*—In severe cases the classical means, as described in the textbooks, are still the main indications; that is to say, a continuance of complete rest, a low diet, and gentle purgation. A comprehensive discussion of the treatment of the complications which may arise at this stage—such as vomiting, rise of temperature, increase of pulse-rate, headache, and sleeplessness—is beyond the scope of the present article. The following points, however, are worthy of special attention: there is, as I have already said, no doubt that the severity of the symptoms in the reaction stage is, other things being equal, less in cases in which bleeding occurs from wounds received at the time of injury. It is equally certain from my experience that all symptoms during the period of reaction are diminished by a moderate blood-letting (say ℥vj. or ℥viij.) at its onset; this operation may safely and with advantage be practised in all cases in which there is distinct increase in pulse-rate or blood-pressure with or without rise of temperature. Indeed, I have good reason for believing from practical experience that, even in cases in which the symptoms of reaction are less marked, a small abstraction of blood by venesection is advantageous. One thing at least is certain, that nothing is so effectual in removing the headache, which is often met with at this stage, as the application of two or three leeches behind the ear, or to the nape of the neck.

It is interesting to note the difference in the estimation, by different surgeons and at various hospitals, of the value of the application of extreme cold by means of the ice-bag, or other means, in the treatment of concussion of the brain. Some years ago at St. George's Hospital the application of the ice-bag was a routine treatment in concussion cases, the head having been previously shaved. At the present time this treatment is altogether exceptional; in fact it has been discontinued. So far as my observation goes, the effect of cold is negative; for in a careful comparison of cases treated by (*a*) the ice-bag or Leiter's tubes, (*b*) by hot applications, and (*c*) without any local application to the head, it was found that the progress in parallel cases was step by step the same; and that if any material relief to pain or discomfort were afforded it was by the application of heat rather than of cold. There appears to be no objection to the discreet use of any sedative or narcotic, excepting morphine, which, unless called for by special circumstances, should be avoided.

Upon the treatment of the stage of reaction in the slight cases it is unnecessary to dwell, since the period of reaction, although it occurs in all such cases, is in them so slight or transitory as to be hardly noticeable. In fact, the treatment of all the slight cases may be said, for practical purposes, to begin with the stage subsequent to reaction; especially as these cases are rarely seen by the practitioner until the period of reaction has passed.

(iii.) *Treatment during the Period subsequent to the Stage of Reaction.*—Upon the termination of the stage of reaction the complete rest and modified diet (beef-tea, soup, milk, and the like) must be continued for a time, which necessarily varies in different cases. Upon the discretion of the practitioner in the management of diet, and in the selection of the proper time for discontinuing complete rest, the recovery of the patient for the most part depends. In this connexion the principal points requiring consideration are the following:—

A. The time for the return to solid food.

B. The time for discontinuance of complete recumbent rest, and the time when the patient may be allowed to leave the bed and by degrees resume the habits of ordinary life.

A. *The Return to Solid Food.*—Solid food should not be allowed until convalescence has fairly set in; and, in determining the time for making the change from a liquid to a solid diet, the condition of the pulse and the facial expression are the sole trustworthy guides. As soon as the dazed, vacant, or frightened look has left the face, soft solid food, such as boiled sole or whiting, may be given in small quantity; the recumbent position being strictly maintained, and the effect carefully watched. If, after food of this kind has been taken for three or four days, or in bad cases for a week or ten days, no ill-effects follow in the form of dyspepsia, giddiness, headache, increase of pulse-rate, or rise of temperature, the same sort of food should be continued, the patient whilst eating being allowed to sit upright in bed. Should none of the ill-effects mentioned then appear, white meat, such as chicken, may be allowed and continued for some days, when, if still no adverse symptoms arise, ordinary diet may by degrees be resumed.

If in altering the kind of diet in the manner indicated any of the symptoms mentioned supervene, the former diet should be resumed for a time, which must be determined to some extent by the severity of the unfavourable symptoms recurrent.

The period which intervenes before solid food can be taken with comfort necessarily varies according to (α) the severity of the case, and (β) the degree of the efficiency of the treatment adopted immediately after the injury; a period which may vary from a few days to months. In all cases it is necessary, upon the resumption of ordinary diet, to insist upon moderation; as it is a singular fact that after some of these injuries a most voracious appetite is awakened, which, if allowed to be indulged unchecked, may lead to untoward complications.

B. *The Time at which the Patient may be safely allowed to leave the Bed, and by Degrees resume the Ordinary Habit of Life.*—As soon as solid food can be taken without giving rise to any symptom of discomfort, or without producing any change in the pulse or temperature, permission may be given for the patient to leave the bed for a short time daily. In allowing this freedom, it must not be forgotten that it is, as a rule, most difficult to induce a patient to return to recumbent rest; it is therefore important that this permission should not be given too soon. Speaking generally, no patient should be allowed to leave the bed until the pulse has resumed its normal character. The progress toward complete liberty of action on the patient's part must of course be gradual; the occurrence of vertigo, and especially of sweating, is a certain indication that too much is being done, and, whether they occur singly or together, should always be regarded as important warnings. If slight exercise is not followed by any abnormal perspiration or other easily appreciable discomfort, it is generally safe to conclude, if the pulse is normal, that recovery will follow in the ordinary course.

In certain cases, which at their onset have been neglected, it will be found that the pulse permanently assumes the morbid characters described, and tends to become intermittent: in such cases, although an attempt to resume the ordinary habits of life must sooner or later be made, complete recovery rarely follows, the incompleteness of recovery varying from slight mental inertia, or slight loss of memory, to unmistakable dementia. The discussion of the various remote phases of partial recovery is beyond the limit of this article, and in substance will be found dealt with elsewhere.

William H. Bennett.

ACUTE POLIOENCEPHALITIS

By Frederick E. Batten, M.D., F.R.C.P.

The name "encephalitis" has been applied to various affections characterised by evidence of inflammation of the brain, and in such a sense would include cerebral abscess and many cases of meningitis, which are necessarily accompanied by some implication of the cerebral substance.

In 1881 Wernicke designated as polioencephalitis acuta haemorrhagica superior a condition of acute ophthalmoplegia in the adult, especially in association with alcohol; and a series of such cases have been collected and recorded by Wilbrand and Saenger. Strümpell in 1884 suggested that some cases of cerebral paralysis in children were the counterpart of the spinal lesion in acute poliomyelitis. It is in this latter sense that the term polioencephalitis is used in this article; and although it has been thought well, for convenience of description, to separate it from poliomyelitis, the two conditions are in every way comparable, the difference in the symptoms being solely due to the different portions of the nervous system affected. Wickman suggested the name "Heine-Medin'sche Krankheit" to include cases of acute poliomyelitis and allied conditions; but it is undesirable to employ a name which does not convey some indication of the nature of the disease. Krause has suggested the name "acute epidemic paralysis." Medin, who certainly had the widest conception of the malady, included cases in which the cranial nerves were affected, cases with cerebral symptoms, cases in which acute ataxia was the prominent symptom, and cases which might be regarded as acute polyneuritis. He also shewed that cases of this nature arose during epidemics of acute poliomyelitis, and thus furnished proof of Strümpell's teaching. Since that time much has been written on the subject, as is shewn by the bibliographies given by Oppenheim and Cassirer, by Wickman, by Farquhar Buzzard, and by Chartier; and the occurrence of poliomyelitis and polioencephalitis in the same individual has been established.

Etiology.—Since polioencephalitis and poliomyelitis are closely allied, their etiological factors should be similar. Polioencephalitis, like poliomyelitis, is commoner in children than in adults. The disease is more apt to occur during the summer than in the winter months, and may be said to be epidemic in London during August. Various epidemics of poliomyelitis have been recorded: in Stockholm (May to November 1887) by Medin; in America (June to August 1894) by Caverly and Macphail; in Stockholm (June to August 1895) by Medin; in Norway (1899) by Leegaard; in America (1899) by Mackenzie, and (1907) by Sachs and Lovett (*vide* also Vol. VII. p. 625); in 1908 at Upminster (Treves); in

Vienna (1908) by Zappert; in Norway (1905 and 1906) by Geirsvold and by Harbitz and Scheel; in Sweden (in 1905 and again in 1906) by Wickman; in Australasia (1907) by Stephens; and in Westphalia by Krause. In the cases recorded by Wickman and Stephens the arms, legs, and trunk were generally affected, but in a small proportion of the total number there were cerebral and cerebellar symptoms, either with or without spinal manifestations. In the epidemic described by Wickman in 1905 the proportion of cerebral to spinal cases was about 1 to 10. In an analysis of 171 cases of polioencephalo-myelitis admitted to the Children's Hospital, Dr. Bremer found that cerebral and cerebellar symptoms were present in 50 cases, 11 of which had both spinal and cerebral symptoms, and that 121 had spinal symptoms only. Several members in a house may be attacked (Wickman, W. Pasteur), and Wickman quotes one instance in which six persons were affected in the same house. From the collected records of thirty-five epidemics prior to 1907, Holt and Bartlett state that in forty instances more than one member of a family have been affected. Wickman proved that the disease is carried by direct contact, and that it is transmitted along the great thoroughfares, roads or railways.

Bacteriology.—The nature of the infection is doubtful. Lumbar puncture performed in numerous cases has led to the description of several distinct micro-organisms; Concetti performed lumbar puncture in 10 cases of poliomyelitis and 3 cases of polioencephalitis and examined the fluid in 9. Bacteria could only be found in the first week of the disease; 6 gave a negative result, 2 shewed the diplococcus of Talamon and Fränkel, and 1 Weichselbaum's meningococcus. Looft and Dethloff found an organism which resembled the meningococcus. Geirsvold examined the cerebrospinal fluid, which was clear or slightly opalescent, from 16 cases; in 12 a micrococcus grew, in 3 others the fluid was sterile, but the organism could be seen on microscopical examination, and the remaining case was negative. Geirsvold, who believes this organism to be the cause of the disease, found that it grows well on the ordinary media; in bouillon a white sediment appears after a few days; on agar fine grey colonies form after two to three days, which after a few days tend to become white; on glycerin-agar, ascitic- and blood-agar the growth is good; but the organism does not grow well on blood-serum. On gelatin, milk, and potato the growth is good. The organism is Gram-positive. Intravenous injection into white mice, rabbits, and pigeons is followed by paralysis. In a series of cases Wickman failed to demonstrate this organism; and the bacteriological examination was altogether negative. Drs. Pasteur, Foulerton, and MacCormac, by inoculating rabbits and mice with the cerebrospinal fluid from a case of poliomyelitis, produced paralysis. They recovered the organism from the rabbits, but they entirely failed to prove that the paralysis was due to any spinal lesion. Dr. J. G. Forbes has examined the cerebrospinal fluid from cases of poliomyelitis and encephalitis under my care, but nearly always with negative results. On one occasion a streptococcus was found in pure growth, and this was injected by Dr.

MacFadyen into the subdural space of a rabbit. The rabbit did not manifest any acute symptoms, but gradually wasted, and after four weeks was killed. No lesion could be found in the nervous system. Potpeschnigg in 14 cases has found a Gram-positive coccus similar to that described by Geirsvold. During 1909 Krause and Meinicke produced paralysis in rabbits by inoculation of the subdural space with material obtained from cases of poliomyelitis. In two rabbits the symptoms appeared acutely eleven days after injection. Landsteiner and Popper reported two successful inoculations of monkeys; the injections were made into the peritoneal cavity. One monkey became paralysed on the sixth day and died on the eighth day after inoculation, the other was killed on the nineteenth day, having been paralysed for two, and possibly for seven, days. Lesions of the cord similar to those found in man in cases of poliomyelitis were present in both. The close resemblance of the spinal lesion to that found in rabies was commented on. Flexner and Lewis have by intracranial, intraperitoneal, intravascular, intraneural, and subcutaneous inoculation carried the virus of poliomyelitis through a series of monkeys. The spinal cords of these monkeys are stated to have shewn, without exception, lesions similar to those of poliomyelitis in man. No organisms could be demonstrated either on culture or on examination; but the clear filtrate mixed with rabbit serum bouillon and incubated for two days became turbid. A monkey inoculated with this fluid developed paralysis on the thirteenth day. Even when the virus was glycerinated or filtered through a Berkefeld filter it still produced the disease when injected into monkeys. Leiner and Wiesner in Austria, Römer in Germany, and Landsteiner and Levaditi in Paris, have produced the same results. The latter found that the incubation-period was in the chimpanzee six days, and in the macaque monkey five days; they also found that the incubation-period was prolonged when the virus was submitted to filtration. Flexner and Lewis, by taking the naso-pharyngeal mucous membrane of a recently paralysed monkey, and after pounding it up and passing it through a Berkefeld filter and injecting the resulting fluid into the brain of monkeys, produced typical poliomyelitis. Römer and Joseph shewed that the virus will withstand the action of glycerin (50 per cent) for as long a period as two months. Levaditi attempted to cultivate and stain the organism, and has described an extremely small organism stained pale pink by fuchsin and pale blue by the Giemsa stain.

The occurrence of cerebral symptoms during the course of the acute specific fevers, scarlet fever, diphtheria, and more especially measles, is recognised. But although these diseases may play an important part as disposing factors, it has not yet been shewn that they are the cause of the affection. Influenza stands in a somewhat different relation, for influenzal meningitis undoubtedly exists, and may give rise to symptoms very similar to those of encephalitis (*vide* p. 187).

In conclusion, although the direct cause of polioencephalitis, like that of poliomyelitis, is still unknown, all the recent experimental evidence points to a virus closely resembling that of rabies. The data on which this is based are: that the disease is transmissible from monkey to monkey; that it is capable of passing through the finest filter; that it will withstand the action of glycerin, and that it is not visible under the highest powers of the microscope. This similarity to rabies has been suggested by more than one writer on pathological grounds alone. Dr. F. Buzzard believes that polioencephalitis and poliomyelitis constitute an acute specific fever, and that immunity follows, so that second attacks are almost unknown.

Morbid Anatomy—*Macroscopical Examination*—Acute Stage.—The external surface of the brain may be normal; on the other hand, it may be flattened and pale, or the superficial vessels may be engorged. On section the cerebral substance is seen to contain numerous small haemorrhagic foci, mainly in the subcortical region; they may be confined to a single area, or may be widely distributed. A similar condition may affect the basal ganglia, the pons, and medulla, as is shewn in Oppenheim and Cassirer's illustrations. The lesion, however, may be quite local and pick out a single nucleus in the medulla, as in a case in which I found an area of engorgement in the region of the seventh cranial nucleus on the right side (1).

When the original lesion has been severe and extensive, an area of softening may be left, as in the case recorded by Drs. White and Worthington.

Late Stage.—In the late stage there may be a wasted condition of the cerebral cortex, with an underlying cavity filled with fluid, or a sclerotic condition may be present. In the cerebrum, porencephaly probably represents the late stage of an encephalitis, in the cerebellum a sclerosis.

The various stages of the condition have been studied far more extensively in the cord than in the brain or cerebellum. In fact, the pathological evidence with regard to the cerebellum is strikingly deficient. In the case recorded by Clapton the cerebellum was less than a third of its normal weight, and there was almost complete absence of the normal nerve substance of the cortex and a great increase of neuroglia. Dr. Gordon Holmes has reviewed the evidence on this point.

Microscopical Appearance.—The most marked change is the perivascular exudation around the sheath of the vessels. Some authors have described a patchy leptomeningitis. The vessels are engorged, but cannot be proved, in the earlier stages, to be thrombosed. There is some difference of opinion as to the nature of the cells around the vessels. The large majority may be regarded as small lymphocytes, characterised by a deeply staining nucleus and a small ring of cytoplasm. Polymorphonuclear neutrophils are absent. Plasma-cells and large mononuclears are present in small numbers only. Other cells with irregular nuclei represent slight proliferation of the adventitia of the vessels. Apart from the vessels, these lymphocytic cells invade the cerebral tissue, and multiple haemorrhages can be found throughout the affected area. Drs. White and Worthington state that the profound cellular infiltration is determined by a great proliferation of neuroglia, with a varying, and in parts an insignificant, amount of lymphocytic exudation; and they point out that by suitable staining a large number of the cells in the perivascular lymphatics are undoubtedly glial cells, and that plasma-cells are not present. The changes in the nerve-cells are in most cases slight, except in the immediate region of the lesion, where their destruction has been more or less complete.

Somewhat divergent views are held as to the path of the infection; Wickman suggests that it is lymphogenous; Dr. F. Buzzard compares the lesion to that found in rabies, and believes that the infection is haematogenous. Evidence on this point is still wanting.

Symptomatology.—The *incubation-period*, as judged by clinical evidence, is probably short, apparently from one to four days, but, according to Stephens, the extremes are three and sixteen days. From experimental data the incubation-period appears to be from five to six days (*vide* p. 243).

The symptoms may be described under two heads: first, those due to the *general* infection, and, secondly, those due to the *local lesion.*

General Symptoms.—The onset of the disease is usually sudden and may be attended by convulsions, in some cases severe and prolonged, in others transient. The temperature, at first slightly raised, quickly becomes normal, and the disease runs an afebrile course. Headache and vomiting may be present at the onset, but do not persist; and during the first few days there may be drowsiness and irritability. Some rigidity of the neck muscles, with pain on movement, is not uncommon. The onset may be preceded by diarrhoea or sore throat. A skin eruption has been described.

The local symptoms may be divided into three main groups, which will be designated as polioencephalitis superior, polioencephalitis inferior, and polioencephalitis cerebelli. The first category includes cases with evidence of affection of the cerebral hemispheres, either the frontal, the motor, or the occipital cortex; the second, cases with evidence of implication of the nuclei of the cranial nerves, or of the basal ganglia; and the third, cases with evidence of affection of the cerebellum. No hard and fast line, however, can be drawn between these groups, for some cases present symptoms belonging to two or even to all three categories.

Polioencephalitis superior may affect various portions of the cerebral cortex, and give rise to various symptoms according to the portion affected. The most common symptom is hemiplegia, which may be associated with aphasia. The onset may be sudden and attended by convulsions, or may be gradual with a slow increase of symptoms. The head and eyes may deviate to the right or left, according to the position of the lesion. The hemiplegia may be transient, or may persist and be followed by contraction and athetosis. Polioencephalitis is by no means a common cause of infantile hemiplegia; out of 100 consecutive cases of hemiplegia in children, collected at the Children's Hospital, Great Ormond Street, and at the National Hospital, the hemiplegia could not be assigned, on clinical grounds alone, to polioencephalitis in more than 10 per cent. The hemiplegia, which is accompanied by the usual alteration in the reflexes, may in some cases be bilateral and give rise in its later stages to the clinical picture of a diplegia.

Although the motor cortex appears to be most frequently attacked, there are clinical, but as yet no pathological, data to shew that the frontal region may be affected and give rise to acute mental symptoms, the disease being ushered in by a series of fits and loss of consciousness, fever, and vomiting. On recovering consciousness, the child may scream, take no notice of its surroundings, and fail to recognise parents or nurse. The child becomes dirty in his habits, destructive, and cannot speak intelligibly. There may not be any evidence of paralysis of the limbs or of the cranial nerves. Recovery may follow, or the patient may become permanently idiotic or defective.

In cases in which the occipital cortex appears to be affected, the symptoms start with convulsions and a period of unconsciousness, and when consciousness returns the patient is completely blind, the pupils being dilated but still reacting to light in whichever direction it is thrown on the retina. No other paralytic phenomena may be present. The intelligence may be unaffected. Recovery may gradually take place, so that in three to six weeks vision is perfectly restored, or permanent blindness may result. The occurrence of this condition is based on clinical, not on pathological, evidence, and may therefore be called in question, but with a fuller recognition of the clinical features the pathological evidence should be forthcoming.

Polioencephalitis inferior may attack any of the nuclei of the cranial nerves or the basal ganglia. The affection of the oculomotor nuclei gives rise to ocular paralyses, which may be complete or partial, or may affect one side only. Ptosis, loss of power in the movements of the eye, dilatation of the pupil, and loss of reaction to light and accommodation may be present. Loss of vision may occur concomitantly with the ocular paralysis, and is presumably due to the interruption of the visual fibres in the region of the external geniculate bodies. Complete recovery of both vision and ocular paralysis may occur, or the ocular paralysis may clear up, leaving the patient completely blind. Retinal haemorrhage has been noted, but the optic disc usually appears normal. A lesion in the sixth nucleus of one side might give rise to a loss of conjugate movements to the side of the lesion, or might cause nystagmus on attempted movement of the eyes to that side. Weakness of the motor portion of the fifth cranial nerve has been observed, commonly in association with other lesions.

Facial paralysis has frequently been noted in combination with other lesions, and also alone; when combined with other lesions the diagnosis may be accepted, but cases with facial paralysis alone are open to criticism; for facial paralysis is commonly due to disease of the nerve itself, and it is difficult in children to distinguish a peripheral from a nuclear lesion of the seventh nerve. That a facial paralysis may be produced by a nuclear lesion is proved by a case under my observation, in which the seventh nucleus was practically destroyed by a vascular lesion, whilst the nucleus of the neighbouring sixth nerve was unaffected (1). Facial paralysis may be accompanied by some weakness of the external rectus, an associated paralysis dependent on the proximity of the sixth and seventh cranial nuclei. Facial paralysis of one side may be associated with hemiplegic weakness of the opposite side of the body. Oppenheim and Cassirer quote the case of a girl, aged twelve, in whom, in addition to a complete paralysis of the left side of the face, there was paralysis of the right side of the body. There was also some inco-ordination of the arms, more especially the left. The lesion was undoubtedly in the pons; complete recovery eventually took place.

Bilateral nerve-deafness may occur suddenly, without any evidence of affection of the inner or middle ear, and in the absence of any implication of the other cranial nerves. It is difficult to prove that these cases are due to a polioencephalitis, and, since such deafness is known to be the result of certain forms of meningitis, it is easy to assume that such has been the lesion. The pathological proof that such cases may be due to a polioencephalitis is wanting.

With regard to affection of the bulbar nuclei of the ninth, tenth, eleventh, and twelfth cranial nerves, cases are recorded by Leyden, Elter, Medin, Wickman, and others, in which there has been weakness of the palate, difficulty in swallowing, hoarseness of the voice, and paralysis and wasting of the tongue. These symptoms may occur in association with spinal lesions of the type of a poliomyelitis. In a case already referred to, in which there was facial paralysis on the right side, there was also weakness of the palate on the same side (1).

Polioencephalitis Cerebelli.—The most striking symptom of this group is the acute onset of ataxia of a cerebellar type. The onset may be preceded by headache, vomiting, loss of consciousness, and convulsions. After recovery of consciousness, ataxia may be first noticed; the ataxia may be unilateral or bilateral, and affect both the arms and the legs, but the latter to a greater extent than the former. The ataxia often escapes notice till the child is "sat up in bed." There is no actual loss of power in the limbs, all movements being capable of being performed in the lying position. The patient is unable to stand, or even to sit up, without support. The intelligence is good, but there is often a marked affection of the articulation, so that the child speaks in a slow jerky manner. Nystagmus may be present.

Medin observed a case of acute ataxia in association with poliomyelitis, and one of the cases in the family recorded by Dr. Pasteur had ataxia, from which she rapidly recovered. Wickman, who observed a similar case in the epidemic of 1905, considers that the ataxia is not always due to the same lesion, and that in different cases it may be caused by a lesion of the cerebellum, or of the mid-brain, a view with which most writers would agree. Dr. R. H. Miller (21) described a case of poliomyelitis, in which nystagmus appeared, and found a focal lesion in the cerebellum as well as other lesions.

These cases run a very variable course; some recover rapidly, so that in a few days or weeks the patient is again perfectly well, others eventually make a complete recovery, although it may take years, as in the case recorded by Dr. F. Taylor. In others, again, recovery never occurs; in these cases there is usually evidence of damage to the motor neurons, and it has been shewn experimentally that although after injury to the cerebellum alone the ataxia produced thereby rapidly clears up—if the motor path be damaged at the same time, recovery does not take place.

Diagnosis.—The diagnosis of polioencephalitis is not easy to prove, first, because the virus producing the condition is unknown, and, secondly, because, as a large majority of the cases recover, pathological investigation is seldom possible. When cases exhibiting cerebral symptoms occur in association with a poliomyelitis, either in the same individual, or in the same family, it is justifiable to assume that the lesion is due to the same cause.

All acute cerebral lesions in children should not be attributed to polioencephalitis, for an inflammatory condition of the meninges, or a vascular lesion, may account for these symptoms. Meningitis should be excluded by examination of the cerebrospinal fluid, for meningitis nearly always produces some change in the cerebrospinal fluid. The cytological and bacteriological examination of the cerebrospinal fluid furnishes most valuable evidence. The fluid obtained from cases of polioencephalitis is almost always clear, flows out under slight pressure, and on cytological examination is perfectly normal or contains a slight excess of lymphocytes. No organism can be found either on culture or microscopical examination. Care must be taken to exclude abscess in the brain. The condition described by Wernicke, under the title polioencephalitis superior, due to a vascular lesion in association with alcohol or arteriosclerosis, should not be included in the group of polioencephalitis.

When, however, a previously healthy child, or one convalescing from an acute specific fever, is taken acutely ill with cerebral symptoms and subsequent local paralysis, the possibility of polioencephalitis should be considered. The association of the cerebral lesion with symptoms suggesting poliomyelitis renders the diagnosis more probable, and this is still further strengthened if the attack occurs when poliomyelitis is prevalent. The onset of cerebral symptoms in a case of poliomyelitis strongly suggests polioencephalitis. It should also be noted that although children have no monopoly of the disease, they undoubtedly suffer from it much more frequently than adults.

The disease may simulate diphtheritic paralysis very closely, not only in the bulbar symptoms, but also in the paralysis of the diaphragm. The more local character of the affection, the less complete recovery, and the history of a sudden onset would all favour the diagnosis of polioencephalitis.

In old and long-standing cases the question of the presence of an intracranial tumour or of hydrocephalus may arise. The stationary and retrogressive nature of the symptoms would strongly favour the diagnosis of polioencephalitis, but it must be borne in mind that tuberculous tumours in children often become quiescent.

Again, the rare condition in which there is congenital absence of one or more of the nuclei of the cranial nerves must be remembered.

In cases with cerebellar symptoms the differential diagnosis from Friedreich's ataxia and allied conditions requires careful consideration. The gradual onset, the progressive nature of the symptoms, and a critical investigation of all the symptoms and deformities which commonly accompany Friedreich's ataxia will in most instances make a diagnosis possible. In other cases, however, the differential diagnosis is often difficult, in some cases impossible.

Prognosis.—The prognosis with regard to life depends on the situation and extent of the lesion. The tendency to recovery is greater in polioencephalitis than in other acute cerebral lesions. Probably a large proportion of those cases in which the cerebral hemispheres are affected recover, whilst many of those in which the bulb is implicated must quickly die. Many of the cases with ocular paralysis recover rapidly and completely, others are left with permanent muscular weakness, and some with loss of sight. Mental defects, hemiplegia, ocular paralysis, facial paralysis, weakness and wasting of the tongue, permanent ataxia, are some of the after-effects of the disease. But there is always a tendency to improvement and recovery, more especially in cases with ataxia, in which complete recovery may take place after the symptoms have been present for two or more years.

Treatment.—In the acute stage symptoms should be met by the ordinary remedies, rest and quietude being enjoined. Nasal feeding is important when there is weakness of the palate and muscles of deglutition.

In the later stages hemiplegia should be treated by massage and movements, ataxia by educative movements, and the local paralysis by electricity. As the tendency of the disease is towards recovery, treatment should be persevered with.

FREDERICK E. BATTEN.

REFERENCES

1. BATTEN, F. E. *Trans. Path. Soc.*, London, 1903, liv. 359.—2. *Idem. Trans. Med. Soc.*, London, 1905, xxviii. 116.—3. *Idem. Lancet*, London, 1902, ii. 1677.—4. *Idem. Brain*, London, 1905, xxviii. 484.—5. BREMER. *Lancet*, London, 1910, i. 421.—6. BUZZARD, E. F. *Brain*, London, 1907, xxx. 1 (References).—7. CHARTIER. *L'Encéphalite aiguë*, Paris, 1907 (References).—8. CONCETTI. *Internat. Med. Congress*, 1900.—9. FLEXNER and LEWIS. *Journ. Amer. Med. Assoc.*, Chicago, 1909, liii. 1639, 1913, 2095; 1910, liv. 45, 535, 1140, 1780; *Journ. Exper. Med.*, N.Y., 1910, xii. 227 (References).—10. FORBES. *Quart. Journ. Med.*, Oxford, 1908, i. 109.—11. GEIRSVOLD. *Norsk Mag. f. Laegevidensk.*, Kristiania, 1905, 5. R., iii. 1280.—12. HARBITZ und SCHEEL. *Deutsche med. Wchnschr.*, 1907, xxxiii. 1992.—13. HOLMES. *Brain*, London, 1907, xxx. 545.—14. KRAUSE und MEINICKE. *Deutsche med. Wchnschr.*, 1909, xxxv. 1825.—15. LANDSTEINER et LEVADITI. *Compt. rend. Soc. de biol.*, Paris,

1909, lvii. 592, 787.—16. Landsteiner und Popper. *Ztschr. f. Immunitätsforsch.* (*Orig.*), 1909, ii. 377.—17. Leiner und Wiesner. *Wien. klin. Wchnschr.*, 1909, lix. 1698.—18. Levaditi. *Presse méd.*, Paris, 1910, xviii. 44.—19. Looft and Dethloff. *Medicinsk. Rev.*, 1901, xviii. 321.—20. Medin. *X. internat. med. Kongress*, Berlin, 1890.—21. Miller. *Brain*, London, 1907, xxx. 117.—22. *Idem. St. Mary's Hosp. Gaz.*, 1907, xiii. 118.—23. Oppenheim und Cassirer. "Die Encephalitis," *Spec. Path. u. Ther.* (Nothnagel), 1907 (References).—24. Pasteur. W. *Trans. Clin. Soc.*, 1897, xxx. 143.—25. Pasteur, Foulerton, and MacCormac. *Lancet*, London, 1908, i. 484.—26. Potpeschnigg. *Wien. klin. Wchnschr.*, 1909, lix. 1334.—27. Prickett and Batten. *Trans. Clin. Soc.*, London, 1900, xxxiii. 157.—28. Römer. *München. med. Wchnschr.*, 1909, lvi. 2505.—29. Römer und Joseph. *München. med. Wchnschr.*, 1910, lvii. 347.—30. Sachs and Lovett. *Journ. Nerv. and Ment. Dis.*, 1909, xxxvi. 619, 681.—31. Schwarz. *Rev. Neurol. and Psychiat.*, 1909, vii. 373.—32. Stephens. *Intercol. Med. Journal of Australasia*, 1908, xiii. 573.—33. *Idem. Rev. Neurol. and Psychiat.*, 1909, vii. 196.—34. Strümpell. *Jahrb. f. Kinderh.*, 1884, n.F. xxii. 173.—35. Taylor, F. *Lancet*, London, 1904, ii. 1416.—36. Treves, W. W. *Brain*, London, 1909, xxxii. 285.—37. White and Worthington. *Arch. Path. Inst. London Hosp.*, 1906, i. 27.—38. Wickman. *Heine-Medin'schen Krankheit*, Berlin, 1907 (References).—39. Wilbrand und Saenger. *Die Neurologie des Auges*, Bd. i. S. 264.—40. Zappert. *Wien. med. Wchnschr.*, 1908, lviii. 2566 ; 1909, lix. 2683.

F. E. B.

ABSCESS OF THE BRAIN

By Byrom Bramwell, M.D., F.R.C.P.Ed.

Introductory Remarks.—An abscess of the brain is very rarely, if ever, primary and idiopathic ; in the great majority of cases it is secondary to one or other of the following conditions : (i.) disease of the skull bones or scalp tissues (ear disease, nose disease, and the like) ; (ii.) traumatic injury to the head ; and (iii.) infection carried from some distant organ, especially from the lung.

An abscess of the brain may be acute, subacute, or chronic. In some chronic cases, in which there are no urgent symptoms, very grave symptoms may suddenly arise in consequence of the rapid supervention of acute meningitis or acute cerebritis.

Morbid Anatomy and Etiology.—The morbid appearances met with after death vary considerably in different cases. In the majority of cases the abscess is in one or other of the cerebral hemispheres, usually in the temporo-sphenoidal lobe ; much less frequently in the frontal, parietal, or occipital lobes. In a considerable proportion of cases the abscess is situated in the lateral lobe of the cerebellum. In rare instances the abscess is in the pons Varolii or medulla oblongata. Statistics seem to shew that the abscess is more frequently in the right than in the left hemisphere. The cause of this (if it be true, as it seems to be) is unknown.

In the great majority of cases the abscess is due to a local cause, either disease of the cranial bones (more especially ear disease) or head injury ; further, in the cases in which the abscess is due to head injury, the immediate cause of the abscess is usually bone disease (suppuration or necrosis) which has resulted from the injury.

In most cases there is one abscess only; but sometimes there are several abscesses in different parts of the brain, more particularly in pyaemic cases in which the brain abscess is due to a distant source of infection. In the great majority of cases in which a brain abscess is due to ear disease there is only one abscess, usually in the temporo-sphenoidal lobe or lateral lobe of the cerebellum; quite exceptionally there may be an abscess in both situations.

The size of the abscess varies. In some cases it is merely a minute point of suppuration; in others, the greater part, or the whole, of one lobe of the brain is converted into a huge pus-containing cavity.

The contents of the abscess generally consist of yellow or yellowish-green pus; in very acute cases the abscess cavity may contain a reddish grumous material, partly composed of broken-down brain tissue. It is said that in very chronic cases the pus is sometimes thin and watery. In some cases the pus is very fetid. In a case which came under my observation a few years ago, although the contents of the abscess were extremely foul-smelling, no putrefactive organisms could be detected, either with the microscope or in cultivations.

In some cases the abscess has no proper wall; the pus is simply contained in an irregular cavity, the walls of which consist of the inflamed and broken-down brain tissue; in other cases there is a distinct lining membrane or capsule, the thickness and density of which are very variable. A thick, dense lining-membrane shews that the abscess has been in existence for some time. This, as Gull and Fagge pointed out, may be of medico-legal importance; in those cases in which a tough, dense membrane is present, it may with confidence be assumed that the abscess has been in existence for three or four months at least, and probably for much longer. Chronic cerebral abscesses with tough fibrous capsules are usually the result of ear disease; the abscesses which follow injury usually run an acute course, and are rarely surrounded by a distinct fibrous capsule.

In some cases the brain tissue around the abscess is softened and inflamed; in others there is purulent meningitis, for the abscess not infrequently proves fatal by bursting into the lateral ventricle, or (but this is less common) upon the surface of the brain. In other cases in which the abscess is due to ear disease there is sinus phlebitis.

We have seen that there are three great causes of abscess: namely, (*a*) local disease of the bones of the skull; (*b*) injury; and (*c*) infection from a distant organ (the lung, general pyaemia, etc.). Cases due to injury usually come under the care of the surgeon. Suppurative disease of the ear is by far the most frequent cause in the cases of cerebral abscess which come under the notice of the physician. In the great majority of cases the ear disease has been in existence for some time; it is usually a chronic otorrhoea which dates from an attack of measles, scarlet fever, etc. The tympanum is usually perforated. In some cases the ear disease has become purulent shortly before the abscess and acute cerebral symptoms are manifested. In other cases the cerebral symptoms follow the arrest of an ear discharge which was previously profuse; in some chronic cases of this kind the cerebral abscess is in direct communication with the cavity of the tympanum. In rare instances the tympanic cavity is in a state of suppuration, but there is no perforation of the drum. In other cases the mastoid cells or external ear are the seat of the disease.

In those cases in which the abscess is due to ear disease it is always, so far as I know, in the cerebral hemisphere on the side of the ear lesion; and in the great majority of cases it is in the temporo-sphenoidal lobe or the lateral lobe of the cerebellum, rarely in the frontal, occipital, or parietal lobes.

The exact manner in which ear disease causes abscess of the brain varies in different cases; in some there is direct extension; in others the inflammation, or the infection, is carried from the ear to the brain either by the small communicating veins which pass between the ear on the one hand, and the petrosal or lateral sinuses and the brain tissue on the other, or along the lymphatic channels which surround the blood-vessels. In those cases of abscess of the temporo-sphenoidal lobe in which the abscess lies immediately above the tympanum, in which the membranes are adherent to the upper wall of the tympanum, and in which a direct channel of communication passes between the cavity of the abscess and the cavity of the tympanum, the disease in the tympanum seems to produce first a local meningitis (adhesion of the membranes of the temporo-sphenoidal lobe to the upper surface of the tympanum), and then, by direct extension, probably along the lymphatic channels surrounding the blood-vessels, an abscess in the brain tissue. The venous and lymphatic connexions between the ear and the brain are of importance in the production of brain abscesses. Small veins pass from the tympanum on the one hand and from the temporo-sphenoidal lobe on the other into the superior petrosal sinus; there is therefore a distinct venous connexion (which may serve as a channel of infection) between the tympanum and the temporo-sphenoidal lobe. Again, small veins pass from the mastoid region on the one hand, and from the lateral lobe of the cerebellum on the other, into the lateral sinus; there is therefore a distinct venous connexion (which may serve as a channel of communication) between the mastoid cells and the lateral lobe of the cerebellum. But in those cases in which there is no sinus phlebitis (that is, in which the blood is flowing freely through the superior petrosal and lateral sinuses) it is difficult to see how infective particles can make their way backwards against the blood-current into the temporo-sphenoidal lobe or the cerebellum. In some cases it is difficult to explain the exact channel of infection and the method of propagation of the infective particles; disease in the tympanum seems apt to produce an abscess in the temporo-sphenoidal lobe, disease in the external ear or mastoid an abscess in the lateral lobe of the cerebellum.

A chronic otorrhoea, because of its liability to produce cerebral abscess, sinus phlebitis, and purulent meningitis, must always be regarded as a possible source of danger. Assurance companies recognise this: they refuse to insure persons affected with otorrhoea at the ordinary rates; they either decline them altogether or "load" them, the amount of the "extra" depending upon the special peculiarities (the exact character of the ear disease—the presence or absence of otorrhoea, the character of the discharge, the size of the perforation in the tympanum, etc.) in each individual case. (See also art. "Intracranial and Intravenous Infections complicating Ear Disease," Vol. IV. Part II. p. 475.)

In those cases in which a cerebral abscess is the result of nose disease the abscess is usually situated in the frontal lobe on the same side as that on which the nasal disease is most marked. In exceptional cases the abscess is situated in some other part of the cerebrum (the parietal, occipital, or temporo-sphenoidal lobes), very rarely indeed in the cerebellum.

A collection of pus may be situated on the surface of the brain between the cortex and the membranes, hemmed in by inflammatory adhesions. In one sense such a condition is an intracranial abscess, but it is not a true abscess of the brain; it is rather the result of a purulent meningitis. A circumscribed collection of pus on the surface, to which the name extra-cerebral abscess may be appropriately applied, is often associated with local disease of the bone, and is frequently the result of injury; punctured wounds of the brain are apt to be followed by cerebritis, and it may be by a cerebral abscess. In traumatic cases, in which there is no wound, local suppuration, or necrosis, the abscess is usually beneath the seat of the original injury. In pyaemic cases the abscesses may be either in the cerebrum, in the cerebellum, or in both; rarely in the cerebellum alone.

Abscess of the brain may occur at any age. The disease is very rare during the first year of life, and after sixty; the majority of cases occur between ten and thirty; cases are not very uncommon, however, between thirty and forty-five.

Abscess of the brain is more common in males than in females, probably because males suffer so much more frequently from head injury than females; if the traumatic cases are excluded there is probably no great difference in the relative frequency with which the disease is met with in the two sexes, though some writers state that even then the majority of cases occur in males.

Abscesses of the brain are much more common amongst the labouring than the upper ranks of society, doubtless because amongst the lower orders a chronic otorrhoea is often allowed to go untreated, and because in certain classes of working people head injuries are more common than in well-to-do people.

Clinical History.—The symptoms which are met with in different cases of cerebral abscess are very variable. These differences depend upon a number of different conditions, namely:—

(1) The character of the abscess.—(*a*) Whether it is acute or chronic; (*b*) whether it is encapsulated or not; and (*c*) if encapsulated, whether there is a channel of communication between the cavity of the abscess and the exterior of the skull, say the tympanum, or whether there is no

such communication. An encapsulated abscess which does not communicate externally, and around which there are no acute inflammatory changes, may, from a practical point of view, be regarded as a cerebral or cerebellar cyst; in such cases the symptoms may closely resemble those of an intracranial tumour. In cases of this kind there may be no marked symptoms, for an encapsulated abscess, so long as there are no acute inflammatory changes in the brain tissue or cerebral membranes surrounding it, may be a very stationary lesion. Cerebral symptoms are usually absent or slight when a free channel of communication exists between the abscess cavity and the exterior of the skull; but in cases of this kind, the arrest of the discharge—the blocking of the exit—is apt to be associated with urgent symptoms. (2) The position of the abscess. (3) The presence or absence of acute cerebritis, meningitis, or sinus phlebitis. (4) The nature of the primary condition with which the abscess is associated, such as ear disease and general pyaemia.

Further, it must be remembered that the symptoms vary at different stages of the disease. In the earlier stages, headache, vomiting, and other symptoms indicative of cerebral irritation are prominent; in the later stages paralysis and coma are developed.

From these statements it will be obvious that it is very difficult or impossible to give a single description which will apply to all cases of abscess of the brain. Certain well-marked clinical types may, however, be constructed.

1. *Acute Cases with very Definite and Distinct Symptoms.*—In these cases, more especially when the abscess is large and of rapid growth, the symptoms are usually very urgent and severe.

2. *Chronic Cases.*—In some cases of this kind, in which the abscess is encapsulated, there are no symptoms, or only slight symptoms; in other cases in which an abscess of the temporo-sphenoidal lobe is in direct communication with the external ear (cases of cerebral otorrhoea) there may be no symptoms so long as the sinus remains patent), but urgent symptoms may arise if the exit for the pus becomes blocked. Consequently, in cases of chronic otorrhoea with a profuse purulent discharge, in which there is a suspicion that the otorrhoea is cerebral, great care is necessary in the use of remedies, such as powerful astringent injections, which are likely to produce sudden arrest of the discharge and blocking of the communication between the abscess cavity in the temporo-sphenoidal lobe and the cavity of the tympanum.

3. *Cases in which the Symptoms of Abscess, such as Headache and Vomiting, are overshadowed by those of the Associated Disease, such as General Pyaemia and Infective Endocarditis.*—In these cases the abscess is usually in one of the "silent" areas of the brain. In some cases of this kind there are no doubt local or focal symptoms, such as hemianopsia, which must be looked for.

Summary of the more important General Symptoms which are met with in Cerebral Abscess.—The nature and severity of the symptoms which are associated with the formation of a cerebral abscess are very variable. In some cases very severe symptoms rapidly appear; in other cases the onset is insidious. Speaking generally, the most frequent and characteristic symptoms are: Headache, sometimes intense, vomiting, giddiness, a slow pulse, delayed cerebration, drowsiness, apathy, and, after the initial onset, absence of pyrexia. Anorexia, a thickly coated tongue, a foul breath, constipation, and emaciation are usually present. In a certain proportion of cases there is optic neuritis. Delirium may occur in the later stages of the case, and especially when the cerebral abscess is associated with meningitis or sinus phlebitis. In most cases increasing drowsiness, apathy, and coma precede death. In many cases there is very rapid and marked emaciation. In cerebral abscess there is often a decided leucocytosis, which may be of considerable diagnostic significance.

Optic neuritis, though a frequent symptom, is often absent. In some cases it is double, in others unilateral. When unilateral it is probably most frequently present on the same side as the abscess. Its intensity varies in different cases, but, speaking generally, it is usually much less marked than in intracranial tumour. In those cases in which the abscess is in the cerebellum, the optic neuritis is, in my experience, more frequent and more intense than in cases in which the abscess is in the temporo-sphenoidal lobe. In very acute cases the short duration accounts, no doubt, for the absence of optic neuritis in some cases.

The absence of pyrexia is a very important point. Of course in some cases there is fever; but in most cases in which there is no meningitis and no sinus phlebitis, the temperature is usually, after the initial onset, either normal or subnormal. A cerebral abscess, in fact, seems to exert a depressing influence upon the temperature. Exceptions to this statement, however, are not infrequent; fever is usually present in the early stages of acute cases, and often in children. When the temperature is above the normal the rise may only be slight—two or three degrees. A high temperature suggests meningitis or sinus phlebitis. A high temperature, especially a fluctuating temperature associated with rigors, strongly suggests sinus phlebitis. In brain abscess due to ear disease there is often a history of rapid onset with earache, headache, vomiting, and fever; such symptoms may, of course, be due to the ear disease only.

A slow pulse, again, is a highly characteristic feature of cerebral abscess. In the later stages the pulse may become quick and irregular; and this is more particularly the case when the abscess has burst into the lateral ventricle, or when the abscess is complicated with sinus phlebitis. High temperature, a rapid pulse, and unconsciousness, if quickly or suddenly developed in the course of a brain abscess, suggest that the abscess has burst into or involved the lateral ventricle.

In the subacute and chronic cases of cerebral abscess in which the pus is gradually increasing in amount, the symptoms are practically the same as those of a rapidly growing intracranial tumour.

In some cases of chronic encapsulated abscess of the brain there is little or no disturbance of the intracranial pressure; and this is more especially seen when a free communication exists between the abscess cavity and the tympanum, that is, in cases with cerebral otorrhoea. Consequently, in cases of this kind, the general symptoms of an intracranial tumour—headache, vomiting, optic neuritis, generalised epileptiform convulsions, impairment of memory and intellect—are usually slight, and in rare cases altogether absent. But it must be remembered that in these cases meningitis or cerebritis may be rapidly set up around the abscess, and that when either of these events occurs, acute cerebral symptoms will of course be manifested.

In those cases in which the abscess is not firmly encapsulated, and in which it is steadily, though it may be slowly, increasing in size—and these cases are usually subacute rather than chronic—the symptoms (general and local) are practically the same as those of a rapidly increasing intracranial tumour.

In cases of cerebral abscess with cerebral otorrhoea, the stoppage of the discharge is apt, as has been previously stated, to be attended with the sudden appearance of headache, vomiting, and other symptoms (general and local), due either to a rapid increase of the intracranial pressure, to acute cerebritis or acute meningitis, or to all of these conditions in combination. In cases in which the acute symptoms depend merely upon the blockage of the channel for the pus, the headache and vomiting may rapidly subside, provided that the block be removed and the otorrhoea quickly re-established. If, however, acute cerebritis or meningitis be set up, the result in all probability will be fatal, unless indeed the life of the patient can be saved by surgical interference.

Summary of the Chief Local or Focal Symptoms which are met with in Cases of Cerebral Abscess.—The local or focal symptoms which are due to a brain abscess are practically the same as those which result from any other local lesion, such as a rapidly growing tumour. It is unnecessary to enter into details, for these symptoms are fully described in the article on cerebral localisation (p. 37 *et seq.*). In speaking of the diagnosis I will refer briefly to the more important localising symptoms which are apt to arise as a result of an abscess in the temporo-sphenoidal lobe and in the cerebellum. In the pyaemic cases, in which several abscesses may be present in different parts of the same brain, the local or focal symptoms may be multifarious.

Diagnosis.—The diagnosis of abscess of the brain is in some cases easy, in others very difficult or impossible; for symptoms identical with or very similar to those which are produced by a cerebral abscess may be due to acute cerebritis, uraemia, meningitis, sinus phlebitis, softening due to thrombosis, a tumour of the brain, or even local disease of the ear.

We have seen that a cerebral abscess very rarely occurs without some obvious cause. This is a most important point for the purpose of diagnosis. When cerebral symptoms suggestive of an abscess are present, the first step towards a definite diagnosis of abscess is to determine whether a cause of cerebral abscess be present or not. If no cause of cerebral abscess be found, the strong probability is that the symptoms are due to some other condition, such as acute cerebritis, uraemia, meningitis, thrombosis, or tumour. The absence of any apparent cause of abscess does not necessarily negative the presence of a cerebral abscess, for in a small percentage of cases of abscess of the brain there is no discoverable cause. In the absence of a definite cause, the presence of a cerebral abscess may be suspected; but a positive diagnosis of abscess should never be given. Conversely, if a definite cause of abscess, such as a septic otorrhoea, be present, the occurrence of head symptoms—such as headache, vomiting, optic neuritis—should always suggest the presence of a cerebral abscess, though the association of the two conditions—a definite cause for and symptoms suggesting a brain abscess—does not prove that an abscess is present. I have seen some cases in which cerebral symptoms, suggesting but not due to a cerebral abscess, developed in a patient suffering from chronic otorrhoea. In cases of this kind (chronic otorrhoea, in the course of which an acute disease, such as influenza, or a chronic condition, such as uraemia, develops and gives rise to headache and often symptoms suggesting brain abscess) the diagnosis is always difficult.

Now, since disease of the ear is by far the most frequent cause of cerebral abscess in the cases which come under the notice of the physician, we first make a careful inquiry into the condition of the ear. We ascertain if there is any otorrhoea. If there is an otorrhoea, we particularly determine whether it is a septic otorrhoea, and whether the perforation in the tympanic membrane is small or large; for a cerebral abscess is much more likely to result from a septic than from an aseptic otorrhoea, and in cases in which the opening in the drum-head is small than in cases in which the opening is large—in which, in other words, there is a free vent for the purulent collection in the tympanic cavity. If there is a history of a former otorrhoea, it is important to ascertain when the otorrhoea stopped, and if the arrest corresponded to the onset of the cerebral symptoms.

Even if there is no ear discharge, the external ear and the membrana tympani should always be most carefully examined, the condition of hearing tested, tinnitus and giddiness inquired for, and the presence of tenderness or swelling over the mastoid ascertained. The presence or a history of facial paralysis is also important, for in some cases of ear disease the facial nerve is implicated. As I have already stated, a brain abscess may develop as the result of ear disease, even when there is no perforation of the tympanic membrane. Mr. Ballance states:—"In childhood the tympanic membrane may appear normal, and yet the patient may be suffering from an intracranial inflammation resulting from temporal bone disease," and, according to Mr. Whitehead, "the most serious and dangerous complication of temporal bone suppuration may occur without the very slightest pain, tenderness, or swelling over the mastoid."

If there is no ear disease, present or past, the nose must next be carefully examined. If there is no nose disease, past or present, the condition of the orbit and the other parts of the skull and scalp must be examined; and the presence or absence of a history of head injury ascertained. Too much importance should not be attached to a history of former head injury, for patients and their friends are only too prone to attribute symptoms, which are due to disease, to slight injuries. When there is no definite history of a head injury, attention must next be directed to the condition of the distant organs, particularly of the heart and lungs; for infective endocarditis and septic lung disease are, as we have previously seen, occasional causes of cerebral abscess. It must, of course, be remembered that it is only in rare cases that a cerebral abscess is the result of lung disease. In the great majority of cases in which acute cerebral symptoms arise in the course of, and as the result of, lung diseases, especially phthisis, the cerebral lesion is tuberculous and not suppurative.

The differential diagnosis between cerebral or cerebellar abscess and cerebral meningitis is sometimes very difficult, or impossible; for the symptoms in the two conditions may be very similar; and further, both conditions may be due to the same cause (to ear disease, for instance); consequently, both conditions may be present in the same case at the same time. In the differential diagnosis of acute generalised meningitis and cerebral abscess, the condition of the temperature and of the cerebrospinal fluid is most important. In most cases of cerebral abscess the temperature is, after the onset, subnormal or normal; whereas in most cases of acute generalised meningitis the temperature is considerably raised. Decided fever, though it does not exclude abscess, is consequently in favour of meningitis; and conversely, a subnormal or normal temperature, though not conclusive, is strongly in favour of cerebral or cerebellar abscess. A case recently under my notice shews the unreliability of the temperature as a diagnostic guide. There were three acute abscesses in the brain, with suppurative meningitis at the base, and great increase of the intracranial pressure, though lumbar puncture shortly before death shewed an enormous leucocytosis; the temperature throughout the whole course of the case was subnormal, and the pulse never exceeded 84. A turbid condition of the cerebrospinal fluid, and the presence of numerous leucocytes, or of micro-organisms, in the cerebrospinal fluid are of great diagnostic value, and point to meningitis.

The absence of a definite cause of cerebral abscess (disease of ear, nose, and so forth) is strongly against abscess and in favour of meningitis. In children tuberculous disease of the ear may cause tuberculous meningitis; hence the importance of ascertaining the exact character of the ear disease in cases with cerebral symptoms. The presence of a tuberculous lesion, either in the lungs or elsewhere, is against abscess and strongly in favour of tuberculous meningitis.

In the differential diagnosis of cerebral abscess and tuberculous meningitis the presence or absence of leucocytosis is of great value. The presence of hemiplegia, of monoplegia, or of aphasic symptoms is in favour of abscess rather than of meningitis. This is especially the case when the paralytic or aphasic symptoms come on gradually and progressively, and when they are not merely temporary and epileptiform in character. Localised spasms, attacks of Jacksonian epilepsy and epileptiform paralysis or epileptiform aphasia are more common in tuberculous meningitis than in cerebral abscess. Retraction of the head and rigidity of the upper part of the spine also suggest meningitis; but they are not infrequent in cerebellar abscess. The course of severe generalised purulent meningitis is usually more rapid than that of a cerebral abscess; symptoms of irritation at the base or on the surface of the brain are generally more prominent, and the pyrexia is usually much more marked than in abscess. There are, however, exceptions to all of these statements.

It will thus be apparent that in many cases the diagnosis turns upon the presence or absence of a cause of cerebral abscess, upon the condition of the temperature, upon the presence or absence of leucocytosis, and upon the condition of the cerebrospinal fluid (lumbar puncture).

In some cases the diagnosis can only be made by operative inspection (trephining) or by watching the course of the case.

The Differential Diagnosis between Sinus Phlebitis and Cerebral or Cerebellar Abscess.—This is usually not difficult, provided that only one of the conditions (abscess or sinus phlebitis) be present. A high temperature, marked fluctuations in the temperature curve, a rapid pulse, recurring rigors, profuse sweatings, and especially tenderness and swelling over the point of origin of the internal jugular vein are strongly in favour of sinus phlebitis; but in cases of ear disease in which cerebral or cerebellar abscess is so frequently present, the presence of these symptoms (that is, of sinus phlebitis) does not, of course, exclude abscess. In such circumstances both conditions may be present.

The Differential Diagnosis between Abscess of the Brain and Ear Disease.—As a rule there is no difficulty in deciding whether the symptoms are merely the result of the ear disease, or whether they depend upon an intracranial lesion (abscess, meningitis, sinus phlebitis) which has resulted from the ear disease. But it must be remembered that headache (not merely local pain in the ear, but radiating pains in the scalp and skull, and severe internal headache), vomiting, giddiness, nystagmus, ataxia, and even epileptiform convulsions and optic neuritis may result from local irritation at the periphery, that is, in the diseased ear. With the exception of giddiness, ataxia, and nystagmus, such symptoms, more especially optic neuritis, are strongly in favour of the more serious condition (abscess).

A slow pulse (especially a slow pulse which is becoming gradually slower and slower), increasing mental dulness, and apathy are highly suggestive of abscess rather than of mere local ear disease. Peripheral irritation in the ear may produce irritative symptoms (headache, vomiting, giddiness, or even epileptiform convulsions), but it is not likely to produce paralytic symptoms (drowsiness, apathy, coma, or motor paralysis).

A marked leucocytosis is in favour of a cerebral abscess, though it may be merely the result of local suppuration in connexion with the ear lesion.

The Differential Diagnosis between Cerebral Abscess and Cerebral Tumour.—In some cases of encapsulated abscess the distinction is extremely difficult, indeed it may be impossible. In order to distinguish these two conditions, attention should be chiefly directed to the following points :—

(i.) The Presence or Absence of a Cause of Abscess.—This point I have already sufficiently emphasised ; but let me here say that I never commit myself to a positive diagnosis of intracranial tumour unless suppurative disease of the ear and nose—the more common causes of (non-traumatic) cerebral abscess—can be excluded.

In those cases in which a local cause of abscess (such as suppurative ear disease) is present, in addition to the general symptoms suggestive of tumour or abscess (headache, vomiting, and, it may be, double optic neuritis), the chances are strongly in favour of abscess rather than of tumour. It is, of course, possible to have an intracranial tumour and an otorrhoea (or other local cause of cerebral abscess) accidentally associated ; but the probabilities are strongly against such a coincidence. Conversely, when no discoverable local cause of abscess is present, the chances are, other things being equal, very strongly in favour of tumour ; and the correct diagnosis of cerebral abscess in such circumstances can seldom be more than a fortunate guess.

In this connexion I may further point out that any acute disease, such as influenza, in which headache is a prominent symptom may, if the patient is the subject of ear disease, for a time closely simulate a cerebral abscess.

(ii.) The previous history, progress, and course of the illness may throw light on doubtful cases ; when no clear or trustworthy evidence on these points is forthcoming, the difficulties of diagnosis are much increased. A history of former ear disease, with the subsequent long continuance of cerebral symptoms, suggests abscess.

The rapidity with which the symptoms progress does not necessarily afford any certain guide ; for in some instances of encapsulated abscess (that is, while the abscess is still encapsulated and unassociated with cerebritis, meningitis, or sinus phlebitis) the course of the case is very chronic ; and there may be few characteristic symptoms, if any. In tumour the same absence of striking symptoms may also of course be observed. Speaking generally, the progress of an abscess is much more rapid than that of a tumour.

(iii.) The Nature of the Symptoms.—In tumour the headache is, I think, usually more severe. This statement, like the former, only applies to encapsulated abscesses (while still encapsulated) ; for in acute cerebral abscess I have witnessed the most intense headache which it is possible to conceive. Double optic neuritis is also much more frequent in tumour. In a doubtful case unilateral optic neuritis is perhaps in favour of abscess, but more information is required before it is possible to generalise on this point. The intensity (degree) of the optic neuritis is also of some importance; it is usually more intense in tumour than in abscess. This is of course a general statement; for in some cases of intracranial tumour there is no optic neuritis, and in others it is only slight; in cerebellar abscess the optic neuritis may be very intense.

The temperature may not afford any guide; for in abscess, as in tumour, it is usually normal or subnormal. A swinging temperature, with rigors and sweatings, is of course strongly in favour of abscess (or of abscess associated with sinus phlebitis) as against tumour. A decided leucocytosis is strongly in favour of abscess.

Marked emaciation, apparently the result of the cerebral lesion, perhaps suggests abscess, though it sometimes occurs in tumour.

In a doubtful case, the sudden occurrence of symptoms indicative of severe diffuse meningitis would be strongly in favour of abscess. Symptoms and signs of sinus phlebitis likewise, of course, point strongly to abscess.

(iv.) The Situation of the Lesion.—This is a matter of some importance: for in the great majority of cases of cerebral abscess due to ear disease the abscess is either in the temporo-sphenoidal lobe or in the cerebellum; the situation of the lesion in some other part of the brain is therefore in favour of tumour.

A history of head injury is not always conclusive; for, like abscesses, tumours especially syphiloma, glioma, and sarcoma, occasionally follow injury.

(v.) The Numerical Chances.—These must only, of course, be considered when other things are equal; a cerebral tumour is a much more common lesion than a cerebral abscess, and infinitely more common than a cerebral abscess of apparent idiopathic formation,—than a cerebral abscess, that is, without a definite and distinct cause.

The Differential Diagnosis between Cerebral Abscess and Thrombosis with Cerebral Softening.—This is usually easy, but cases occasionally occur in which it may be very doubtful which of the two is present. The chief points to which attention should be directed are:—(*a*) the presence or absence of a cause of abscess; (*b*) the age of the patient; (*c*) the presence or absence of a history of syphilis; (*d*) the intensity of the headache and vomiting.

Arterial thrombosis with softening is essentially an acute lesion. It is therefore only in cases of rapidly formed abscess, or in cases of chronic abscess in which acute cerebritis or meningitis is set up around the abscess, that this question of diagnosis is likely to occur.

Cases of atheromatous thrombosis are rarely, if ever, likely to be confounded with cerebral abscess. It is in the syphilitic cases which occur at an earlier age, and in which there may be headache, vomiting, and optic neuritis due to associated gummatous meningitis, that there is likely to be any real difficulty.

The Differential Diagnosis between Acute Cerebral Abscess and Acute Haemorrhagic Encephalitis.—In both of these conditions urgent cerebral symptoms—intense headache and vomiting, with, it may be, focal symptoms, such as a rapidly developed hemiplegia or aphasia—may occur. Both conditions may follow influenza. In both conditions there may be fever. The diagnosis may consequently be very difficult; but as a matter of fact it very rarely indeed occurs, for acute haemorrhagic encephalitis is very rare. The presence or absence of a cause for abscess, such as ear disease, is a very important point. The temperature is usually high and the pulse rapid in acute haemorrhagic encephalitis; whereas in abscess, except in the early stage, the temperature is usually subnormal or normal, and the pulse not quickened.

Local Diagnosis.—When the symptoms and signs justify a diagnosis of abscess, the next question is, What is the situation of the abscess? This point will be more appropriately considered on p. 263 *et seq.*

Prognosis.—The prognosis depends upon the presence or absence of complications and upon the chances of successful surgical interference.

In acute cases, unless the abscess can be successfully treated by surgical interference, death is almost certain to take place speedily. But in a certain proportion of cases the surgeon cannot hope to interfere successfully. In some cases it is difficult or impossible to determine the exact position of the abscess; in some cases there is more than one abscess; and in other cases, even if the abscess be successfully diagnosed, reached, and evacuated, the patient dies of some associated complication (acute cerebritis, purulent meningitis, sinus phlebitis, lung disease, etc.).

In the subacute and chronic cases the prognosis is more favourable, provided that the position of the abscess can be diagnosed; for complications, such as cerebritis, meningitis, and sinus phlebitis, are much less likely to be present. Further, in those cases in which the abscess is encapsulated, it may remain latent or unattended with any serious symptoms for many years. But it must be remembered that in cases of encapsulated abscess the duration is always a matter of great uncertainty; for acute complications, such as cerebritis, oedema of the brain, and meningitis, may arise at any moment, and if so, unless the case can be successfully treated by the surgeon, rapid death is the result.

As a matter of fact, the prognosis in cases of cerebral and cerebellar abscess is very much more favourable than it was a few years ago. Sir W. Macewen gives a very optimistic opinion on this point; he states that "One might almost conclude that in uncomplicated abscess of the brain, operated upon at a fairly early age, recovery ought to be the rule."

Local Diagnosis and Treatment.—Since in the great majority of cases of abscess of the brain the only hope for the patient is successful surgical treatment, the next question which has to be considered when the symptoms and signs seem to indicate the presence of an abscess in the brain is, Where is the abscess situated?

The pyaemic cases may be left out of account, for in cases of this kind surgical interference has very little chance of success; the abscesses—for there are usually several—are often of small size and unattended with definite localising symptoms, or in consequence of the multiplicity of the abscesses the local diagnosis is at fault.

In those cases in which there is reason to suppose that the abscess is single, the local diagnosis has to be determined in exactly the same way as in any other case of cerebral disease, such as an intracranial tumour; namely, by observing the positive symptoms (paralytic or irritative) indicative of disturbance of function in localised areas of the brain, and the negative symptoms indicative of the absence of disturbance of function in other localised areas of the brain.

The points to which attention should be particularly directed are:—

(i.) The exact nature of the cause (injury, ear disease, nose disease, etc.); and if ear disease, whether the disease affects (α) the tympanum, or (β) the external ear or mastoid.

(ii.) The presence of localising symptoms (positive and negative), shewing that the abscess is situated in the temporo-sphenoidal lobe, the cerebellum, the frontal lobe, and so on.

(iii.) The relative frequency of an abscess in these different situations. It is only, of course, when there are no positive symptoms that any weight should be given to a probability of this kind.

In the traumatic cases the abscess is usually situated immediately below the seat of the injury, or (but this is less frequent) at the point of contre-coup.

In the traumatic cases in which there are no localising symptoms, the centre of the incision should of course be made over the wound, or any local tender spot or local swelling which happens to be present on the surface of the skull. If an abscess be not found on the surface of the brain, the brain tissue should be explored with a trocar. If the abscess be not found below the seat of the injury, the point of contre-coup should perhaps be trephined and explored, but the surgeon must determine this point.

When the abscess is due to ear disease, and when there are no definite localising symptoms, the temporo-sphenoidal lobe should be explored first, since in this situation the localising symptoms (more especially if the right temporo-sphenoidal lobe is the seat of the abscess) are apt to be less marked than when the abscess involves the lateral lobe of the cerebellum.

Abscess in the Temporo-sphenoidal Lobe.—The positive symptoms indicative of the presence of an abscess in the temporo-sphenoidal lobe are often very indefinite; indeed, in some cases there are no positive symptoms. The temporo-sphenoidal lobe is one of the so-called "silent" areas of the brain; for lesions in this situation are not infrequently latent, that is, unattended with definite localising symptoms. But in some cases positive localising symptoms, such as disturbances of hearing, smell, and taste, and, if the lesion is left-sided and the patient is right-handed, word-deafness and especially, it is said, inability to name objects are present. As a matter of fact loss of hearing, smell, and taste is rarely met with, for these sensations are largely represented on both sides of the brain; consequently, when one temporo-sphenoidal lobe is destroyed, its functions are taken up and carried on by the corresponding centres on the opposite side. The same statement applies, though less forcibly, to word-deafness. Further, in cases of cerebral abscess due to ear disease, the deafness, usually unilateral, sometimes bilateral, due to the ear disease is a complicating factor which has to be taken into account.

Irritative symptoms, such as tinnitus and hallucinations of hearing, and crude sensations of smell and taste, such as are apt to attend and usher in the peculiar form of fits described by Dr. Hughlings Jackson as "uncinate" fits, occasionally, no doubt, occur in cases of temporo-sphenoidal abscess; but these symptoms were not noticed in any of the cases under my own care. Mr. Ballance states that a peculiar "dreamy state" in many cases results from temporo-sphenoidal abscess, and that this symptom is more frequent in cases of right-sided temporo-sphenoidal abscess. But since a dazed mental condition is a common feature of cases of cerebral abscess, whatever their position, too much localising value should not, I think, be given to this peculiar mental state. Large abscesses in the temporo-sphenoidal lobe are apt to press upon the adjacent parts of the brain and to produce various symptoms. Paralysis of the opposite side of the face, of the opposite arm, less frequently of the opposite leg, and motor aphasia may result from compression of the ascending frontal convolution, left third frontal convolution (? the motor speech-centre), or the motor division of the internal capsule. With the paralysis there is loss of the abdominal reflex, an extensor toe-response, exaggeration of the knee-jerk, and, it may be, ankle-clonus on the opposite side to the abscess. Occasionally localised Jacksonian attacks involving the face and arm on the opposite side may be produced by pressure on, and irritation of, the motor cortex. Pressure on the ascending parietal convolution, the parietal lobe, and the posterior end of the internal capsule may produce disturbance of sensation (numbness, tingling, pins and needles, anaesthesia, astereognosis) in the face, arm, or even leg on the opposite side; pressure on the angular gyrus and subjacent white matter may produce word-blindness, agraphia, and hemianopsia. Pressure on the sixth and third nerves at the base of the brain may produce paralysis in the corresponding ocular muscles. Lesions (abscesses, tumours, etc.) in the temporo-sphenoidal lobe are in some cases attended with marked hysterical symptoms; a voracious appetite is sometimes present.

Abscess in the Cerebellum.—When the abscess is in the lateral lobe of the cerebellum, occipital headache, vertigo, and vomiting are usually prominent features; optic neuritis is more frequently present and more marked than in temporo-sphenoidal abscess; rigidity of the neck and retraction of the head are often present. Some loss of power, but not complete paralysis, loss of tonicity (hypotonia), and inco-ordination (diadochokinesis) are often present in the limbs (arm and leg) on the same side as the lesion. If the patient can walk, a reeling gait and a tendency to fall or walk to the side of the lesion are usually present. A peculiar position of the head, in which the head is depressed to the side of the lesion and the face turned to the opposite side, is present in some cases. The conjugate movement of the eyeballs to the side of the lesion may be impaired; there may be slow nystagmus when the patient attempts to look to the side of the lesion; skew deviation of the eyeballs is sometimes present. The functions of the fifth, sixth, and seventh nerves are in some cases impaired, but as regards the seventh nerve it must be remembered that facial paralysis may be due to the local ear disease. The localising signs of cerebellar abscess are well described by Mr. Trotter.

A more detailed description of the focal symptoms which result from disease in the temporo-sphenoidal lobe, cerebellum, and other parts of the brain is given in the article on "Cerebral Localisation" (see pp. 90, 134).

From the statements which have just been made, it will be obvious that the localising symptoms which result from abscess in the different areas of the brain (more particularly the temporo-sphenoidal lobe) are in many cases indefinite. Nevertheless, in those cases in which the abscess is due to ear disease we can usually, by a careful consideration of all the facts, decide upon the exact position of the abscess.

When the abscess is due to nose disease, it is most frequently situated in the frontal lobe on the same side; hence the localising symptoms indicative of a lesion in this situation must be carefully looked for. These symptoms are often very indefinite; in some cases of frontal abscess there are no focal symptoms.

Treatment.—The only treatment for a cerebral abscess—surgical interference—is fully described in other parts of the work (see Vol. IV. Part II. p. 492). Let me merely state that, in addition to surgical measures, internal remedies (such as quinine, the tincture of the perchloride of iron, bromide of potassium, chloral hydrate, the local application of cold to the head) are in some cases advisable, and that appropriate feeding, careful nursing, the prevention of bed-sores, attention to the condition of the bladder and rectum, and to the general hygienic surroundings of the patient, are of course essential.

Byrom Bramwell.

REFERENCES

1. Ballance, C. A. *Brit. Med. Journ.*, 1908, ii. 1266.—2. Barker, A. E. J. *Hunterian Lecture on Intracranial Inflammations starting from the Temporal Bone*, 1890.—3. v. Bergmann. *Die chirurgische Behandlung von Hirnkrankheiten*, Berlin, 1889, and *Berlin. klin. Wchnschr.*, 1895, xxxii. 337.—4. Bramwell, B. *Edin. Hosp. Rep.*, 1898, v. 128.—5. Broca et Maubrac. *Traité de chirurgie cérébrale*, Paris, 1896.—6. Chipault. *Chirurgie opératoire du système nerveux*, 1894.—7. Dana. *New York Med. Journ.*, 1899, i. 253.—8. Ferrier. *Brit. Med. Journ.*, 1888, i. 530.—9. Greenfield and Caird. *Ibid.*, 1887, i. 317.—10. Gowers. *Diseases of the Nervous System*, 1893, ii. 435.—11. Huguenin. Ziemssen's *Cyclopaedia*, 1877, xii. 375.—12. Macewen, W. *Pyogenic Infective Diseases of Brain and Cord*, 1893, p. 334.—13. Oppenheim, H. "Die Encephalitis und der Hirnabscess," *Spec. Path. u. Therap.*, Nothnagel, Wien, 1897, ix. 1.—14. Starr, M.A. *Brain Surgery*, 1893.—15. Trotter, W. *Brit. Med. Journ.*, 1908, i. 612.—16. Whitehead, A. L. *Ibid.*, 1908, ii. 1268.

B. B.

INTRACRANIAL TUMOURS

By Byrom Bramwell, M.D., F.R.C.P.Ed.

Definition.—Under the term "intracranial tumour" are included all new growths which occur within the cranial cavity.

Morbid Anatomy and Pathology.—Intracranial tumours are very common, almost every known form of new growth may occur within the cranial cavity. The most common forms are glioma, sarcoma, tuberculoma, syphiloma, endothelioma, and simple cysts, next in frequency carcinoma; then fibroma and osteoma; and, lastly, some rare forms (such as parasitic cysts, psammoma, neuroma, and lipoma), which, because of their rarity or because some of them, such as psammoma, are usually unattended with clinical symptoms, are of little practical importance.

Large aneurysms at the base of the brain, and some (simple) enlargements (hypertrophies) of the pituitary body may for clinical purposes, since they may be attended with exactly the same symptoms, be regarded as intracranial tumours. Further, some chronic encapsulated abscesses may, for practical purposes, be considered as cystic tumours.

Some intracranial tumours are primary, others are secondary. In some cases the new growth is single, in others multiple. In some cases the tumour is situated within the substance of the brain, in others on its surface. In some cases the new growth has its point of origin in the membranes or cranial bones, in other cases in the neuroglial tissue. In some cases the new growth is more or less localised, it may be encapsulated; in others (glioma, glio-sarcoma), diffused and infiltrating the nervous tissue. Any part of the brain (cerebral hemispheres,—grey or white matter,—basal ganglia, corpus callosum, crura cerebri, pons, medulla, or cerebellum) may be the seat of the tumour.

The size of the tumour varies greatly in different cases.

The rapidity of growth—an important point so far as the clinical symptoms are concerned—is very different in different cases; in some cases the tumour increases with great rapidity; in others the growth is very slow and gradual; in exceptional cases the tumour may remain stationary for years; in very rare instances, and especially in tuberculomas, the tumour ceases to grow, undergoes caseation or calcification, and then becomes encapsulated. I have seen two examples of this, one verified by necropsy (33).

The effects produced on the nervous tissues and on structures with which the tumour comes in contact are also very variable; this point will be considered in connexion with the pathological physiology.

Etiology.—Intracranial tumours are due to the same causes as tumours elsewhere. In many cases we are quite ignorant of the causation. In cases of secondary intracranial tumour the primary growth is often situated in the lung. In many tuberculous and syphilitic cases there is evidence of tuberculosis or syphilis in some other part of the body. When an intracranial tumour is a parasitic cyst, cysts of the same nature may be situated in other parts of the body.

Hereditary influences seem occasionally to play a part in the production of some forms of intracranial tumour (carcinoma, tuberculoma, syphiloma). Injury is in some cases undoubtedly a proximate cause, especially in tuberculous, syphilitic, and gliomatous new growths. Mental anxiety, mental strain, worry, depressing influences of all kinds, ill health, and alcoholic excesses seem in some cases to dispose to the development of the new growth, in other cases actually to excite it. Some of these conditions probably act by disturbing the cerebral circulation and nutrition, and so disposing to the production of the new growth.

Age has an important influence in the production of intracranial tumours. New growths rarely occur within the cranial cavity in early infancy, or in old age. Tuberculous tumours are common in children. Melanotic sarcoma occasionally occurs in childhood and youth. Syphilitic tumours usually occur between the ages of thirty and forty-five; in rare instances they are found in young subjects as the result of inherited syphilis. Sarcoma occurs both in youth and middle age, particularly in middle life. Glioma is chiefly seen in young and middle-aged adults; it is, comparatively speaking, rare in childhood and early youth, and also in old age. Carcinoma occurs most frequently in middle-aged and old people.

The male *sex* is more frequently the subject of intracranial tumours than the female—probably because syphilis, head injuries, mental worry, over-strain, and alcoholic excesses are all more common in men than in women.

Pathological Physiology.—This is of great importance; an intelligent comprehension of the symptomatology of brain tumours largely depends upon a knowledge of the manner in which the symptoms are produced. In studying the pathological physiology of intracranial tumours it is important to remember that the cranium, after the fontanelles are closed, is practically a closed cavity with rigid walls; consequently an intracranial tumour, as it increases in size, is apt to produce symptoms by pressing upon the surrounding parts. These symptoms, which are either "irritative" or "destructive" in character, may be arranged in the three following groups:—(1) *General symptoms*, due to disturbance of the function of the intracranial contents as a whole. (2) *Localising symptoms*, due to the irritative or destructive effects which the tumour causes in the structures (brain tissue, membranes, nerve-roots and blood-vessels) with which it is in immediate contact; and (3) *False localising symptoms*, due to derangement of function in localised parts of the brain at a distance from the tumour.

These false localising symptoms, which are very deceptive, may be produced by distant pressure, stretching of nerves, impeded circulation, haemorrhagic extravasation, and oedema of the brain.

Degenerative changes in the posterior (spinal) nerve-roots and posterior columns of the spinal cord are often met with in cases of intracranial tumour; they seem to be due to traction on the posterior nerve-roots by the distension of the arachnoid, the result of increased intraspinal pressure. Loss of the knee-jerks and other symptoms may, it is believed, be due to this cause. Lejonne reports fifteen personal observations of this kind. Drs. Batten and Collier found degeneration of the posterior columns of the cord in 65 per cent of their cases of brain tumour.

The *general* symptoms, due to disturbance of the function of the intracranial contents as a whole, are usually the result of increased intracranial pressure, and the derangement of the circulation and nutrition which results therefrom (61). In some cases the general symptoms appear to be due to irritative changes widely diffused throughout the nervous tissues and cerebral membranes; in others to auto-intoxication resulting from the tumour or the defective removal of effete products due to lymph stasis (78).

A growing tumour which causes marked increase of the intracranial pressure, which disturbs the vascular and lymphatic circulations, which stretches the membranes, and which compresses the contents of the intracranial cavity or which produces great distension (dropsy) of the ventricles, will necessarily produce more or less disturbance of the cranial contents as a whole, and will give rise to general symptoms; while at the same time it may produce *localising* symptoms by direct pressure, or *false localising* symptoms in the manner described above.

Ventricular distension is an important cause of general symptoms. In young subjects the cranial sutures may become separated, the bones of the skull thinned and the head enlarged as the result of long-continued increase of the intracranial pressure. The spontaneous discharge of cerebrospinal fluid, usually through the nose, occasionally through the ear, sometimes, though rarely, occurs. A case in which the symptoms subsided after a discharge from the nose came under my notice.

The cranial cavity is divided into two sub-cavities by the tentorium cerebelli. Tumours which grow below the tentorium, and which are hemmed in by this firm, resisting membrane, produce very definite effects upon the structures in the lower cavity; tumours situated above the tentorium cerebelli have much greater room for expansion and growth. Tumours below the tentorium are apt to impede the circulation through the veins of Galen, to produce dropsy of the ventricles, and to cause great increase of the intracranial pressure as a whole. Further, the upper cranial cavity is imperfectly divided into two sub-cavities by the falx cerebri; consequently a tumour in one hemisphere of the brain is apt to compress the structures on its own side more than on the opposite side.

The degree of the increased intracranial pressure produced by tumours varies considerably in different cases. Small tumours of slow growth may not cause any pressure-symptoms; these cases are, however, rare.

The severity of the general symptoms seems chiefly to depend upon the size of the tumour, the rapidity with which it grows, and the condition of the ventricles. When the increased intracranial pressure reaches a certain degree of intensity the functions of the whole brain become more or less impaired, particularly if the increased intracranial pressure is rapidly developed.

The symptoms due to local alterations in the brain tissue, cerebral membranes, nerve-roots, and bony coverings with which the tumour is in direct contact, also depend upon a number of different factors. The more important are :—the function of the part which is directly implicated; the manner in which the nerve tissue is affected (whether irritated or destroyed); and the amount of substitution and compensation, if any, which can be established. It is unnecessary to enter into a detailed discussion of these different points.

In some cases large tumours which infiltrate or even destroy extensive portions of the brain are unattended with any obvious localising symptoms. I have recorded several remarkable cases of this kind (11).

As I have already stated, the false localising symptoms are very deceptive; but they are comparatively rare. As a matter of fact, in the great majority of cases in which symptoms indicative of disturbance of function in a localised area of brain tissue are present, they are due to the destructive or irritative changes produced by the new growth in the structures with which it is immediately in contact; in other words, they usually have a distinct and definite localising value. Dr. James Collier found that in 20 of 161 cases, or 12·5 per cent, of brain tumour, false localising symptoms were present.

Clinical History.—The nature of the symptoms and the manner in which they are grouped together are very variable in different cases of intracranial tumour. Further, the individuality of the patient in this, as in almost every form of disease, plays an important part in determining the severity and to some extent the character of the symptoms. Similar tumours may produce somewhat different effects in different individuals.

In the great majority of cases an intracranial tumour is a chronic lesion which is unattended with fever; in some cases acute symptoms arise as the result of haemorrhage, meningitis, cerebritis, rapid dropsical effusion into the ventricles, and so forth.

In many cases of intracranial tumour both *general* and *local* symptoms are present; in some, general symptoms only are present; in others, the symptoms are chiefly local. In rare cases there are absolutely no symptoms. Cases occasionally occur in which all the characteristic symptoms are present during life, but in which no tumour is found after death.

The more important general or "non-localising" symptoms are :—Headache, vomiting, giddiness, double optic neuritis, optic atrophy, and loss of vision resulting therefrom; general epileptic convulsions which begin without any definite aura or local starting-point, symptoms indicative of disturbance of the mental balance and intellectual functions, such as loss of memory, apathy, hysterical symptoms, mental depression, melancholia, mania, and coma.

The more important localising symptoms are :—Paralyses of various forms, such as hemiplegia, monoplegia, more rarely paraplegia or paralysis of all four limbs, paralysis of the individual muscles and groups of muscles supplied by motor cranial nerves, nystagmus, derangements of the associated movements of the ocular muscles, skew deviation of the eyes, a peculiar position of the head (in some cerebellar cases), rigidity of the neck, localised epileptiform convulsions either with or without a sensory aura ; localised tonic, tetanic-like spasms in the muscles of the back, neck or limbs, fine tremor in one hand, rhythmical tremors resembling those of cerebrospinal sclerosis, hypotonia or hypertonia in a limb or limbs on one side of the body ; disturbance of co-ordination, a reeling gait, a tendency to fall to one side, or forced movements, unilateral alterations of the superficial or deep reflexes, localised derangements of sensation, such as anaesthesia, hemianaesthesia, hyperaesthesia, shooting pains or hyperaesthesia in the area of distribution of the fifth nerve, astereognosis, hemianopsia of various forms, hemiopic flashes of light, visual and auditory hallucinations, peculiar epileptiform attacks attended with a dreamy state and ushered in by a peculiar taste or smell, and, it may be, attended with smacking of the lips, chewing or spitting movements, unilateral disturbances of hearing, smell, or taste, aphasic symptoms, motor or sensory, polyuria, albuminuria, glycosuria, and to these may be added the enlargement of the face, feet, hands, and other parts—the signs of acromegaly — which in some cases results from enlargement of the pituitary body.

Some of these symptoms are common, others are rare. Headache, vomiting, and optic neuritis are more frequent than the most common of the localising symptoms, such as hemiplegia ; whilst of the general symptoms, headache, vomiting, and double optic neuritis are much more common than mania or melancholia. Of the localising symptoms, motor alterations, such as monoplegia, hemiplegia, localised epileptiform convulsions are more common than sensory alterations, such as hemianaesthesia, astereognosis, hemianopsia, or localised sensory Jacksonian attacks (hemiopic flashes of light, due to irritation and discharge of the half-vision centre, auditory hallucinations, etc.).

There is no absolute distinction between the "general" and "localising" symptoms. Thus, pain in the head, if limited to one spot, more especially if associated with tenderness on firm pressure or skull-percussion, may have a very definite localising value. Even mental apathy, if developed early, and in the absence of marked signs and symptoms of intracranial pressure, may have a localising value.

It may now be well to consider some of these symptoms individually, and in detail.

Headache.—This is the most frequent of all the symptoms. It is present in the great majority of cases, but its severity is very variable ; in some cases the pain is intense. The headache is often paroxysmal ;

during the intervals the patient may be entirely free from pain. In many cases the pain is felt when the patient first wakes in the morning; in such cases it is apt to be associated with vomiting. In some cases the pain is worse at night, and is then suggestive, though by no means pathognomonic, of a syphilitic lesion. The pain is usually referred to the interior of the head. Superficial pain limited to a localised area of the scalp, especially when associated with tenderness on pressure, suggests a gumma or other form of tumour invading the bones, membranes, or superficial parts of the brain beneath the tender area. Neuralgic pains in the distribution of the fifth nerve may result from the pressure of a tumour on the trunk of this nerve. In my experience, headache is apt to be especially severe in large and rapidly growing tumours, and in cases with great increase of the intracranial pressure.

Double Optic Neuritis.—Next to headache this is the most frequent symptom, or rather sign, of an intracranial tumour. From a diagnostic point of view double optic neuritis is a much more important symptom than headache; for optic neuritis is comparatively rarely met with in conditions other than tumour; and, further, it is an alteration which can be seen by the physician himself, and which cannot be produced or simulated by the patient. In the great majority of cases the optic neuritis is bilateral. Even when considerable in degree it is not necessarily associated with any diminution of the acuity of vision. In many cases both the acuity and fields of vision are considerably impaired; in some cases vision is completely lost; this is usually due to advanced post-neuritic atrophy, but in more than one case I have seen complete loss of vision suddenly or rapidly developed before there was any atrophy, apparently as the result of the acute onset of ventricular effusion. So far as my experience enables me to judge, optic neuritis is present in at least 80 per cent of cases of intracranial tumour at some period or other of their course. The degree of change in the optic disc varies in different cases, and at different stages of the same case. In the early stages there may be merely distension of the retinal veins; in the later stages most intense papillitis. The optic neuritis is frequently more marked in one eye, and, in rare cases, is unilateral throughout the whole course of the case. A one-sided difference of the optic neuritis seems to be much more frequently due to a cerebral than to a cerebellar (subtentorial) tumour, and is usually due to a tumour on the same side of the brain. Optic neuritis is very frequent and severe in cases of cerebellar tumour and in large, rapidly growing tumours above the tentorium. Optic neuritis is often absent in pontine tumours and in small, slowly growing tumours above the tentorium, in fact in all tumours in which there is little or no increased intracranial pressure (30, 47). James Bordley, jun., and Harvey Cushing have recently shewn that in a large proportion of cases of intracranial tumour the colour-fields are altered (interlaced) in the same way as in cases of hysteria.

Vomiting.—The severity and time of occurrence of the vomiting are variable. In many cases the vomiting chiefly occurs when the patient first gets out of bed in the morning, and is then apt to be associated with headache and giddiness. Paroxysmal attacks of headache and vomiting frequently occur together. In some cases the vomiting occurs after eating. Very frequent and severe vomiting suggests a subtentorial tumour or great meningeal irritation.

Vertigo.—This is usually a less important and constant symptom than headache and vomiting, but it is often present in a slight degree and as a temporary condition. Slight and temporary giddiness is probably due to disturbances in the cerebral circulation. Severe and constant vertigo (if the result of an intracranial tumour and not due to ear disease) suggests a tumour involving the middle lobe of the cerebellum, or the nerve which carries the labyrinthine impressions from the internal ear to the cerebellum; tumours in the cerebello-pontine angle may be attended with severe giddiness. In other cases of intracranial tumour, vertigo is the result of paralysis of one or other of the ocular muscles. From these statements it is obvious that the exact cause of the vertigo must be determined before a definite localising value can be attributed to it

General epileptiform convulsions sometimes result from intracranial tumours, quite irrespective of their seat and position. Occasionally an epileptic fit is the immediate cause of death. In some cases a general epileptiform convulsion is the result of haemorrhagic extravasation from the thin-walled vessels of a glioma; but in most cases it is probably due to the irritation of the cerebral tissues which the tumour produces. In rare cases, general epileptic convulsions may for a long time be the only symptom produced by an intracranial tumour (16).

Disturbances of the mental faculties are frequent; in most cases the mental alterations (changes of disposition, irritability, impairment of memory, mental apathy, inability to concentrate the attention, hysterical manifestations, and the like) are so slight that they are apt to pass unrecognised, or to be regarded as of little or no importance. More pronounced mental alterations—such as melancholia, mania, or dementia—are rare; in a few cases symptoms of actual insanity occur. In the terminal stages of intracranial tumours, especially when the intracranial pressure is greatly increased, mental apathy, impairment of memory, inability to concentrate, stupor, and coma frequently set in. The mental alterations which occur in the course of intracranial tumours are, in the majority of cases, of no localising value; but tumours involving the corpus callosum and frontal lobe are especially apt to produce taciturnity, want of attention, inability to concentrate the thoughts, and marked deterioration or impairment of the mental faculties, even in those cases in which the intracranial pressure does not appear to be greatly increased. When mental symptoms appear early, when they do not seem to depend upon increased intracranial pressure, and when the patient's mental condition was normal before the onset of the symptoms of intracranial tumour, and when the influence of alcoholic excess and other factors likely to produce mental symptoms can be excluded, the presence of mental symptoms has a definite localising value. A marked tendency to sleep (intense drowsiness) is a striking feature in some cases of tumour of the pituitary body, the corpus callosum, and the frontal lobe (72). Opinions differ whether mental changes are more marked when the left frontal lobe is the seat of the tumour, but there seems to be little doubt that the psychical symptoms are marked when both frontal lobes are affected (73). Knapp (41) noted mental symptoms in 58 out of 64 cases of uncomplicated tumour. Raymond associates with tumours of the corpus callosum a mental syndrome, which is characterised by eccentricity in manner and in actions, hiatuses in memory, want of sequence in ideas, coupled with an apparent conservation of the intelligence (*vide* also p. 99).

Apoplectic or pseudo-apoplectic attacks occur in some cases, and may be the cause of death. In some cases the apoplectic symptoms are due to haemorrhagic extravasations, which are especially prone to occur in cases of vascular glioma. In other cases pseudo-apoplectic attacks appear to be due to congestion, oedema, or inhibition.

Paralysis of various forms is a common and important localising symptom. Localised paralyses are frequently due to implication of one or other of the motor cranial nerves. Hemiplegia and monoplegia are the most common forms of paralysis; paraplegia and paralysis of all four limbs are much more rare. The duration, completeness, and extent of the paralysis, and the exact form of the hemiplegia (whether of the ordinary common form or of the "crossed" variety) differ in different cases. A slowly progressive cerebral paralysis—a monoplegia, for example, which slowly and gradually passes into a hemiplegia—strongly suggests tumour of the brain. This is a point of definite diagnostic value. The paralysis is sometimes merely temporary. Localised paralysis, monoplegia or hemiplegia, of this temporary kind is common after a localised epileptiform convulsion (Jacksonian attack). Bilateral paralysis involving all four limbs, the arms usually more than the legs, is generally due to a tumour in the pons or medulla, or to the pressure which a subtentorial (cerebellar) tumour exerts upon the motor strands of the pyramidal tract. In cases of this kind a rhythmical volitional tremor, which closely resembles that due to cerebrospinal sclerosis, may be present. In some cases of frontal tumour I have observed giving way of the legs, without any obvious paralysis. Paralysis of the cranial nerves, more especially of the 6th nerve, less frequently of the 3rd, 5th, and 7th nerves, may be a false localising symptom.

The *condition of the reflexes* is variable. When hemiplegia is present, the knee-jerk and Achillis-jerk on the affected side are usually exaggerated, there may be ankle-clonus, and there is an extensor plantar response (the Babinski sign); in some cases the knee-jerks are exaggerated on both sides, and there is a double extensor response. In some cases of cerebellar tumour, less commonly in cases of cerebral tumour (van Gehuchten, Raymond, and others), the knee-jerk on one or both sides is diminished or abolished. Dr. Grainger Stewart has pointed out that in tumours of the frontal lobe the abdominal reflex is absent or diminished on the side opposite to the tumour.

Paralysis of the bladder and rectum is comparatively rare, unless the mental condition of the patient is considerably impaired. Beevor stated that incontinence of urine is common in tumours of the frontal lobe.

Localised spasms and convulsions, like localised paralysis, are also most important "focal" or localising symptoms. Localised epileptiform convulsions—attacks of Jacksonian epilepsy—are usually due to a tumour which involves and irritates the motor cortex. Such tumours, when they are situated on the surface of the brain, are usually syphilitic, less frequently tuberculomas or sarcomas; when they have their starting-point in the cortex or subcortical white matter they are usually gliomas or tuberculomas. The extent and distribution of the convulsions depend upon the extent of the grey matter which is irritated and discharged (*vide* also p. 57).

The diagnostic value of localised epileptiform convulsions is very great, for the muscles which are first convulsed give a clue to the position of the motor grey matter which is irritated and first discharged.

Tumours which do not directly involve but which are situated in the immediate neighbourhood of the motor area (ascending frontal convolution) are frequently attended with Jacksonian attacks. When the tumour is situated behind the motor area (in the ascending parietal convolution or the parietal lobe), the Jacksonian attack is usually (?always) preceded by a sensory aura (a feeling of numbness, pins and needles, tingling, or pain in the part convulsed). It must also be remembered that localised epileptiform convulsions may result from tumours which are *remote* from the motor area (11, 20).

Tonic spasms and contractures also are occasional symptoms. Rigidity of the muscles of the neck and spine, with retraction of the head and arching of the back, occurring in paroxysms and in some degree resembling a tetanic fit, occurs in some cases of cerebellar tumour. Dr. Hughlings Jackson described these cerebellar fits, and pointed out that in some cases of disease of the middle lobe of the cerebellum the patient assumes a peculiar (the cerebellar) position.

Contractures may, of course, occur in cases with hemiplegia or monoplegia. Conversely, hypotonia in the weakened or paralysed limbs on the same side as the tumour is frequently present in cases in which the tumour is in the lateral lobe of the cerebellum.

A fine, rapid, vibrating tremor of the limbs, especially of the upper limb, *on the same side as the tumour* has been described as a characteristic feature of frontal tumours by Dr. Grainger Stewart. It is absent during muscular rest, and can be best demonstrated by making the patient extend both arms horizontally in front of him with the palms directed downwards and the fingers extended. I have recently seen a characteristic case of frontal tumour (verified by necropsy) in which this fine tremor was not present, and have also examined after death a case of cerebellar tumour in which it was present. This tremor closely resembles that of exophthalmic goitre. A characteristic volitional tremor, closely resembling that of disseminated sclerosis, may occur. I have seen it in some cases of subtentorial (cerebellar) tumour and in some cases of dilated ventricles without tumour. Dr. Gordon Holmes has shewn that tremor, both static and volitional movements, when associated with focal cerebral disease, may depend on a negative lesion of the cerebello-rubral system.

Athetoid movements are occasionally observed, and appear to be most frequently due to tumours which involve, or are situated in the neighbourhood of, the optic thalamus.

Inco-ordination.—A reeling (drunken) gait is common in cases of cerebellar tumour; in some cases in which the tumour involves the superior peduncle of the cerebellum, the patient tends to fall to one side, or in walking to go to one side or to perform circus movements; in other cases forced movements are present. In more than one case of frontal tumour I have noticed a peculiar gait, the patient first taking a few steps to one side and then a few steps to the other in a zigzag fashion. In some cases in which the tumour is in one lateral lobe of the cerebellum, the movements of the upper extremity on the side of the tumour are incoordinate and ataxic (*vide* also pp. 46, 143).

Sensory derangements, other than headache, are less frequent and, speaking generally, less important from a diagnostic point of view than disturbance of motion. A general impairment of sensation and loss of the tactile sense are not uncommon in cases in which the patient is apathetic, and the cerebral functions greatly depressed. Hemianaesthesia is not common, though it occasionally results from the presence of a tumour which involves, or is situated in the immediate neighbourhood of, the posterior end of the internal capsule, and of tumours which destroy the ascending parietal convolution and the anterior part of the parietal lobe. A certain degree of localised anaesthesia is also seen in some cases in which the tumour involves the motor area of the brain (ascending frontal convolution); cortical growths in this position are in some cases attended with loss of the sense of position and some sensory loss (inability to localise perfectly) at the periphery of the limbs. Recent observations shew that the ascending parietal convolution is chiefly a sensory area and that tumours which destroy this convolution are attended with loss of sensation on the opposite side of the body. Astereognosis occurs in some cases of tumour in the parietal lobe, and has also been noted in some cases in which the tumour involved the ascending parietal and ascending frontal convolutions. Mills believes that the stereognostic centre is situated in the superior parietal lobule. Anaesthesia, hyperaesthesia, neuralgic and shooting pains in the area of distribution of the fifth nerve may, of course, be present in cases in which the tumour is in the pons, or involves the trunk of the nerve at the base of the brain.

Derangements of the sense of sight are very common; some of them are of great importance from a localising point of view. Diminution of the acuity of vision, a more or less generalised contraction of the fields of vision, dimness of vision, and even complete blindness are frequently met with as a result of optic neuritis, and more especially of post-neuritic atrophy. Alterations of vision of this kind have no localising value. In more than one case of cerebral tumour I have seen sudden or rapid loss of vision without any obvious alteration in the fundus to account for it, apparently due to a sudden increase of the intracranial pressure. Blindness due to primary optic atrophy (optic atrophy not preceded by optic neuritis) is occasionally due to a tumour at the base of the brain which exerts direct pressure upon the optic chiasma or optic nerve-trunks, or to the pressure of the dilated infundibulum (due to great ventricular distension), enlarged pituitary body, or aneurysm, on the centre of the optic chiasma.

Hemianopsia is occasionally met with, and is a most important localising symptom. Homonymous lateral hemianopsia may, of course, result from a tumour which involves the optic tract, the fibres which pass from the optic tract to the half-vision centre (radiating fibres of Gratiolet, etc.), or the half-vision centre in the tip of the occipital lobe (calcarine region). When the tumour directly involves the optic tract, the hemiopic condition of the pupil is present. Bilateral temporal hemianopsia is occasionally observed as the result of the pressure of a tumour on the centre of the chiasma.

Flashes of light, referred to one or other side of the visual field (usually by the patient to the eye on the opposite side to the tumour), may result from a tumour in the back part of the occipital lobe which irritates the half-vision centre on the opposite side. Flashes of light occurring in paroxysms (visual Jacksonian attacks) are in some cases followed by temporary hemianopsia (paralysis). Visual hallucinations may be produced in the same way. I have recently published a case in which flashes of light and visual hallucinations were apparently due to irritation of the fibres of the optic tract; in this case the tumour involved the basal ganglia (14).

Derangements of the sense of hearing are much less common than derangements of the sense of sight. A tumour which presses upon or involves the auditory nerve at the base of the brain may, of course, produce loss of hearing. Deafness, usually complete, is a highly characteristic feature of tumours in the cerebello-pontine angle; these tumours often invade and, in many cases, have their starting-point in the sheath of the auditory nerve. Deafness may also result if the tumour destroys the auditory nerve nucleus in the pons Varolii. Loss of hearing is rarely due to a tumour situated in the cerebrum itself; but a tumour which destroys the first temporo-sphenoidal convolution (the auditory centre) may produce some impairment of hearing in the opposite ear; a bilateral tumour of the temporo-sphenoidal lobe may cause complete deafness in both ears. Some impairment of hearing (and in very exceptional cases marked deafness) may be due to increased intracranial pressure, effusion into the ventricles, and the general impairment of the cerebral functions which results therefrom.

A tumour which causes irritation of the auditory centre may be attended with subjective sensations of sound (tinnitus) or auditory hallucinations. A tumour which destroys the posterior two-thirds of the left first and the adjacent part of the second temporo-sphenoidal convolutions may produce word-deafness.

Dana states that the fits due to tumours in the cerebellum and posterior fossa, which cause irritation and pressure, are apt to be preceded by loud, high-pitched tinnitus or roaring and crackling noises, suddenly increased in intensity. In cases of this kind, vertigo, usually objective and with or without forced movements; a tendency to fall in one direction or another instantly to the ground; sometimes sudden blindness or loss of consciousness; and, in severe attacks, tonic spasms generally of an extensor type, which last from one or two to five minutes, are also, he finds, apt to follow and form, he thinks, a characteristic syndrome.

Disturbances of the Sense of Taste and Smell.—Loss of smell is comparatively rare in cerebral tumour, unless the olfactory bulb or root should happen to be directly implicated. Tumours which involve the under surface of the frontal lobe and tumours of the frontal lobe which compress the olfactory bulb may be attended with loss of smell in the nostril on the same side as the tumour. Destruction of the anterior part of both temporo-sphenoidal lobes by symmetrically placed tumours would probably produce complete loss of smell. A sensation of smell or taste may result from a tumour which irritates the centres for smell and taste in the tip of the temporo-sphenoidal lobe. Dr. Hughlings Jackson in 1876 described peculiar fits in which the patient had what is termed an "intellectual aura" or "dreamy state," and in which the attack was ushered in by a peculiar taste or smell (uncinate fits) (*vide* also p. 96).

Aphasic Derangements.—Marked and persistent aphasia is comparatively seldom due to an intracranial tumour; this is no doubt chiefly due to the facts that an intracranial tumour comparatively rarely produces complete destruction of the speech-centres, and that the lesion being a chronic one and the destruction of the speech-centres being gradually effected, substitution and compensation are in many cases established. Temporary aphasia is occasionally observed after an epileptic fit due to a tumour. I have seen some cases of this kind in which the tumour involved the left frontal lobe. Word-blindness, word-deafness, motor vocal aphasia, and agraphia may occur if the tumour completely destroys the cortical speech-area or the strands of white matter connected with it (see p. 385).

Altered Percussion Note.—In some cases in which the ventricles are dilated the percussion note over the skull is raised (high-pitched); a difference in the percussion note on the two sides of the head is also sometimes met with when the tumour involves the cerebrum (46, 62). Bruns lays stress on the value of local percussion of the skull in the diagnosis between a cortical and subcortical tumour.

Urinary Derangements.—Polyuria, phosphaturia, glycosuria, albuminuria, and peptonuria, which are occasionally associated with tumours of the pituitary body, in the region of the floor of the fourth ventricle and adjacent parts, may have some localising value, provided that they are not merely associated or accidental complications. In a case of pituitary tumour which recently came under my notice, extreme thirst and marked polyuria were present (15*a*, 65).

The *general state of nutrition* varies in different cases. In many cases the patient is well nourished; in others, a certain degree of emaciation is the result of long-continued pain and sleeplessness; marked emaciation is very exceptional and is usually due to some associated complication in the thoracic or abdominal viscera. In rare cases—in my experience more particularly in subtentorial tumours—extreme emaciation is rapidly produced, apparently as the direct result of the cerebral lesion (tumour).

The *temperature* in the great majority of cases is normal or subnormal. Pyrexia is usually the result of some complication, such as meningitis or cerebritis. Hyperpyrexia occasionally occurs before death in cases in which the tumour is situated in the region of the basal ganglia, pons, or medulla.

The *pulse* is usually normal in frequency, or slower than normal; but towards the end it may become markedly accelerated, and in some cases irregular.

Cheyne-Stokes respiration occasionally occurs, particularly in those cases in which the tumour is situated in the neighbourhood of the respiratory centre. Obstinate hiccup is occasionally present.

Bed-sores are not uncommon in the terminal stages of the case, particularly of course if the nursing be inefficient.

Clinical Types or Groups.—Cases of intracranial tumour may be grouped under the following clinical types:—

1. Cases in which an intracranial tumour is present, but in which its presence is not indicated by any symptoms during life. Cases of this kind are extremely rare, but they do occur occasionally.

2. Cases in which there are general symptoms (such as headache, vomiting, double optic neuritis, giddiness) which shew that there is a tumour in some part of the intracranial cavity, but in which there are no localising symptoms indicative of its exact site. The cases included in this group are frequent. In some of these cases, minute and exhaustive examination shews that some localising symptoms are actually present; but very large tumours, or even multiple tumours, in parts of the brain (such as the motor area and cerebellum) in which a tumour is usually attended with striking symptoms, not infrequently occur. I have already referred to several cases of this kind which have come under my own notice (11).

3. Cases in which, in addition to the general symptoms, well-marked localising symptoms, which indicate more or less clearly the exact position of the new growth, are also present. These cases are also common. In connexion with this group of cases I must again emphasise the facts (1) that in some cases false localising symptoms occur and give an altogether erroneous notion of the position of the tumour; and (2) that localising symptoms appearing in the early stages of the case are of much

greater diagnostic value than localising symptoms which are developed in the later stages; for in the later stages, when the intracranial pressure is greatly increased or the tumour is of large size, false localising symptoms are apt to be produced.

4. Cases in which definite cerebral symptoms indicative of functional disturbance or organic disease within the cranial cavity are present, but in which the symptoms are not distinctive of a tumour. This group is also a large one, and contains most of the cases in which difficulty in diagnosis arises.

Diagnosis.—In connexion with the diagnosis of cases of intracranial tumour, three questions have to be solved:—(*a*) Is an intracranial tumour present? (*b*) If so, where is it situated? And (*c*) what is its pathological nature?

In some cases the solution of all of these questions is easy; in others, although the diagnosis of the presence of a tumour may be readily arrived at, it may be difficult or impossible to determine its locality and pathological nature. The diagnosis of an intracranial tumour may be confidently made when the general symptoms (headache, vomiting, and double optic neuritis) are present, and when other conditions, such as Bright's disease, lead poisoning, abscess of the brain, great dropsical distension of the ventricles, and a few other conditions in which the same symptoms (headache, vomiting, double optic neuritis) may be present, can be excluded. The diagnosis is still more easily made when, in addition to the general symptoms, localising or focal symptoms are present.

Let us now consider the *differential diagnosis* of intracranial tumour and those other conditions which are most likely to be confounded with it.

Bright's disease must be excluded. In most cases this is easily done by an examination of the urine, heart, arteries, blood-pressure, and fundus oculi; but in some cases of fibrotic Bright's disease in which there is no dropsy and no albuminuria, the diagnosis may be very difficult; it is important to remember that the same ophthalmoscopic appearances may be produced in both conditions (13).

Lead poisoning must also be excluded. The blue line on the gums should be looked for, the occupation of the patient ascertained, and the presence or absence of other symptoms indicative of plumbism, such as dry colic and wrist-drop, determined; further, the urine should be examined for lead (15).

Hypermetropia in anaemic girls is sometimes attended with headache and a slight degree of papillitis, and such a case may thus be mistaken for one of tumour. The facts that the headache is relieved by suitable glasses, and that the symptoms disappear with the cure of the anaemia, are against an intracranial growth. I have, however, met with more than one case in which the diagnosis was extremely difficult.

Profound anaemia without hypermetropia is also in some cases attended with all the general symptoms of an intracranial tumour (headache, vomiting, double optic neuritis, etc.).

Amenorrhoea in young women is sometimes attended with optic neuritis, headache, vomiting, and the other general symptoms of an intracranial tumour. In some cases of this kind it is absolutely impossible to come to an exact diagnosis; this is more especially so in cases in which there is little or no anaemia.

Great dropsical distension of the ventricles (hydrocephalus) may also be attended with all the symptoms of an intracranial (more particularly cerebellar) tumour. In a case of this kind under my observation all the characteristic symptoms of a cerebellar tumour were present during life. After death the condition was found to be due to distension of the ventricles, the result of an old (healed) tuberculous meningitis. Several other cases of the same sort have also come under my notice (11, 51).

Cerebral Abscess.—The differential diagnosis of cerebral tumour and brain abscess is rarely attended with much difficulty. The question has been discussed on page 260. The chief difficulty occurs in cases in which an intracranial tumour occurs in a patient with a chronic otorrhoea, and in cases of chronic or subacute cerebral abscess in which no apparent cause for abscess, such as middle-ear disease, is present.

Epilepsy.—In rare cases of intracranial tumour the only symptom may for a long time be recurring epileptic fits. Dr. Edwin Bramwell has recently described a case of this kind, and pointed out that it may be impossible, until double optic neuritis, headache, and other signs appear, to distinguish such a case from ordinary idiopathic epilepsy.

Hysteria.—In some cases of intracranial tumour in which hysterical symptoms are prominent, the true nature of the case may be easily overlooked. The diagnosis is especially difficult in the early stages of the case. The golden rule, never to commit one's self to a diagnosis of hysteria alone (nothing but hysteria) without previously having definitely excluded organic disease, cannot be too forcibly insisted upon. The examination of the optic discs and the condition of the plantar reflex are especially important; for, so far as I know, optic neuritis has never been observed as the result of hysteria alone, and a definite extensor response (the Babinski sign) is indicative of organic disease; it is doubtful, too, if true (sustained) ankle-clonus ever occurs in hysteria alone. In doubtful cases it is usually possible to come to a correct conclusion by a careful and judicial consideration of all the facts and circumstances of the case.

Acute generalised meningitis can usually be distinguished from tumour without much difficulty. It is the less acute and more localised forms of meningitis in which mistakes are likely to be made. In some cases of this kind the diagnosis is impossible, for it is quite common in cases of tuberculous and syphilitic tumours to have more or less associated meningitis (*vide* also p. 223).

General Paralysis of the Insane.—The differential diagnosis of frontal tumour more especially and of general paralysis of the insane is, in some cases, very difficult indeed. I have examined more than one case in which the symptoms seemed to indicate a frontal tumour, but in which the necropsy shewed that the condition was general paralysis; in one of these cases tenderness on firm pressure over the right frontal lobe was a very misleading symptom. In differentiating the two conditions lumbar puncture may help; in general paralysis a more or less well-marked lymphocytosis may be expected; a lymphocytosis may occur in cases of brain tumour, but it is rare. I have recorded a case of general paralysis of the insane in which there was double optic neuritis, and in which for a time the diagnosis was most difficult (12).

Disseminated Sclerosis.—In the early stages of cases in which there is giddiness, it may be vomiting, and symptoms indicative of a chronic, non-vascular, focal lesion in the brain, without the characteristic symptoms of disseminated sclerosis, the diagnosis may be very difficult.

Chronic cerebritis, migraine, trigeminal neuralgia, arteriosclerosis, empyema of the accessory nasal sinuses, dural haematoma, cortical encephalitis, cerebral haemorrhage, thrombosis and *embolism, neurasthenia,* and *hypochondriasis* may also sometimes give rise to difficulty and doubt (Bruns).

Local Diagnosis.—The determination of the exact position of the tumour depends upon the exact nature—the definiteness, so to speak—of the localising symptoms. Slight hemiplegic symptoms, for example, have no very definite localising value; they may merely shew that the tumour is situated on the opposite side of the brain. Again, the false localising symptoms are apt to lead to an erroneous opinion as to the exact position of the new growth. Further, it is by no means very uncommon to have more than one tumour in the same brain.

In trying to determine the exact locality of the new growth, both the positive and the negative symptoms and signs must be taken into account. The positive symptoms are more important than the negative; for even large tumours in the motor area may be unattended with paralysis.

In the future the *x*-ray examination, cranial auscultation (43, 49), and puncture and aspiration through the intact skull will probably be of considerable value in the diagnosis and localisation of intracranial tumours.

The exact significance of the localising or focal symptoms indicative of tumours in special parts has already been fully considered in the article on "Regional Diagnosis of Cerebral Disease."

Pathological Diagnosis.—This can only be approximately arrived at in a certain proportion of cases. Attention should be specially directed to the following points:—The history of the case, whether a new growth has been removed from some other part of the body or not; the pathological tendencies, so to speak, whether tuberculous, syphilitic, and the like, of the patient; the family history; the presence in the body, on the surface or in the internal organs, of associated lesions (tuberculous, syphilitic, carcinomatous) indicative of the nature of the new growth; the age of the patient, certain tumours being more common at certain periods of life.

The "style" of the symptoms and the position of the tumour are in some cases important; for tumours in certain positions are apt to be of a certain pathological character. Tumours on the surface of the brain, for example, are very often syphilitic, sarcomatous, or tuberculous, occasionally carcinomatous. Tumours in the cerebellum are often tuberculous or gliomatous. Tumours which occupy the central regions of the brain are often glioma or glio-sarcoma. Cortical or subcortical tumours in the motor area are suggestive that the tumour is a syphiloma, tuberculoma, sarcoma, or subcortical glioma. Tumours involving the third nerve are usually syphilitic.

The duration is of some importance. Fibromas are often extremely chronic. Gliomas, glio-sarcomas, and tuberculomas not infrequently persist for a long time; but there are many exceptions to this general statement. Cancerous tumours usually pursue a somewhat rapid course. Syphilitic tumours, provided that they are untreated, often advance with great rapidity. The results of treatment may shew whether a tumour is syphilitic or not.

The nature of the new growth may be in some cases ascertained by puncture and aspiration through the intact skull. Lumbar puncture (shewing a lymphocytosis and the Wassermann reaction) may indicate the syphilitic nature of the growth.

Prognosis.—The prognosis in cases of intracranial tumour is always very grave. It varies, of course, in different instances. Syphilitic tumours are much more amenable to drug treatment than other forms of new growth; in syphilitic cases the prognosis is in many cases favourable, provided that the case is properly treated in its early stages, that the vessels are unaffected, that is, that there is no marked degree of syphilitic endarteritis—but it must be remembered that it is by no means always easy to determine this point—and that there is no extensive destruction of the adjacent cerebral tissue. In cases other than syphilitic the prognosis largely depends upon the possibility of removing the tumour by operative procedure.

It is important to remember that in all forms of brain tumour there is a liability to sudden death; this is especially so when the tumour is large, when the intracranial pressure is greatly increased, when there is great ventricular distension, when the tumour is rapidly increasing in size, and when the tumour is situated in the pons or medulla.

Speaking generally, we may say that the prognosis must be guided chiefly by the pathological nature of the tumour; by the effects of drug treatment; by the size and position of the new growth and the possibility of removing it by surgical operation; by the length of time which the symptoms have persisted; by the severity of the symptoms, and the rapidity with which they are progressing, and by the degree of increased intracranial pressure which is present.

Treatment. — In connexion with the treatment of intracranial tumours it is necessary to consider (i.) the *curative* treatment—the possibility of (*a*) curing the new growth by drugs, or (*b*) of completely removing it by surgical procedure; and (ii.) the *palliative* treatment—the relief of the symptoms which are present, and the prevention of injurious results likely to arise, amongst which blindness due to post-neuritic atrophy is the most important.

Drug treatment is very unsatisfactory except in syphilitic cases, in which iodide of potassium must be given in large doses, at least 30 grains three times daily. I have repeatedly seen most striking results obtained by large doses (30 to 100 grains, three times daily) when smaller quantities (5 to 10 grains) had been previously given with no benefit. If the iodide alone does not relieve, mercury, either internally (in the form of corrosive sublimate or grey powder) or by inunction, should be added. I have seen several cases in which there was no improvement under very large doses of the iodide alone, but in which most marked benefit or complete cure resulted after the use of mercurials.

Although iodide of potassium is chiefly useful in syphilitic cases it is sometimes beneficial in other forms of intracranial tumour; and, conversely, the fact that iodide produces benefit in a case of intracranial tumour does not necessarily shew that the new growth is syphilitic.

In tuberculous cases I usually give the drug in smaller doses than in cerebral syphilis—5- to 10-grain doses for an adult three times daily. In tuberculous cases cod-liver oil and other remedies, such as the syrup of the iodide of iron and arsenic, which are useful in other forms of tuberculosis, are sometimes beneficial. In tuberculous cases general hygienic measures (open air and good feeding) are valuable. In at least one case of tuberculous tumour, I have seen apparent arrest of the new growth occur under medicinal and hygienic treatment.

In some cases of sarcoma arsenic seems to restrain the development of the new growth; but I cannot say that I have ever seen any distinct benefit from this remedy in intracranial sarcoma.

Perhaps in the future x-ray treatment may prove to be of use in the treatment of some (malignant) forms of brain tumour.

Surgical Treatment.—Though very brilliant results have been obtained by surgical interference, cases in which an intracranial tumour can be completely and successfully removed, and the patient completely cured, by operation constitute, so far as my experience enables me to judge, a very small proportion of the whole.

The fact that comparatively few cases of intracranial tumour can be successfully removed and the patient cured by operation is due to the following circumstances :—(i.) In very rare and exceptional cases of intracranial tumour there are no symptoms, either general or local. (ii.) In a considerable proportion of cases in which there are general symptoms, which distinctly shew that an intracranial tumour is present, there are no localising symptoms. (iii.) In some cases in which localising symptoms are present these symptoms give an erroneous impression of the position of the tumour. (iv.) In many of the cases in which the exact position of the tumour is clearly demonstrated by definite and distinct localising symptoms, the tumour cannot be completely removed and the patient cured by operation, for one or other of the following reasons :—(*a*) In some cases the position of the tumour precludes successful operative interference. Tumours in the medulla, the pons, the front part of the middle lobe of the cerebellum, the basal ganglia, and in the deeper parts of the centrum ovale, are unsuitable for operative interference. (*b*) In other cases the tumour is so extensive, and infiltrates such a large area of brain tissue, that its complete removal is impossible. Sir Victor Horsley (35) has reported several cases in which the partial removal of large tumours was followed by good results (striking and apparent arrest of growth); but Spiller and Frazier conclude from a review of the literature on the subject that the partial removal of a tumour, especially of a glioma, is a questionable procedure. This, too, is my own experience. (*c*) In other cases there is more than one tumour. Further, an intracranial tumour may be complicated by some other brain lesion, such as advanced disease of the cerebral vessels; a remarkable case in point, in which a cerebral abscess and cerebral tumour were present in the same case, came under my own observation a few years ago. (*d*) In other cases the tumour is malignant in character. (*e*) In some cases the cerebral tumour is complicated by associated lesions in other organs which contra-indicate operative interference.

The most favourable cases for operation are those in which the tumour is encapsulated and situated on the surface of the brain, and cases in which the new growth is an encapsulated sarcoma, a fibroma, an endothelioma, a gumma, or a cyst (not merely a cyst developed in connexion with a glioma—such cysts are less favourable).

These are not merely abstract conclusions; they are based on a large clinical and pathological experience. (See an analysis of cases seen by me during life and examined after death, reported in the *Edinburgh Medical Journal*, 1894.) The cases which have come under my notice since the year 1894 confirm the conclusions arrived at in that communication. Furthermore, and this is perhaps the most important argument, in only two of the cases of intracranial tumour which have come under my notice in which an operation has been performed has a complete cure resulted.

Still, while I am strongly of opinion that the percentage of cases in which an intracranial tumour can be successfully removed and the patient completely cured by operation is very small, it must be remembered (1) that in almost all cases in which the symptoms are not relieved by large doses of iodide of potassium and mercury, the disease proves fatal, and often rapidly fatal, and that in cases of cerebral tumour, especially if there is great increase of the intracranial pressure, there is a considerable risk of sudden death; (2) that many cases have been recorded in which intracranial tumours have been successfully removed and the patients cured by surgical operation; (3) that in a considerable proportion of cases in which the tumour cannot be completely and successfully removed by the surgeon, the operation of trephining is attended with temporary benefit and the relief of symptoms, with marked improvement so far as dimness of vision is concerned, and with the prevention of blindness; and (4) that the diagnosis as to the exact position, size, and pathological nature of an intracranial tumour is often a matter of much uncertainty, and that in some of the cases in which the clinical conditions seem to preclude successful removal (though the reverse is much more likely), it may be possible, when the parts are exposed, to remove the growth successfully.

In cases of intracranial tumour, then, in which iodide of potassium and mercury have been fully and fairly tried and have failed to give relief, an operation, for the removal of the tumour and in the hope of a radical cure, is indicated, provided that the locality of the tumour can be even approximately determined, and provided that there be a reasonable probability that the tumour can be safely reached.

For all these reasons I am, after mature consideration, of opinion that in many cases of intracranial tumour the radical operation should be advised and performed. Further, I am strongly of opinion that, except in syphilitic and perhaps tuberculous cases, the operation should be performed as early as possible—as soon as the tumour can be localised or approximately localised.

The opinions of other physicians and the results obtained by different surgeons vary considerably with regard to the percentage of cases of brain tumour which can be successfully removed by operation. In considering these statistics it must be remembered that whilst a very large proportion of the successful results are certain to be published, a considerable proportion of the unsuccessful cases are probably never recorded.

Seidel calculates that the proportion of operable brain tumours is 3 per cent, whilst Dana puts it as high as 17 per cent. Knapp has collected records of 828 cases operated upon up to 1905. In 471 the tumour was removed; 112 of these patients recovered—frontal 15, central 52, parietal 7, temporal 2, occipital 3, cerebellum 7, not stated 26. A study of the recorded cases which "recovered" leads him to believe that most of these simply lingered on for a time, paralytic, epileptic, or blind; that in many cases the growth recurred; and that the cases of actual recovery, that is of complete restoration to health, are exceedingly few. Harvey Cushing's results are much more satisfactory. In 64 cases of cerebral tumour operated upon during ten months, he performed 84 operations either for palliation, exploration, or removal of the tumour. In 23 of these cases, or 36 per cent, a radical extirpation of the tumour was performed; 16 of these patients (25 per cent of the total 64 cases) recovered with more or less complete restoration of function. In 41 of the 64 cases, or 64 per cent, a palliative operation was performed; of these 41 cases, 5 proved immediately fatal, 15 lived on for varying periods, some with considerable, others with little or no improvement until death supervened, and 21 are still living with symptoms in complete abeyance or so far relieved that most of them have been able to resume their occupations and thus to earn a livelihood. Thus, in the 84 operations on these 64 patients there were 8 fatalities directly attributable to the surgical procedure (an operative mortality of 9·5 per cent); 16 cases after removal and 21 cases after decompression were greatly benefited, and a number were probably cured, making a percentage of 57·8 out of the 64 patients who have regained, temporarily at least, a fair measure of health. Cushing advises the subtemporal region as the most favourable site in cases in which the tumour is not subtentorial and in which it is not complicated by an internal hydrocephalus. He further states that combined exploration and decompression in the subtentorial region have proved of great value in a number of difficult cases.

Palliative Trephining.—Every experienced observer now admits that in many cases in which an intracranial tumour cannot be removed (cured) by surgical interference the operation of trephining is attended with temporary benefit—with relief of the agonising headache and urgent vomiting, diminution or disappearance of the optic neuritis, marked recovery of vision, prevention of blindness, and prolongation of life. Consequently, it must, I think, be allowed that in those cases of intracranial tumour in which the symptoms indicative of increased intracranial pressure are marked, and in which drug treatment and other palliative measures have had a fair trial and have failed to give relief, and in all cases in which the optic neuritis is intense, and especially if vision is beginning to be impaired, trephining as a palliative measure ought to be carried out. Mr. Leslie Paton found that in 30 cases which survived operation in the National Hospital, Queen Square, during the years 1903 and 1904, useful vision was saved in 22. In the remaining 8 cases, 6 were practically blind at the time of the operation and regained no vision; in 2 cases failure of vision took place subsequently in spite of the operation. Cushing advises that where there are no certain localising signs, or in cases in which tumour is believed to be irremovable, a silent area of the brain should be selected over which to trephine so as to limit the paralysis which is sure to follow herniation. When the tumour is subtentorial, palliative trephining should be practised over the posterior fossa.

Lumbar puncture has been suggested as a substitute for palliative trephining, but is to be strongly condemned; for in a considerable number of cases a fatal result has followed immediately or soon after the operation.

Ventricular puncture has also been practised for the relief of increased intracranial pressure The results, as in lumbar puncture, are merely temporary and palliative and appear to be unsatisfactory.

Palliative Medical Measures.—For the relief of pain the application of cold in the form of ice-bags to the head, free watery purgation, the administration of iodide of potassium, phenacetin, and in some cases (though these remedies must be cautiously given) morphine, Indian hemp, croton-chloral, are the most useful remedies.

For the relief of spasms and epileptiform convulsions bromide of potassium and chloral hydrate are the chief remedies. Localised spasms or attacks of Jacksonian epilepsy, due to syphilitic deposits on the surface of the motor area, are best treated by large doses of iodide of potassium. When the epileptiform attacks are very frequent and very severe, bromide of potassium and chloral hydrate may be given with the object of restraining the spasms until the iodide has had time to exert its specific effects.

Apoplectic attacks due to extravasation are to be treated in the same way as ordinary apoplexies. In pseudo-apoplectic attacks the application of cold to the head, blistering, free purgation, or venesection are the best remedial measures. The paralyses caused by intracranial tumours must be treated in the same way as paralyses due to other lesions. Paralysis due to a syphilitic lesion of a nerve-trunk should be treated in the early stages with large doses of iodide of potassium, alone or in combination with mercury.

In all cases of intracranial tumour in which double optic neuritis is present, the administration of iodide of potassium is advisable. Under this treatment the inflammation of the optic papillae is in some instances reduced or altogether removed, and the occurrence of post-neuritic atrophy and blindness thereby prevented. In syphilitic cases I have seen most marked improvement under this treatment, even when the optic neuritis was intense and the patient almost completely blind.

The beneficial effects which the operation of trephining affords, as a palliative means of treatment, for the relief of headache, vomiting, and other urgent symptoms, and for the treatment of optic neuritis and the prevention of blindness which is so apt to result therefrom, have already been insisted upon.

It is unnecessary to refer here to the treatment of bed-sores, cystitis, and the complications and associated lesions which arise in the course of an intracranial tumour.

To sum up: in the treatment of a case of intracranial tumour the first object of treatment is to endeavour to obtain a cure (absorption of the new growth) by internal (drug) treatment. In order to carry this indication into effect the patient should be placed at rest and kept as quiet as possible; everything likely to disturb the intracranial circulation should be avoided; he should be carefully fed on light, nutritious food; the condition of the bowels should be carefully regulated and a brisk purgative occasionally given; and large doses of iodide of potassium should be administered. If the iodide does not give relief, mercury should be added. The second indication is to relieve the symptoms by the various palliative measures (other than trephining) which have been enumerated above, while the curative drug treatment is being employed. The third indication is to endeavour to remove the tumour by surgical operation. The operation should be performed as early as possible, provided that the tumour can be accurately located and the conditions for its removal are satisfactory. In syphilitic and perhaps tuberculous cases, operative procedure should be delayed until the medicinal treatment has been thoroughly tried and has failed to give relief.

And the fourth indication is to endeavour to relieve the symptoms, to prevent blindness and to guard against the risk of sudden death by palliative trephining in those cases in which curative drug treatment has failed, in which the tumour cannot be localised or removed by surgical operation, and in which other palliative measures have been employed without benefit. Palliative trephining should not be long delayed. If iodide of potassium, mercury, and other general measures (drug, dietetic, and hygienic) do not produce marked relief in the course of a few weeks, in cases in which the intracranial pressure is very greatly increased, in other words, in cases in which there is a risk of sudden death, in cases in which vision is beginning to fail, in cases in which the optic neuritis is advanced, the palliative operation should be immediately performed.

Byrom Bramwell.

REFERENCES

1. Armour, D. "Discussion on the Indications for Operation in Cases of Intracranial Tumour," *Brit. Med. Journ.*, 1907, ii. 1127.—2. Auvray, Maurice. *Les Tumeurs cérébrales*, 1896.—3. Batten, F. E., and Collier, J. "On Spinal Cord Changes in Cases of Cerebral Tumour," *Brain*, London, 1899, xxii. 473.—4. Beevor, C. E. "On the Diagnosis and Localisation of Cerebral Tumours," *Lancet*, London, 1907, i. 718.—5. Bennett, A. Hughes. "Cerebral Tumour with Symptoms simulating Hysteria," *Brain*, London, 1878, i. 114.—6. v. Bergmann. *Die chirurgische Behandlung von Hirnkrankheiten*, Berlin, 1889; and *Berlin. klin. Wchnschr.*, 1895, xxxii. 337.—7. Bernhardt. *Beiträge zur Symptomatologie und Diagnostik der Hirngeschwülste*, 1881.—7*a*. Bordley, J. (Jr.), and Cushing, H. "On Alterations in the Colour-Fields in Cases of Brain Tumour," *Arch. Ophth.*, N.Y., 1909, xxxviii. 462.—8. Bramwell, Byrom. "Cases of Intracranial Tumour," *Edin. Med. Journ.*, 1878, xxiv. 141; 1879, xxv. 141.—9. *Idem.* *Intracranial Tumours*, 1888; (causation of optic neuritis), *Intracranial Tumours*, 1888, p. 53.—10. *Idem.* "On the Surgical Treatment of Intracranial Tumours," *Edin. Med. Journ.*, 1894, xxxix. 1067.—11. *Idem.* "On the Localisation of Intracranial Tumours," *Brain*, London, 1899, xxii. 1.—12. *Idem.* "On Errors in Diagnosis," *Clinical Studies*, 1905, iii. 122.—13. *Idem.* *Ibid.*, 182.—14. *Idem.* "On Scrofulous Tumour of Basal Ganglia and Internal Capsule," *ibid.*, 1908, vi. 392.—15. *Idem.* "On the Differential Diagnosis of Intracranial Tumour and Lead Encephalopathy," *ibid.*, 1909, vii. 91.—15*a*. *Idem.* "On Case of Sarcoma of Pituitary Body with Diabetes insipidus," *ibid.*, 1910, viii. 179.—15*b*. *Idem.* *Lancet*, London, 1910, i. 681.—16. Bramwell, Edwin. "On a Case of Large Intracranial Tumour in which the Patient suffered for Four and a Half Years from Epileptic Attacks," *Rev. Neurol. and Psychiat.*, Edin., 1908, vi. 577.—17. Broca et Maubrac. *Traité de chirurgie cérébrale*, Paris, 1896, chap. iii.—18. Bruns, Ludwig. *Die Geschwülste des Nervensystems*, 1897 and 1908.—19. Chipault. *Chirurgie opératoire du système nerveux*, 1894.—20. Collier, J. "On the False Localising Signs of Intracranial Tumour," *Brain*, London, 1904, xxvii. 490.—21. Cushing, H. "On the Establishment of Cerebral Hernia as a Decompressive Measure for Inaccessible Brain Tumours," *Surgery, Gynecol. and Obstetrics*, 1905, i. 297.—21*a*. *Idem.* "Mitchell Banks Memorial Lecture," *Lancet*, London, 1910, i. 90.—22. Dana. Quoted by Walton and Paul, *Journ. Nerv. and Ment. Dis.*, 1905, xxxii. 481.—23. *Idem.* *New York Med. Journ.*, 1905, lxxxi. 270.—24. Erb. "Zur Chirurgie der Hirntumoren," *Deutsche Ztschr. f. Nervenh.*, 1892, ii. 414.—25. Ferrier. *Goulstonian Lectures on Localisation of Cerebral Disease*, 1878; *Croonian Lectures on Cerebral Localisation*, 1890; "Cerebral Localisation and its Practical Relations," *Brain*, London, 1890, xii. 36.—26. Frazier, C. H. *The Eye and Nervous System*, 1906, 885.—27. *Idem.* *Ibid.* 892.—28. *Idem.* *Ibid.* 893.—29. Gowers. Article in vol. ii. *Diseases of the Nervous System*, 1893; *Syphilis and the Nervous System*, 1889.—30. Gunn, M. "On the Localisation of Intracranial Tumours," *Brain*, London, 1898, xxi. 332.—31. Heubner. "Syphilis of the Brain," von Ziemssen's *Cyclopaedia of the Practice of Medicine*, 1877, xii. 293.—32. Holmes, Gordon. "On Certain Tremors in Organic Cerebral Lesions," *Brain*, London, 1904, xxvii. 327.—33. Hoppe, H. H. "On Brain Tumour Symptom-Complex, with Termination in Recovery," *Journ. Nerv. and Ment. Dis.*, 1907, xxxiv. 97.—34. Horsley, Victor. "Brain Surgery," *Brit. Med. Journ.*, 1886, 1887-

1890 ; "On Localisation of Disease of the Cerebrum," *Amer. Journ. Med. Sc.*, Phila., 1887, xciii. 342.—35. *Idem.* "On the Technique of Operations on the Central Nervous System," *Brit. Med. Journ.*, 1906, ii. 411.—36. JACKSON, J. HUGHLINGS. "Syphilitic Affections of the Nervous System," *Journ. Ment. Sc.*, London, 1875. —37. *Idem.* "On Choreal Movements and Cerebellar Rigidity, etc.," *Brit. Med. Journ.*, 1875, i. 636 ; *Brain*, London, 1906, xxix. 441.—38. *Idem.* "On Intellectual Aurae or Intellectual Warnings of Epileptic Seizures," *Med. Times and Gaz.*, London, 1876, ii. 700 ; *Brain*, London, 1880, iii. 192 ; 1888, xi. 179 ; 1889, xii. 346 ; 1898, xxi. 580 ; and *Lancet*, London, 1899, i. 79.—39. KEEN, W. "Cases of Brain Tumour ; Three Operations," *Amer. Journ. Med. Sc.*, Phila., 1894, cvii. 605.—40. KNAPP, P. C. *The Pathology, Diagnosis, and Treatment of Intracranial Growths*, 1891.—41. *Idem.* "On the Mental Symptoms of Cerebral Tumour," *Brain*, London, 1906, xxix. 35.—42. *Idem.* "On the Results of Operation for the Removal of Cerebral Tumours," *Boston Med. and Surg. Journ.*, 1906, cliv. 124.—43. KNAPP, A. "Fortschritte in der Diagnostik der Gehirntumoren," *München. med. Wchnschr.*, 1908, lv. 1003, 1081.—44. KOLLARITS, J. "Hypophysistumoren ohne Akromegalie," *Deutsche Ztschr. f. Nervenh.*, 1905, xxviii. 88.—45. LEJONNE, P. "La compression radiculo-ganglionnaire dans les tumeurs de la moelle," *Encéphale*, 1907, ii. 232.—46. MACEWEN, W. "Surgery of the Brain and Spinal Cord," *Brit. Med. Journ.*, 1888, ii. 302.—47. MARTIN, J. M. "On Analysis of 601 Cases of Intracranial Tumour," *Lancet*, London, 1897, ii. 81. —48. MILLS, C. K. "On the Localisation of Brain Tumours, especially with reference to the Parietal and Prefrontal Regions," *Philadelphia Med. Journ.*, 1901, vii. 478.—49. *Idem.* "On Tumour of the Brain localised clinically and by the Röntgen Rays," *Phila. Med. Journ.*, 1902, ix. 268.—50. MILLS, C. K., and LLOYD, J. K. "Tumours of the Brain and its Envelopes," Pepper's *System of Medicine*, 1886, v. 1028.—51. NONNE, M. "Über Fälle vom Symptomenkomplex 'Tumour Cerebri' mit Ausgang in Heilung (Pseudotumor Cerebri). Über letal verlaufene Fälle von 'Pseudotumor Cerebri' mit Sektionsbefund," *Deutsche Ztschr. f. Nervenh.*, 1904, xxvii. 169.—52. OBERNIER. "Tumours of the Brain and its Membranes," von Ziemssen's *Cyclopaedia of the Practice of Medicine*, 1877, xii. 231.—53. OPPENHEIM, H. "Die Geschwülste des Gehirnes," Nothnagel's *Specielle Pathologie und Therapie*, 1896.—54. *Idem.* *Beiträge zur Diagnostik und Therapie der Geschwülste im Bereich des Zentralen Nervensystems*, S. 193. Mit 20 Abbildungen im Text und 6 Tafeln. Verlag von S. Karger, Berlin, 1907.—55. OPPENHEIM und BORCHARDT. "Über zwei mit Erfolge operierte Fälle von Geschwulst am Kleinhirnbrückenwinkel," *Berlin. klin. Wchnschr.*, 1907, xliv. 875.—56. PATON, L. "On the Course of Optic Neuritis and its Subsidence after Operation in Cases of Cerebral Tumour," *Rev. Neurol. and Psychiat.*, Edin., 1905, iii. 541.—57. PEITAVY. *Contribution à l'étude des tumeurs cérébrales.* Thèse de Paris, 1893.—58. PRESTON. "Tumour of the Cerebellum, with Report of Cases," *Journ. Nerv. and Ment. Dis.*, 1892, xvii. 257.—59. RANSOM, W. B. "Tumours of the Corpus Callosum," *Brain*, London, 1895, xviii. 531.—60. RAYMOND. "Sur deux cas de tumeurs du corps calleux, avec autopsie," *Rev. neurol.*, Paris, 1906, xiv. 772.—61. REDLICH, E. "Über diffuse Hirnrindenveränderungen bei Hirntumoren," *Arb. a. d. neurol. Inst. a. d. Wien. Univ.*, Leipz. u. Wien, 1908, xv. 320.—62. ROBERTSON, A. "On Skull Percussion," *Lancet*, London, 1893, . 14.—63. SACHS. Article in the *Nervous Diseases of Children*, New York, 1895, and in *Twentieth Century Practice of Medicine*, 1897, vol. x.—64. SAENGER, A. "Über die Palliativtrepanation bei inoperablen Hirntumoren zur Vermeidung drohender Verblindung," *Klin. Monatsbl. f. Augenh.*, 1907.—65. SCHÄFER, E. A. "On Experiments on the Physiological Action of Pituitary Extracts," *Phil. Trans. Roy. Soc. London*, 1907, Ser. B., cxcix. 1-29.—66. SCHÖNTHAL. "Beitrag zur Symptomatologie der Hirntumoren," *Berlin. klin. Wchnschr.*, 1891, xxvii. 254.—67. SCHULTZE. "Zur Diagnostik der Operabilität der Hirn- und Rückenmarkstumoren und über Operationserfolge bei denselben," *Mitt. a. d. Grenzgeb. d. Med. u. Chir.*, Jena, 1907, xvii. 613.—68. SEIDEL. Quoted by Walton and Paul, *Journ. Nerv. and Ment. Dis.*, 1905, xxxii. 481.—69. SPILLER and FRAZIER. "On Palliative Operation in the Treatment of Tumours of the Brain, based on the Observation of Fourteen Cases," *Univ. Penna. Med. Bull.*, Phila., 1906, xix. 146.—70. STARR, M. ALLEN. Articles on Brain Tumours in Keating's *Cyclopaedia of the Diseases of Children*, iv. 51 ; in Dercum's *Diseases of the Nervous System*, and *Brain Surgery*, New York, 1895, 470.—71. *Idem.* "On the Results of Surgical Treatment of Brain Tumours," *Journ. of Nerv. and Ment. Dis.*, 1903, xxx. 398.—72. STEWART, PURVES. "On Four

Cases of Tumour in the Region of the Hypophysis Cerebri," *Rev. Neurol. and Psychiat.*, Edin., 1909, vii. 225.—73. Stewart, T. Grainger. "On the Diagnosis and Localisation of Tumours of the Frontal Regions of the Brain," *Lancet*, 1906, ii. 1209.—74. Stewart, T. G., and Gibson, G. A. "Cases of Cerebellar Disease," *Edin. Hosp. Rep.*, 1898, v. 173.—75. Taylor. "Optic Neuritis in Relation to Intracranial Tumours," *Trans. Oph. Soc.*, 1894.—76. Vignol. "Diagnostic des tumeurs du cervelet," *Arch. de méd. et de pharm.*, 1894, xxiii. 413.—77. Walton and Paul. "On Brain Tumours, a Study of Clinical and Post-mortem Records bearing on their Operability and their Symptomatology," *Journ. Nerv. and Ment. Dis.*, 1905, xxxii. 481.—78. Weber, R., et Papadaki, A. "De quelques altérations du tissu cérébral dues à la présence de tumeurs," *Nouv. icon. de la Salpêtrière*, 1905, xviii. 140.—79. White, W. Hale. "Analysis of One Hundred Cases of Cerebral Tumour," *Guy's Hosp. Rep.*, 1886, xliii. 117.

B. B.

OCCLUSION OF THE CEREBRAL VESSELS

By James Taylor, M.D., F.R.C.P.

Introductory.—The blocking of cerebral vessels is of importance because of the effects thus secondarily produced upon the nervous structures which they serve. By any vascular occlusion the nutrition of the area of the brain related to the affected vessel is seriously interfered with, and its function impaired or abolished. This effect may be temporary; more usually it is permanent, whether the vessel occluded be a cerebral artery, vein, or sinus. The effect of such an occlusion on other parts of the body depends on the part of the brain affected; and the character of the symptoms in any particular case is determined by the situation and extent of the area from which the blood-supply is cut off, and by the nature of the process by which the occlusion is effected. Blocking of vessels, as it occurs in the brain, may be divided into three classes:—I. blocking of an artery by an embolus, usually a piece of fibrin, non-infective or infective, carried in the blood-stream from the heart or some part nearer to it; II. thrombosis in an artery; III. thrombosis in a vein or sinus.

I. Cerebral Embolism.—The most common source of an embolus, which finds its way to the brain, is a diseased cardiac valve. The accident does not happen frequently in the first acute inflammation which affects such a valve; it is more commonly the result of a second attack of endocarditis, affecting a part already roughened by old disease. From such a valve some loosely attached piece of fibrin may easily be separated and carried away in the blood-stream. By far the commonest form of cardiac disease which gives rise to embolism is mitral stenosis; and it will be readily understood how any piece of fibrin detached from the mitral valve may find its way into a cerebral vessel. Aortic valvular disease is a comparatively rare source of embolism, though occasionally it does give rise to it. An embolus may also originate from clotting in the left auricle. The condition likely to give rise to such an occurrence usually depends upon a loss of compensation in mitral disease, leading to a condition in which the auricle is never completely emptied. If, furthermore, a rheumatic condition be superadded, with a consequent state of the blood which renders it prone to coagulate, it will be seen how easily a clot may form, and how a piece of such clot may be washed into the circulation and so occlude a cerebral vessel. The state of the blood during pregnancy and the puerperium, and also during lactation, is such as to render it apt to coagulate; and embolism, the result of clotting in the left auricle, is usually the cause of the hemiplegia which takes place in any of these conditions. Similarly, an aneurysm of any of the large vessels at the base of the heart may be the source of a clot which blocks a cerebral artery. A diseased or roughened vessel-wall may likewise give rise to the formation of a clot upon it. Such a clot, or a portion of it, may be small, and narrow the lumen without obstructing it; but it may be swept into the blood-stream, and lead to the complete occlusion of a smaller vessel more distant from the heart. In certain diseased conditions of the pulmonary vein a clot may pass from it into the left auricle, and thence into the circulation; and in morbid conditions of the lung, particles, possibly infective, may pass into the circulation through the pulmonary vein and its branches, and reach the brain, causing not only occlusion of a vessel, but also, if the particle be infective, meningitis (*e.g.* tuberculous) or abscess. It is by such a mechanism, no doubt, that old-standing empyema is so apt to give rise to cerebral abscess. And with reference to the other embolic conditions enumerated, it will be understood that if the source of the embolus be an infective one—if, for example, the endocarditis be of the infective variety—the embolus from such a source may, and often does, set up changes at the spot at which it lodges in the brain, changes which depend on its infective character, and are quite ndependent of the usual mechanical consequences. It has also been said that an inflammatory focus in the pelvis may give rise to embolism; it is unlikely, however, that any particle large enough to cause discoverable mechanical obstruction in a cerebral vessel could traverse the pulmonary capillaries uncaught. Yet in certain cases a patent foramen ovale may permit of cerebral embolism, even if the clot come originally from the venous system (*vide* "Paradoxical Embolism," Vol. VI. p. 765). It is also possible that minute organisms might traverse the pulmonary capillaries which are larger than the cerebral; but in such a case any ubsequent cerebral symptoms would be regarded, not as the result of the slight mechanical obstruction, but rather of the infection which is associated with it. In cases in which symptoms of cerebral vascular occlusion occur in association with pelvic inflammation, these are possibly he effect of an embolus detached from a secondarily inflamed cardiac valve; or of thrombosis resulting from one of the events to be enumerated presently.

The local effects of embolism of a cerebral vessel are, in the first place, a local anaemia in the parts supplied by the vessel. There is also oedema of the surrounding area; and this probably gives rise to impairment of the function of parts of the brain not supplied by the blocked vessel: thus the extent and degree of paralysis present in the early stage of embolism are as a rule considerably greater than turns out to be the case after this temporary oedema has subsided. But the blocking of the artery leads also to a degree of venous engorgement and consequent slight rupture of small vessels around the spot, constituting the condition which is known as *red softening*. It is possible, indeed, so far as clinical evidence can inform us, it seems certain, that in some cases, probably those only in which a small vessel is blocked, the collateral circulation is such as altogether to prevent any permanent impairment of the nutrition of the part supplied by the vessel which is blocked; for in a number of cases recovery from the paralysis which follows embolism is both rapid and complete. In other cases, however, recovery is but partial; and in them changes take place in the softened and venously engorged area which ultimately lead to the formation of a cyst,—the contents of which are fluid and contain blood-crystals, or to the formation of a patch of *yellow softening*. In the description of the clinical consequences of embolism and thrombosis it will be pointed out how frequently, especially in young people with vessels otherwise healthy, recovery takes place in the former to a very considerable degree, or even completely, offering a marked contrast to the usually permanent effects of thrombosis. In the latter case the failure to restore nutrition by collateral circulation is probably to be explained by the nature of the process, and by the degenerated and diseased condition of the vessels by which such collateral circulation would have to be effected.

But from embolism local changes in the vessel which is blocked may also result. If the plug be of an infective or irritating character, its lodgment may lead to inflammatory changes in the vessel-wall itself, and consequent extension to other vascular areas or even to other vessels. Again, on the side of the embolus proximal to the heart, more especially if the vessel be atheromatous, its coat may yield, and an aneurysm may be formed: in some cases of embolic hemiplegia ending in sudden death months after the onset of the paralysis, the rupture of such an aneurysm has been recognised as the cause of death (*vide* also p. 315).

It has been commonly stated that embolism is more likely to occur on the left side of the brain, on account of the greater facility with which an embolus may enter the carotid of that side. Yet this tendency to more frequent affection of the left side is not very well marked, for in 639 cases specially investigated as to this point, 307 were on the right and 332 on the left side (E. Jones (10*a*)). The middle cerebral artery, in its trunk or one of its branches, is the vessel most liable to be affected. The internal carotid itself may be blocked; and the order of affection of the other arteries is—posterior cerebral, anterior cerebral, basilar, and cerebellar arteries. The angle at which the branches from the basilar come off makes the occurrence of embolism in them unlikely.

An embolus is most apt to lodge where a vessel bifurcates, and where accordingly a diminution in lumen renders the further passage of clot impossible.

The *symptoms* of embolism are the sudden paralysis, its extent and degree depending on the vessel affected. But, as has already been said, the degree of paralysis in every case of embolism is probably more severe at first than it is subsequently, on account of the temporary impairment of a large area by the conditions consequent on the blocking of the artery. Consciousness may or may not be lost. In the majority of the cases it is lost; and in those in which it is not affected it will probably be found that the vessel blocked is minute, and that the paralysis is slight and probably temporary. It is rarely that the profound coma attending haemorrhage is present in cases of embolism; nor, unless the embolus be infective, are there the febrile indications which accompany the former condition. Convulsions may occur if the vessel blocked be a cortical one. Of course the great indication for differential diagnosis is to be found in the presence or absence of some condition likely to give rise to embolism; although it must also be borne in mind that the presence of such a condition does not exclude the possibility either of haemorrhage or thrombosis. The clinical features of the paralysis arising from embolism will be considered in detail along with those resulting from thrombosis.

II. Vascular Occlusion from Thrombosis in an Artery.—The two factors determining this condition are:—(i.) diseased vessels, and (ii.) morbid states of the blood.

(i.) *Diseased Vessels.*—The most common disease in vessels—atheroma—is that which is present as a causal condition in most cases of thrombosis. Atheroma is, of course, a senile or degenerative change. It produces roughening in the vessel-wall, and on any injury of the endothelium coagulation may take place, and thus start the process of occlusion. This mechanism may become effective only in conditions in which the blood-stream is very slow, or the blood, on account of some alteration in it, has become viscid or more ready to coagulate than usual. The smaller cerebral arteries are peculiarly liable to be affected with atheroma; and the point at which clotting starts is often at the emergence of a branch from an artery, the lumen of the smaller vessel having become narrowed by atheroma just at this point.

(ii.) Besides the disease in the vessel-wall, *the state of the blood* is of importance. Various conditions render it apt to coagulate. In old people it would seem as if great or unusual exertion might have this effect; for one of the commonest conditions in which thrombosis occurs is during the profound sleep which follows unusual fatigue in persons advanced in years. Chlorosis is another condition in which there is great liability to coagulation; but in this condition the clotting not infrequently takes place in the veins or sinuses (*vide* Vol. V. p. 714). During pregnancy, during the puerperium and apparently after this has passed but while lactation is still going on, clotting may occur; the blood states peculiar to gout and diabetes also favour this change. Although after enteric fever the veins of the limbs are frequently the seat of coagulation, it may also occur in the cerebral vessels—in arteries as well as veins (*vide* Vol. VI. p. 728). After diphtheria, also, it is not very uncommon to see hemiplegia resulting from vascular obstruction of a cerebral vessel; and this depends, probably, on the blood state with consequent thrombosis; or it may result, as some believe, from an embolism coming from a diseased valve, the result of associated endocarditis. Yet in 6 cases of this condition of hemiplegia following diphtheria seen by me, no evidence of any morbid cardiac condition could be detected. Further, any condition or disease which very greatly lowers the general strength may lead to a state in which thrombosis may readily occur; and it must be remembered that the blocking of cerebral veins may, so far as the clinical condition is concerned, lead to results practically identical with those of arterial occlusion.

The result, then, of vascular occlusion occurring in any such way is to lead to very grave interference with the nutrition of the related part of the brain. It is probable that oedema does not occur in anything like the degree in which it does with embolism, on account of the condition of the blood and of the vessel-wall. And in thrombosis there is not the same capacity for collateral circulation, because the diseased vessel is probably in the centre of a district all the vessels of which are similarly affected; so that the recovery from thrombosis, which occurs as the result of atheroma in patients fairly advanced in life, is never so substantial as it may be from embolism occurring in young people. Besides the oedema, which may result, there is also venous turgescence, and the consequent appearance of so-called *red softening*. This red softening usually undergoes certain changes, during which the colour becomes more and more pale until it turns to *yellow softening*. But in some instances a more widespread change takes place, especially if a large cerebral vessel has become blocked; in these, large yellow patches —the *plaques jaunes* of the French—are scattered throughout the affected area, and the whole, although softer than usual, is not in the diffluent condition which is the usual result of local thrombosis.

Besides thrombosis arising in connexion with atheromatous or other degenerative conditions of blood-vessels, we have also to consider a by no means uncommon result of syphilis, occurring in what is known as the tertiary stage. By such a period no particular time after infection can be indicated, but a stage at which, among other structures, blood-vessels are affected. As one of the phenomena of this stage changes occur in these structures, which result in thickening of the vessel-wall, and obliteration, either complete or partial, of the lumen. The change consists essentially in proliferation of the intima, and, associated with this, there is usually some thickening of the adventitia also. The essential features of the process are considered elsewhere ("Disease of Arteries," Vol. VI. p. 462); the process, as it concerns us in this place, is narrowing of the lumen, and consequent limitation (it may be entire cutting off) of the blood-supply of a certain area. The effect is interference with the function of the limb or limbs related to the area of the brain affected; and while the usual result is hemiplegia, from a blocking of a vessel on one side of the brain, there may, if the basilar be affected, be interference with the functions of all four limbs, of the facial muscles, and of the parts subserved by the other cranial nerves. In short, the cerebral conditions resulting from occlusion of a vessel or vessels affected with syphilitic endarteritis are not to be distinguished from the results of haemorrhage, of embolism, or of thrombosis resulting from atheroma; unless it be by a consideration of the mode of onset, the extent of the affection, the age of the patient, the history, and the condition of other viscera.

In many cases of so-called syphilitic hemiplegia the onset of the paralysis is sudden. It is sometimes preceded for some days, or longer, by severe continuous or paroxysmal headache, or by symptoms of general but indefinite illness, not uncommonly with mental symptoms. Convulsion may also precede the paralysis, and this is sometimes unilateral. The paralysis, as has been stated, is usually sudden in its onset, and frequently accompanied by coma which may last for days, and, indeed, may not be recovered from. There may be evidence of syphilitic processes elsewhere, such as gumma in the liver, nodes on the bones, and such like; and not uncommonly there is marked thickening and hardening of the coats of the radial arteries so that they feel like cords under the finger. The victims of the condition are usually under forty, and it is often impossible to obtain a definite history of syphilis. But given a patient under forty, in whom there is no evidence of cardiac or renal disease, or any other condition likely to give rise to blocking or rupture of vessels, we are justified, even in the absence of any confirmatory history, in assuming, for purposes of treatment at least, that the condition is the result of syphilitic arterial disease. Wassermann's reaction also might be resorted to in such a case. A positive result would of course be confirmatory, but a negative one would not necessarily prove that the case was not syphilitic. Unless such a case is met with in the early stage, and submitted at once to energetic antisyphilitic treatment, the hemiplegia is likely to be persistent. When occlusion of an artery sufficiently large to cause hemiplegia has taken place, necrotic changes are as apt to occur in the area supplied by the artery as in any other condition leading to complete blocking. And it may be that much less is to be hoped for from the collateral circulation than in cases of embolism, because it is probable that the other arteries in the vicinity are similarly affected, if not quite so severely. If, however, the patient is at once submitted to energetic treatment, with iodides and mercury, the thickening of the walls of the wholly or partially occluded vessel may be resolved to the extent of permitting the re-establishment of the circulation through it; or perhaps the walls of the neighbouring vessels may be so influenced as to enable them to assume the collateral circulation. If such a happy result be not obtained, then the affected area undergoes necrotic changes similar in character to those following the other kinds of vascular occlusion already described.

Arteries or veins may be blocked as a result of a blood state such as we find present, for example, in rheumatic conditions, in diabetes, in enteric fever, and in chlorosis. In these conditions the vessel occluded may be an artery or a vein; in the two former usually an artery, in the two latter perhaps more often a vein: the condition will be referred to in dealing with the occlusion of veins and sinuses (p. 303).

In all forms of vascular occlusion occurring in the brain there is danger of death at the onset; and in many cases this actually occurs, the patient becoming comatose, Cheyne-Stokes respiration perhaps coming on, and death supervening in the course of a few days or even hours. This is particularly true of cases in which the occlusion affects vessels, such as the basilar, which subserve bilateral functions; or those most nearly associated with the vital centres, such as the vertebral: and it may be stated generally that occlusion of a vessel at the base of the brain is always of more serious significance than occlusion of a vessel over the convexity, or of one supplying the subjacent white matter. And in any paralytic condition following supposed vascular occlusion, it is always important to examine into the condition of the cranial nerves, as a definite affection of one of these may furnish important evidence as to the vessel occluded and, through that, to the likelihood of recovery. Such evidence is not infrequently to be met with; for instance, in cases of syphilitic disease of the basilar artery.

The usual result, however, of the occlusion of a cerebral vessel is hemiplegia, uncomplicated with any affection of cranial nerves or nuclei. The initial condition as regards paralysis may be the same, whether the vessel be large or small. In many cases, in two or three weeks, when the effects of the first attack are seen to be passing off, symptoms suggesting an extension of the original mischief, or some secondary effects of it, may take place, and lead to a fatal issue. This is especially true of cases of thrombosis in patients who are the subjects of senile changes in the arteries.

The character of the paralysis resulting from vascular occlusion will naturally depend upon the function of the parts supplied by the occluded vessel or vessels. The ordinary form of paralysis is *hemiplegia*, in which there is weakness of one side of the face and trunk, and of one arm and leg. The whole of one side of the face is usually affected, although the weakness is more obvious in the lower part. The weakness of the affected side of the trunk is not, as a rule, very marked, although it may be distinct enough. The arm, in the great majority of cases, is more paralysed than the leg; and the explanation of these peculiarities is to be found in the hypothesis advanced, in 1866, by the late Sir William Broadbent, and usually known as Broadbent's hypothesis. The hypothesis is that bilaterally associated movements are represented on both sides of the brain, and that the closer the bilateral association the more nearly equal is the representation on the two sides of the brain. Thus, it is well known that the two lower limbs are much more closely associated in their movements than the two upper. One arm is very frequently used quite independently of the other, whilst a movement of one leg without some movement of the other is comparatively uncommon; and in the bilateral use of the lower limbs for the purposes of locomotion the association is a very close one. The same is true, even to a greater degree, of the movements of the two sides of the trunk: for it is impossible to move voluntarily one side of the chest or abdomen without moving the other. Facial movements again are nearly always bilateral; the movements of the two sides of the forehead especially being very closely associated. The orbiculares palpebrarum are less closely associated, although some people find it impossible to shut one eye without shutting the other. Of the parts about the mouth the association is still close, although less so; much closer, however, than that between the two legs, and still more close than that between the two arms. If, for example, we could suppose a vessel occluded in such a position as to affect the face, arm, leg, and trunk areas or fibres equally, the arm on the opposite side would be most affected, the leg less affected, the face still less so, and the trunk scarcely at all—in any obvious way. Similar considerations explain the phenomena of the condition known under the name of pseudo-bulbar paralysis, or double hemiplegia; a condition in some ways resembling bulbar paralysis, but really resulting from cerebral lesions—haemorrhage or vascular occlusion—affecting both sides of the brain (*vide* p. 106). The condition resembles bulbar paralysis in that swallowing and articulation are affected. The movements subserving these actions are strong in their bilateral association, and consequently, according to Broadbent's hypothesis, in their bilateral representation in the brain; so that a lesion of one side, if it affect them in any noticeable degree, does so but temporarily. If, however, in addition to the weakness caused by a unilateral lesion, we have the weakness produced by a second lesion on the opposite side, it will be understood at once that considerable interference with these actions is not only likely, but almost inevitable; so that with such a double lesion we have a condition similar, in many respects, to that produced by true bulbar paralysis.

Returning now to the usual form of paralysis resulting from vascular rupture or vascular occlusion,—namely, paralysis of one side of the face and trunk, and of the arm and leg on the same side,—such a paralysis may be purely motor. If, however, the area subserved by the posterior cerebral artery be affected, anaesthesia of the same side of the body may be present with affection of the special senses also on this side; the result of a lesion affecting the posterior part of the internal capsule, the "sensory crossway." In such a condition there may be, and indeed there is apt to be, an affection of the fibres which subserve the two halves of the retinae on the same side as the lesion (therefore the opposite halves of the fields of vision), causing *homonymous hemianopsia*. In such a condition the leg is likely to suffer more than the arm; as the leg fibres in the internal capsule are placed far back, and lie next to the sensory fibres. A similar condition, as regards vision, may also arise from occlusion of a vessel supplying the occipital lobe, and especially the cuneus; and in such a condition, as well as in that resulting from a lesion of the fibres of the optic radiation, there is usually, in addition to the hemianopsia, distinct peripheral restriction of the parts of the field in which vision is preserved; more particularly of that which is on the side opposite to the lesion.

In cases of lesion on the left side of the brain, speech is likely to be interfered with, so as to result in some form of *aphasia* (*q.v.* p. 385); and in left-handed patients a right-sided lesion of the brain may give rise to this defect, whilst a left-sided lesion in such patients may not have aphasia as one of its results. Dr. Hughlings Jackson has also pointed out that in cases of right hemiplegia in which the patient is not left-handed, and in which there is no aphasia, the leg suffers, as a rule, more than the arm; and the converse is also true that in cases of right hemiplegia, in which the leg suffers more than the arm, aphasia is not so likely to result. The reason for this lies, of course, in the considerable area intervening between the leg area or fibres and the speech area or fibres, as compared with the small space which separates the hand and arm centre and fibres from those subserving speech.

Besides the actual powerlessness which is present in the paralysed limb in hemiplegia, there is usually a considerable degree of *rigidity* also. Three kinds of rigidity are described: (*a*) initial rigidity occurring at the onset of the lesion and ascribed to irritation; (*b*) secondary rigidity which comes on after the lapse of weeks, or it may be of months; and (*c*) permanent contracture, in which condition structural changes have taken place in muscles, and perhaps in joints. This is really a natural consequence of secondary rigidity. The rigidity is a varying condition; that is, it is not always equally well marked. Often in the early morning the limbs are less stiff; and one curious phenomenon is that frequently, while the patient yawns, the hand or arm, at other times rigidly contracted, opens or relaxes, and becomes momentarily limp. The explanation of the rigidity is still obscure. Dr. Hughlings Jackson's view that it is the result of unantagonised cerebellar influx is the most feasible explanation yet offered; although it is difficult on such a hypothesis to understand why it should not be established from the very beginning, when the cerebral influence is cut off (*vide* art. on Spasm, Vol. VII. p. 321).

Along with this rigidity there is increased activity of the *deep reflexes.* The knee-jerk is unusually active, not only on the side paralysed, but also on the other. It is really more correct to speak of both sides of the body being weakened, one considerably more so than the other; and thus it is also with the knee-jerks; both are increased, that on the weaker side more than the other. Often a rectus-clonus can also be obtained, and usually a foot-clonus,—at any rate, on the paralysed side. There is also increased activity in the wrist- and elbow-reflexes, and often also in the jaw-jerk. The *superficial reflexes* on the paralysed side are usually diminished in activity, but that from the sole is altered in character. Stroking the sole normally causes flexion of the toes, but in hemiplegia and indeed in any condition in which the pyramidal fibres are sclerosed, stroking the sole produces an extension or dorsi-flexion of the big toe; the other toes may be flexed (Babinski's sign). At first there is often some difficulty with the bladder—usually a difficulty in passing water, so that retention may result, and, unless this is relieved, overflow incontinence. This, however, as a rule, is a transient phenomenon. The bowels are usually constipated, but there is rarely any incontinence of faeces. There may also be difficulty at first in swallowing, and some interference with articulation; but, except in cases in which there is a lesion on both sides of the brain, these difficulties are usually temporary.

Such are the conditions met with when a vessel is either occluded or ruptured in the cortex, under the cortex, or in the vicinity of the internal capsule. When the lesion affects the crus cerebri on one side, there is induced not only the hemiplegic condition described, but also an affection of the third nerve on the side opposite to the hemiplegia. The affection may be complete, involving the ocular movements, the levator palpebrarum, and the pupil; and depends upon a simultaneous affection of the third nerve on the one side and the motor tract on the same side, but of course subserving the limbs of the opposite side. If the diseased vessels be those supplying the upper part of the pons, the hemiplegia is of the usual type—face, arm, and leg—all on the side opposite to the lesion. If, however, the affected area be the lower part of the pons—that is, below the point where the tract subserving facial movement crosses to its nucleus—the paralysis is of another type, crossed or alternate hemiplegia; the face being affected on the same side as the lesion, the limbs on the opposite side. With such a lesion the external rectus muscle, on the same side as the lesion, is also liable to be paralysed. Similarly, there may be wasting and weakness of the tongue on one side, and of the limbs on the opposite side, from a lesion in the vicinity of the hypoglossal nucleus of one side; but such a case of crossed hemiplegia is exceedingly rare.

Certain conditions occasionally present in cases of hemiplegia should be mentioned. Among these is that form of mobile spasm, also named athetosis, which usually occurs in association with the hemiplegia of early life. Fuller reference is made to it in the article dealing with the cerebral palsies of children (p. 464). Although, as a rule, it occurs in such cases, it is occasionally met with in cases in which the onset has been later, especially in such as have been determined, apparently, by injury. In such cases the spasm may be of such extreme violence as to make it necessary to control the affected parts forcibly. In nearly all the cases of athetosis examined after death the apparent determining lesion has been below the cortex; but in some cases in which both sides are attacked with the spasm the time and mode of onset are such as to suggest some injury during birth, such as usually results in submeningeal haemorrhage. It is also noteworthy that in one case, in which a part of the cortex was removed, the spasm ceased, and remained absent during the time the patient lived. In another case, however, the spasm, after being in abeyance a little time, recurred, although not with such severity as before.

KING'S COLLEGE HOSPITAL
MEDICAL SCHOOL.

It sometimes happens that much wasting of muscles occurs on the hemiplegic side. Such wasting is, for the most part, the result of inactivity. In cases of hemiplegia occurring in early life the dwarfing of the affected side is not to be regarded as the result of wasting, but of interference with development. In the cases in adult life, however, the wasting is often well marked, more so in left hemiplegia ; and is usually associated with joint changes, probably trophic in character. The shoulder-joint is especially prone to suffer, and almost complete immobility of this joint is not uncommon. If no further change have taken place the electrical reactions are found to be unaltered. Sometimes, however, the cords of the brachial plexus are involved in the changes round the joint ; in which case we have the usual symptoms characterising nerve inflammation—neuritis—namely, wasting, tenderness of nerve-trunks, loss or impairment of electrical reactions, perhaps the true reaction of degeneration, and glossy skin—a condition of brachial neuritis. Such a condition is often extremely painful, and in many cases must be regarded as of ominous significance.

Another phenomenon, which must also be regarded as of grave import, is the acute sloughing bed-sore which occurs on the paralysed side in certain cases of hemiplegia. It is commonly known as *decubitus acutus*, and, as a rule, does not come on until after the paralysis has been established at least a few days. This occurs unexpectedly, and usually is first seen as a reddish spot over the sacrum, the great trochanter, the outer side of the tibia, or the heel. In spite even of the greatest care rapid necrosis may take place ; and, when the slough has separated, it is seen that the sore is not merely superficial, but that the deeper parts are affected, it may be down even to the bone. Convulsions, accompanied with loss of consciousness, are also of occasional occurrence after hemiplegia. These are doubtless the result of the irritation caused by the initial lesion. Although, as has been stated, convulsion is of occasional occurrence in connexion with the onset of embolic hemiplegia, it seems to be of very rare incidence as a later phenomenon in this condition. In some cases in which such convulsion occurs as a post-hemiplegic phenomenon there has been evidence of concomitant kidney disease, so that the attacks may be determined to some extent by a toxic blood state. But it is probable that in the great majority of cases the exciting cause of the convulsions is an unstable condition of the cells in the vicinity of a cortical or subcortical lesion.

In certain cases of hemiplegia mental derangement is a grave symptom. This derangement may be of the nature of acute maniacal excitement, and by some observers such a condition is considered to be strongly suggestive of cortical lesion. In other cases the mental peculiarity may be very slight, and manifest itself only as emotional instability—an undue readiness to laugh or to cry. This condition is more likely to occur in cases in which the lesion is a basal one, or a double one. Between those two extreme conditions varying degrees of mental disturbance may be present. If this be considerable it adds materially to the gravity of the condition; not only because the initial lesion is probably severe, but also because of the additional difficulties in feeding and general management.

Treatment.—The treatment of these forms of cerebral occlusion is naturally best considered as two separate problems—A. The treatment at the onset of the condition, which is directed to limit the extent of the morbid state, and to counteract the severe and perhaps threatening symptoms which it may have produced; B. The treatment to be given to the parts of the body whose function may have been impaired by the cerebral lesion; with the object of restoring their function as far as possible, and of preventing the contractures and after-deformities which are wont to arise.

A. **Treatment at the Onset.**—This must depend upon the diagnosis which has been made. If the case be one in which, relying upon the data already given, the diagnosis of embolism has been made, the treatment which suggests itself is that of almost complete rest; both in order to minimise the danger attending the presence of a block in a cerebral vessel which has already taken place, and to obviate the danger of another embolus becoming detached. If the patient be unconscious, even greater care is needed, in order to prevent any trophic disturbance, or such danger as is apt to arise from a paralytic condition of the sphincters. Retention of urine is very apt to occur, and must be guarded against, if necessary, by the passing of a catheter. The patient will have to be fed regularly with liquid food, chiefly milk and beef-tea, with the addition of alcohol if necessary. When consciousness has been restored, a gradual increase in the quantity and character of the dietary should be ordered, but digestive troubles must be avoided with the greatest care. The bowels will be kept open, of course, but anything like violent purgation must be carefully avoided. If any acute cardiac disorder be present, or if the circulation be deranged in any way whatever, through loss of compensation or otherwise, regard must be had to this in the line of treatment adopted. If the headache be severe, as occasionally it is, a blister, or, still better, a leech, applied behind the ear, or to the temple on the side of the lesion, will often afford great relief.

If the lesion appear to be thrombosis, the treatment, as regards rest, light easily-digested food, and attention to the state of the bladder, will differ in no essential particular from that recommended for embolism. If there be no reason to suspect syphilis, then the condition is one of clotting occurring in an atheromatous artery; and the three factors which may lead to such a condition have to be borne in mind—the state of the vessel with its roughened wall, the state of the blood with its undue tendency to coagulate, and a weak heart. It is probable that in many cases the indiscriminate use of strong, or at any rate active, purgation in all cases of hemiplegia does infinite harm. It is no doubt desirable to act quickly and smartly on the bowels in order to get rid, if possible, of any toxic influence of the blood which may be present; but it must be borne in mind that, before removing fluid and so perhaps rendering the blood

LIBRARY

more viscous, a fluid of a bland character should be administered, and in considerable quantity; and along with it stimulants—alcohol and some form of strychnine—are of the highest importance. Often also the combination with them of nitroglycerin (in one-drop doses of the 1 per cent solution) seems to intensify the action of the strychnine, possibly by giving it more rapid and complete access to the part which it affects. When the immediate danger of any extension of the clotting has been removed by these means, the patient must still rest, have light food frequently administered, and the bowels carefully regulated. Not uncommonly, after a few days, there is evidence of a slight increase in the paralysis, and such a condition is always attended with anxiety. Death has been known to follow such an extension occurring about the third day, and again in more than one case about the tenth day. Convulsions also may occur, but have not necessarily the same serious import, and they are usually well controlled by fair doses (at least half a dram a day, or more) of a bromide. The tendency to them usually soon passes off, and the bromide may be omitted. If they recur later it is merely as a sequel, and without the more dangerous significance which may be attached to them in the more acute stage of the illness.

If there be any reason to suspect that the symptoms are the consequence of thrombosis occurring in a vessel the seat of endarteritis obliterans, energetic treatment by the administration of iodide of potassium in full doses, and by the inunction of mercury, should be undertaken at once. In some cases which are treated in this way the evidences of cerebral lesion disappear entirely. But it must be confessed that in the great majority of patients undoubtedly suffering from vascular occlusion from syphilitic endarteritis the result of the most energetic treatment by antisyphilitic remedies is very disappointing. If the blocking be complete, and the tissue has already lost its blood-supply and is beginning to decay, it is clearly hopeless to try to effect any improvement. It is probably only in cases in which the process of obliteration is not quite complete that improvement may be expected. Nevertheless, every case should have the same thorough treatment, as the condition of other vessels and other organs may be much benefited thereby. The mercury should be rubbed in twice a day, if necessary, until the gums are slightly touched, and this slight affection of the gums should be kept up for at least a month.

B. The second division of the treatment has reference to those measures by which the functions of the parts of the body, which have been weakened by the central lesion, may be restored as far as possible; and the contractures and other deformities which are apt to arise may be prevented or minimised. Stiffness and restriction of movement at the different joints, especially of the upper extremity, are very prone to occur; and these parts should, from the very first, be passively moved at least once a day. If firm adhesion have taken place, as it is especially apt to do at the shoulder, it may be necessary to give an anaesthetic in order to have the joint freely moved. After this has been done, the patient will have to submit to some pain and discomfort in the daily passive movements which will be necessary to prevent refixation. As I have said already, this joint inflammation, or, as some hold, this trophic arthritic change, is sometimes complicated with neuritis, resulting in severe pain, wasting, and glossy skin. The pain in such a condition is most distressing, but great relief may be afforded by the copious application of glycerin and belladonna. When the pain has subsided, the use of the constant current and massage are the means best calculated to improve the nutrition and mobility of the limb. The passive movements recommended will also combat the inevitable tendency to contractures; and a similar purpose is served by gentle faradisation of the extensor muscles of the fingers and wrist, so as to counteract the liability to flexor contractures. In a case in which, either in an early stage or later, any tendency to the occurrence of bed-sores shews itself, the use of a water-bed or water-pillow is essential; and the most scrupulous care in treatment of the skin must be exercised by the nurse (*vide* Vol. I. p. 135). Frequent change of position, the use of "bird's nests" of cotton-wool over tender parts, rubbing the back with spirit, and all the varying attention implied in the most assiduous nursing, are of the utmost importance. In many such cases the internal administration of opium seems to exercise a very beneficial effect. As soon as the patient is convalescent, he should be got out of bed daily, and for a longer time each day. He should be encouraged to walk, and to use the affected limbs as much as possible. Improvement, once begun, often goes on to a surprising extent, especially if the patient is young and his vessels healthy. This, of course, is equivalent, or almost so, to saying that such improvement occurs especially in embolic cases. But even in others there is no need to despair, although patient perseverance in treatment is always necessary.

III. Thrombosis in Cerebral Veins and Sinuses.—Besides the occlusion occurring in cerebral vessels already described, in which the vessels affected are for the most part and characteristically arteries, thrombosis may also occur in cerebral veins or sinuses. The blocking which occurs in these structures is either (A) simple or non-infective, the result of some general condition; and (B) inflammatory, or, more properly, infective, due to local infection.

(A) *Simple Thrombosis.*—This, the most common form, which is nearly always the result of exhausting or depressing disease, is named marasmic. It occurs in the very young, especially in the first six months of life, or in the very old; or it may occur in middle life, at the end of any long or exhausting illness. The sinuses, probably because of their more rigid walls, their peculiar structure, and the manner in which the veins pour their contents against the stream into some of them—especially into the longitudinal, are more liable to thrombosis than the veins. The longitudinal sinus is that most commonly affected; but, in cases examined after death, the process has usually been found to extend into one or more of the immediately related sinuses as well. The cerebral veins may also be affected; and in some cases, as for instance at the end of an attack of enteric fever, a vein may be the only vessel blocked. In early life the most common cause is exhausting diarrhoea; and the cardiac dilatation and consequent slowing of the circulation associated with the condition produced are important factors not only in determining the thrombosis but also in causing its spread. Pulmonary affections, such as pneumonia or pleurisy, also seem to have a specially strong tendency to the production of this condition. It occurs, however, not so frequently in the course of the illness as after the subsidence of the acuter symptoms, when the patient is suffering from the consequent exhaustion and prostration.

Morbid Anatomy.—As I have already said, the longitudinal sinus is that most frequently affected. Sometimes the affection is solitary; in most cases, however, other sinuses or veins may be involved. In fatal cases the thrombosis, as a rule, extends into more than one vessel. Occasionally a surface vein only seems to be affected, giving rise to the usual symptoms of a focal lesion—unilateral convulsions and local weakness. The clots are dense, resistant, stratified, and non-adherent to the walls of the vein or sinus. They do not, as a rule, completely fill the lumen, and they tend to become organised or absorbed; they rarely disintegrate. By becoming tunnelled, they may permit the re-establishment of the circulation. As a consequence of the thrombosis there is intense congestion of the cerebral veins and capillaries; and numerous minute extravasations from the latter may give rise to a form of red softening similar to that already described as occurring in arterial thrombosis. Considerable oedema may also result. The ventricles may become distended, and a spreading of the fluid into the retrobulbar tissue may give rise to pressure on the eyeballs and consequent exophthalmos. The condition resulting on recovery is not definitely known; probably it is one of atrophy and induration of the affected area.

According to some observers, the form of infantile hemiplegia characterised by paralysis of one side, and preceded by unilateral convulsion and a condition of acute and serious illness, is determined by thrombosis occurring in the surface veins of weakly children. But in some of these cases, at least, there is acute illness with high temperature, and the conditions generally which characterise an inflammatory affection.

Symptoms.—The symptoms are rarely sufficiently definite to indicate a certain diagnosis during life. If an indefinite illness of cerebral character, ushered in by a convulsion or series of convulsions, occur in a patient after prolonged and exhausting diarrhoea or other serious illness attended with great prostration, the probability of sinus thrombosis is great. If this illness be further attended with oedema of the scalp and if partial convulsions be present, it is to be surmised that the thrombosis in the sinus has extended so as to implicate a cortical vein.

Treatment will be directed to combat the condition which has led to the occurrence of thrombosis. The copious imbibition of stimulating fluid would first be thought of; but if diarrhoea be still present this may be inadvisable. A large hot-water enema may be retained and absorbed; or it may even be necessary to give an intravenous injection of saline fluid. As soon as the alimentary canal can tolerate hot fluids, these should be given in good quantity, and the patient's general strength maintained. If the condition have been in any degree the result of pleural effusion or other pulmonary condition, this must, of course, be treated in such a way as to relieve the right side of the heart, and so to accelerate the venous circulation.

A sub-variety of simple or non-infective **thrombosis of veins or sinuses** is that which occurs in **chlorotic patients**, usually about the age of twenty. The sufferers are almost invariably women, and the signs of anaemia are, as a rule, unmistakable. The sinuses are the usual seat of the thrombosis, and in this variety, as in the last, the longitudinal is most frequently affected. Small haemorrhages from engorged veins or capillaries are usually present also. The clots in the thrombosed vessels are usually firm, but may be softened. In these cases there is no evidence of any other disease (*vide* also Vol. V. p. 713, and Vol. VI. p. 737).

The onset of the *symptoms* is, as a rule, preceded by a period of unusually hard work, and is signalised at the onset by the presence of intense headache, generally accompanied with vomiting. In the more severe cases convulsions come on; these may be unilateral or bilateral, but are usually succeeded by paralysis of one side. When this takes place the condition is usually serious, and ends, as a rule, in coma and death. Occasionally, indeed, recovery from such a condition may take place, but the patient remains more or less hemiplegic. Optic neuritis is frequently present; and it is probable that many of the unexplained cases of optic neuritis occurring in anaemic girls originate in thrombosis of veins or sinuses. In these cases recovery without serious damage to vision is the not invariable rule. In some cases the thrombosis seems to occur in the cavernous sinus, giving rise to proptosis of one eye and limitation of its movements, with optic neuritis. In such a case the clotting may spread and a serious or fatal condition ensue. In some cases, however, recovery takes place, but some interference with ocular movements usually persists, and the sight of the affected eye may be lost or impaired.

(B) *Infective Thrombosis.*—This second variety of sinus thrombosis, also spoken of as inflammatory or secondary, to distinguish it from the simple or primary thrombosis already described, occurs much more frequently in adults than in children. The name implies that there is a focus of primary disease to which the sinus affection is secondary. In the great majority of cases the infective focus is in the middle ear; but it may also be found in the nose, in the tonsils, in erysipelatous inflammation of the face or orbit, or in an anthrax pustule on the face. Compound fractures of the skull, in which the wound has become septic, may also be the starting-point of infective thrombosis; and in these conditions the sinus may become involved, either by actual spread of inflammation along the tissue intermediate between the original inflammation and the sinus wall, or by way of infection of a small vein opening into the sinus, setting up thrombosis in it. When the sinus is first affected its inner surface becomes denuded of its epithelium, and the consequent roughness determines the point at which thrombosis starts. The wall of the vessel becomes thickened and infiltrated, so that giving way of the wall, with consequent haemorrhage, is not a complication to be feared in this condition. But in this way—by direct infiltration around the wall—the area of the process may become definitely enlarged, collections of pus may be formed, and general meningitis may ensue. The existence of a clot in the sinus, especially of a clot the nature of which facilitates softening and breaking down, constitutes an even more serious danger of general infection in the course of the descending blood-current: septic particles are caught in the lung capillaries; and abscess, pneumonia, or gangrene may result. Sometimes thrombi may form lower in the venous stream than the actual place of infection, and the clot may have little or no tendency to break down. In such cases a venous trunk, like the jugular, may be completely blocked, and be palpable, as a hard cord along the anterior border of the sterno-mastoid muscle.

The *symptoms* of sinus thrombosis are somewhat indefinite, but their broad general features are sufficiently marked to permit the condition to be diagnosed, especially if a source of infection is ascertained. The local symptoms will vary, of course, with the sinus affected; but the most prominent general symptoms are severe headache with vomiting, frequent and severe rigors with profuse sweating, anorexia, diarrhoea, and an irregular temperature with marked oscillations. Not infrequently also there is oedema of the area of the scalp which is near the thrombosed sinus—over the mastoid, for instance, if the sigmoid or lateral sinus is affected; and there may also be pain, either spontaneous or only to be elicited on pressure. If infarction of the lung have taken place there may be no symptoms during the first twenty-four hours, except some cough and occasional sharp pain in the side. At the end of that time, however, or even earlier, "prune juice" expectoration occurs, and this becomes more brown from the admixture of purulent secretion. Rales are now audible over the chest; the purulent secretion may be abundant, and is very offensive, rendering extensive fumigation necessary. The stools may have the same offensive odour. Pain over the liver may also be complained of, probably from extension of inflammation through the diaphragm. The lungs are found extensively disintegrated, so that one or both are studded with abscesses. The mental condition is usually unimpaired, and may remain so almost to the very end.

In other cases abdominal symptoms—pain, and diarrhoea, pea-soup in character and very offensive, with meteorism—may be present, a condition closely simulating enteric fever. The symptoms usually come on about the second week, and are accompanied by a dry, foul tongue, and frequently by a dark measly rash, sufficiently distinct from the rose-coloured typhoid eruption. Headache is also more severe and persistent than in typhoid. Pallor of the face, languor, muttering delirium, and great prostration ensue, and death usually occurs in these cases rather earlier than in those of the preceding type.

In another class of cases, a much smaller one, the symptoms of meningitis—excitement, agitation, twitching of muscles, squint, head-retraction, and delirium—are most prominent. In the great majority of cases, however, the symptoms are made up of a combination of those enumerated in the three varieties, and in the last one especially may be complicated further by those of abscess of the temporo-sphenoidal lobe.

Medical treatment of this condition is of course inefficacious. In some cases surgical measures may relieve, or even cure. For details of surgical procedures with this object the reader is referred to surgical works, especially to the classical work of Sir W. Macewen, from which much of the preceding description is taken (*vide* also Vol. IV. Part II. pp. 476, 494).

JAMES TAYLOR.

REFERENCES

1. BROADBENT, W. H. "An attempt to remove the Difficulties attending the application of Dr. Carpenter's theory of the Function of the Sensori-motor Ganglia to the common form of Hemiplegia," *Brit. and For. Med.-Chir. Rev.*, London, 1866, xxxvii. 468.—2. BUZZARD, T. *Clinical Aspects of Syphilitic Nervous Affections*, 12mo, London, 1874.—3. CHARCOT, BOUCHARD, BRISSAUD. *Traité de médecine*, tomes ix. et x., 8vo, Paris, 1904, 1905.—4. DANA, C. L. *Textbook of Nervous Diseases*, 4th edit., 8vo, London, 1898.—5. DICKINSON, W. L. "Spontaneous Thrombosis of the Cerebral Veins and Sinuses in Chlorosis," *Trans. Clin. Soc.*, London, 1896, xxix. 63.—6. FAGGE, C. H., and PYE-SMITH, P. H. *The Principles and Practice of Medicine*, 2 vols., 4th edit., 8vo, London, 1901-2.—7. GOWERS, W. R. *Manual of Diseases of the Nervous System*, 3rd edit., 8vo, London, 1899.—8. GRASSET, J., et RAUZIER, J. *Traité pratique des maladies du système nerveux; ouvrage couronné par l'Institut*, 4me édit., 2 vols., 8vo, Montpellier, 1894.—9. JACKSON, J. H. "Cases of Disease of the Nervous System," *Clinical Lectures and Reports, London Hospital*, 1867-8, iv. 314.—10. JONES, A. E. "The Onset of Hemiplegia in Vascular Lesions," *Brain*, London, 1905, xxviii. 527.—10*a*. *Idem. Quarter. Journ. Med.*, Oxford, 1910, iii. 233.—11. MACEWEN, W. *Pyogenic Infectious Diseases of the Brain and Spinal Cord: Meningitis; Abscess of Brain; Infective Sinus Thrombosis*, 8vo, Glasgow, 1893.—12. OPPENHEIM, H. *Lehrbuch der Nervenkrankheiten*, 5te Aufl., 2 vols., 8vo, Berlin, 1908.—13. OSLER, W. *The Principles and Practice of Medicine*, 7th edit., 8vo, London, 1909.—14. ZIEGLER, E. *Lehrbuch der allgemeinen und speciellen pathologischen Anatomie für Ärzte und Studirende*, 4te Aufl., 8vo, Jena, 1886.—15. ZIEMSSEN, H. "Diseases of the Brain and its Membranes," *Cyclopaedia of the Practice of Medicine*, vol. xii., 8vo, London, 1877.

J. T.

CEREBRAL HAEMORRHAGE

By HOWARD H. TOOTH, C.M.G., M.D., F.R.C.P.

FOR the purposes of the present article the title may be taken to include intracranial haemorrhages in any position. There is no part of the contents of the cranium which can be said to be free from the liability to rupture of blood-vessels, but of course some parts are more prone to haemorrhage than others.

That a diseased condition of the arterial walls is a necessary condition seems certain; yet there are many cases, those, for instance, occurring in apparently healthy young persons, in which such a condition has not been demonstrated.

Causation.—*Remote Causes.*—Persons of certain physical conformation have long been regarded as especially liable to cerebral haemorrhage; such as have a short, thick neck, a florid complexion, and so forth; but experience teaches that the lesion is at least as common, if not more so, in persons of quite opposite build. Undoubtedly, however, there are certain general conditions which dispose to rupture of the cerebral vessels.

First among these must come that state in which are associated more or less interstitial fibrosis of the kidneys, hypertrophy of the left ventricle, and other evidences of a high arterial blood-pressure. Whatever the causal relation of these three factors to one another, there can be no doubt that in such cases there is a strong tendency to degeneration of the cerebral arteries; unfortunately, however, in the present state of knowledge, the extent of such degeneration must be one of inference only. Perhaps in the greater number of cases the only hint of such a condition during life is the raised arterial pressure, as evidenced by tortuous radial and temporal arteries, incompressible pulse, accentuated aortic second sound, and perhaps some evidence of hypertrophy of the left ventricle. Such patients may or may not present a history of the arthritic phenomena of gout, and after death uratic deposits may be found in the cartilages of their joints. In 50 necropsies in cerebral haemorrhage at St. Bartholomew's Hospital—in which the great toe and other joints were opened—uratic deposits were found in 19 instances (38 per cent); in the remaining 31 the cartilages were quite normal. There can be no doubt that the part played by gout is a very important one, yet the arthritic aspect of the matter is perhaps the least important.

There is a striking tendency to heredity in cerebral haemorrhage. A single lady, aged forty-one, during a violent attack of sea-sickness, became unconscious, and died in the hospital: a large haemorrhage was found, in the usual position, which had burst into the lateral ventricles. There was no atheroma of the cerebral arteries, and but very little of the aorta. The kidneys were but slightly affected, and the heart scarcely at all hypertrophied. But nine members of her family, who during life had presented little or no evidence of arterial or kidney disease, had died in the same way.

Certain poisons dispose to cerebral haemorrhage: of these the most important are syphilis, lead, and perhaps alcohol. Whether alcohol may operate as a direct cause is not decided; more usually its influence is indirect, by inducing a tendency to degeneration, an influence which may affect the offspring. In the later stages of syphilis the cerebral vessels seem to share in the general degradation of tissue, but in some instances the brunt of this degradation may be said to fall upon these vessels;

it is possible that this may explain some of those cases in which extreme cerebral arterial change is found without corresponding heart and kidney signs. Persons exposed to the influence of lead are, as is well known, particularly prone to gout; and it is probably through the mediation of gout that the liability in such persons to cerebral haemorrhage arises.

Certain general blood states dispose to haemorrhage, and in these the brain tends to suffer equally with other tissues: such are pernicious anaemia, leukaemia, purpura, and scurvy.

Age.—No period of life is exempt from cerebral haemorrhage, but inasmuch as arterial degeneration is most common in the later periods, it is after middle age that we find the chief liability to haemorrhage. On this point, however, there seems to be a want of agreement in the published statistics. The following table has been prepared from the post-mortem records of St. Bartholomew's Hospital during eleven years. In that period 3790 necropsies (medical) were made, including 132 cases of intracranial haemorrhage.

The age was ascertained in 124 cases:—

	Males.	Females.	Total.	Per cent.
8 – 20 years . .	6	2	8	6·5
21 – 30 ,, . .	4	3	7	5·6
31 – 40 ,, . .	11	6	17	13·7
41 – 50 ,, . .	32	11	43	34·7
51 – 60 ,, . .	18	6	24	19·4
61 – 70 ,, . .	14	6	20	16·1
71 – 80 ,, . .	5	0	5	4·0
	90	34	124	100

From this it will be seen that the liability seems to be greatest between forty and fifty, and to decline appreciably with each succeeding decade. These figures correspond in the main with those of Fagge.

Gintrac's figures have been freely quoted by succeeding authors. They have the value of large numbers, but they are largely made up of recorded cases, and are therefore to a great extent selected. According to Gintrac's tables, the percentage of cases increases with each decade from the age of thirty, to attain the maximum between sixty-one and seventy. It is also noticeable that as many as 17 per cent fall between seventy and eighty. This estimate, then, does not accord with the experience of two of the large London hospitals.

It is possible that senile degenerative processes may in some cases fall more particularly upon the arterial system, a circumstance which will explain the rupture of vessels in old people; but it is contended that an inherited or acquired tendency to arterial degeneration will lead to a dangerous condition of the vascular wall long before old age.

Sex.—As to sex, there can be no doubt that women are much less liable to cerebral haemorrhage than men. Here perhaps the statistics of the post-mortem room of a general hospital may lead to an erroneous conclusion. Reference to the above table shews 90 males (72·6 per cent) and 34 females (27·4 per cent). Many of the men were brought in from the street or from their work, where they had fallen; women, who would be more likely to be seized at home, would more probably remain at home. Perhaps Gintrac's conclusions are nearer the truth; namely, that the relation between male and female cases is about 57 to 43 per cent. Although women are equally liable with men to the hereditary causes, they are less likely than men to acquire the arterial degeneration.

It seems to be accepted almost as an axiom that cold *weather* disposes to cerebral haemorrhage; to test this point the following table was drawn up:—

December	9	March	11	June	14	September	2
January	10	April	10	July	9	October	20
February	13	May	10	August	12	November	10
	32		31		35		32

It is true that the figures are few, but the agreement in numbers between the seasons is rather unexpected. Gintrac's figures sustain the impression that the accident is more likely to happen in the cold months.

An appreciable percentage of cases occurs in the course of *infective endocarditis.* In the present series there are 6 only out of 132; but it is probable that this figure rather understates the true percentage. In this case the accident usually occurs in persons under middle life.

The circumstances in which haemorrhage may take place are very various. It is quite obvious that if a vessel-wall is so weakened as to be on the point of rupture, any sudden rise of blood-pressure (or acceleration of heart-beat) may precipitate such an accident. Hence we find cerebral haemorrhage occurring under every possible form of excitement or strain, physical or mental—as, for instance, in whooping-cough, defecation, stooping or lifting, vomiting, coitus, or sudden mental excitement. Many of the cases brought to the hospitals have begun under the preliminary exaltation of drink. Yet in many instances the rupture takes place in circumstances of complete quiescence, though on this point figures are wanting. A certain number of attacks occur during sleep. Of 246 hemiplegics, in 47 (19·1 per cent) the onset was in sleep. It is, of course, impossible to say how many of these were due to haemorrhage, and how many to thrombosis; in fact, it is probable that a large number were thrombotic. Though the general circulation is quieter in sleeping than in waking, and though it is probable that the cortex in sleep is anaemic, it does not follow that there is any great diminution of the blood-pressure in the main cerebral system of vessels; indeed, Spehl arrives at the conclusion that, though the brain as a whole contains less blood during sleep than in waking hours, certain parts of the base (the ganglia?) are probably in a state of relative congestion. His conclusions, however, are open to question, inasmuch as they are based on experiments on animals during narcotic sleep.

General Anatomical and Physical Considerations.—The copious blood-supply necessary for the cerebral functions is brought to the base of the brain by four large vessels, which here form the remarkable anastomosis known as the circle, or polygon, of Willis. The anterior or carotid element of this is perhaps the most important in the present connexion.

The internal carotid divides into two important branches. The anterior division, the anterior cerebral, passes forwards and upwards over the genu of the corpus callosum to supply the inner aspect of the hemisphere and the greater part of the anterior lobe. It anastomoses with its fellow by the anterior communicating artery, and it gives off branches internally to the head of the nucleus caudatus and to the corona radiata of the anterior lobe. It also supplies the lower half of the anterior limb of the internal capsule, and part of the lenticular nucleus, the anterior part of the external or third segment.

The other division, the middle cerebral or Sylvian artery, is practically the direct continuation of the internal carotid; it is a large vessel, but its calibre is much smaller than that of the carotid, and its diminution in size is almost sudden. It is the most important of the cerebral vessels; it courses laterally over the anterior perforated space into the fissure of Sylvius, to be distributed to the greater part of the anterior lobe, and to all the parietal region, including therefore the Rolandic area and a portion of the posterior cortex. In passing across the anterior perforated space it gives off a number of small branches at right angles to its own axis, which enter the perforations to supply, according to C. E. Beevor, the anterior commissure, the middle and external segments of the lenticular nucleus, the internal capsule, its posterior limb above the horizontal level of the upper angle of the middle lenticular segment, and also the superior half of the anterior limb, and the caudate nucleus. One especially large and important branch passes between the external capsule and the lenticular nucleus, and then into the latter; and this branch has been shewn by Charcot to be particularly prone to disease and rupture.

Before its division the internal carotid gives off the posterior communicating, which passes backwards to join the posterior cerebral, thus completing the circle of Willis. In normal circumstances this is a small vessel; but it is an important one, as it forms the basic anastomosis between the carotid and vertebral systems. It also gives branches to supply the optic chiasma, the tuber cinereum, regio subthalamica, part of the pes pedunculi, and the anterior third of the posterior limb of the internal capsule below the level of the upper angle of the middle lenticular segment (Beevor).

Next to and close to the posterior communicating (and arising, like it, from the internal carotid (Beevor), not the middle cerebral as described in most of the textbooks) is the anterior choroid, which passes backwards to enter the descending cornu of the lateral ventricle to end in the choroid plexus. This vessel sends twigs to the optic tract, the anterior third of the pes pedunculi, part of the lenticular nucleus, and the nucleus amygdalae. But its most important function is to supply the posterior two-thirds of the posterior limb of the internal capsule as high as the superior angle of the second lenticular segment (Kolisko, Beevor), together with the beginning of the optic radiations. From the foregoing description it will be seen that the internal capsule has an extensive and varied blood-supply, namely, from the anterior and middle cerebrals, the posterior communicating, and the anterior choroid.

The vertebral arteries on entering the cranium give off the posterior cerebellars, and then join to form the basilar artery, which runs forward, grooving the pons Varolii, and giving off many branches, laterally and vertically, to supply the pons and cranial nerve nuclei. Close to the origin of the basilar arise the median cerebellars distributed to the inferior aspect of the cerebellum; and at the anterior edge of the pons are given off the superior cerebellars which supply the superior aspect of the cerebellum. The basilar divides a little in front of this into the two great posterior cerebrals, which course round the crura cerebri to supply the cortex of the temporo-sphenoidal and posterior lobes, sending in branches to nourish the crus, optic thalamus, and corona radiata of the posterior lobes. An important branch of the posterior cerebral, arising close to the origin of the latter, is the posterior choroid ending in two branches, the postero-lateral to the choroid plexus and membrane in the descending cornu of the lateral ventricle, and the postero-median to the choroid plexus and membrane of the third ventricle. The posterior cerebral also supplies the fornix, corpora quadrigemina, corpus geniculatum internum, and very frequently the corpus geniculatum externum.

The dura mater is supplied by (i.) the anterior meningeal, derived from the anterior ethmoidal branch of the ophthalmic artery; (ii.) a few twigs from the ascending pharyngeal; (iii.) the middle meningeal, from the internal maxillary, a large vessel which supplies practically the whole of the dura mater except the extreme anterior part and that of the posterior fossa covering the cerebellum; (iv.) the small meningeal, a branch of the last to the Gasserian ganglion and dura mater of the middle fossa; (v.) the posterior meningeal from the vertebral, supplying the dura mater of the posterior fossa.

Besides the basic anastomosis of the circle of Willis there is every reason to believe that there is a free anastomosis between the cortical cerebral vessels. Duret admits the occasional existence of an anastomosis, but considers it to be less free and constant than other writers. However, Heubner and others, Biscons, Mendel, and Tedeschi seem to have demonstrated this anastomosis conclusively, and their observations are confirmed by the admirable work of Beevor, so freely quoted above. The branches to the nuclei of the base, however, are terminal. This point is illustrated by a case in which the middle cerebral was found completely plugged soon after its origin from the carotid; the caudate and lenticular nuclei were so softened that they could be almost shelled out, while the nutrition of the cortex was everywhere perfect. If therefore, as has been suggested (Tedeschi), there is any anastomosis between basic and cortical branches, it must be very inconsiderable.

Peculiarities of the Cerebral Circulation.—The following features of the cerebral circulation may explain to some extent the liability of the vessels to disease.

(*a*) The brain receives its blood by two distinct channels, the carotid and vertebral.

(*b*) In comparing these two systems it may be noted that the diminution in size is more gradual in the vertebral than in the carotid; which may explain the lesser liability of the former to atheroma and rupture.

(*c*) The part of the organ requiring the greatest blood-supply is outside, therefore the main divisions of the great vessels are distributed over the cortex by gradual dichotomous division, comparable with the usual division of arteries all over the body. It must be owing to this circumstance that haemorrhage is less common by far in these vessels than in those to the internal parts.

(*d*) The vessels for internal distribution come off, as above described, at right angles from the main trunks; such as those from the middle cerebral, posterior cerebral, basilar. This means friction, for the less the angle made by two branches the less the resistance to the flow; but these vessels are very small, they have a comparatively short course, and are practically terminal. Indeed, as Mendel suggests, it is probable that the pressure in these small vessels is not very much less than that in the carotid itself. The strain then upon the walls of these vessels is probably far in excess of that on the walls of vessels of a corresponding calibre in the cortex or elsewhere in the body.

(*e*) The question of the existence of vasomotor nerves on the cerebral vessels and the factors concerned in intracranial pressure have been fully considered elsewhere (p. 11 *et seq.*).

Pathology and Morbid Anatomy.—(A) The state of the blood-vessels. (B) Associated lesions elsewhere. (C) Mechanical effects of haemorrhage. (D) Situation of the focus of haemorrhage. (E) Secondary degenerations.

(A) *The State of the Blood-vessels.*—It is obvious that before a vessel can rupture there must be some preparatory weakening of its wall, and of the causes of this weakening the most important is atheroma.

Atheroma generally manifests itself in the form of patches of yellow thickening of the internal coat of the vessels, shewing distinctly through the thin walls. These patches may be of considerable size, and may affect arteries of all sizes from the carotids and basilar to small arterioles of less than a millimetre in diameter. The condition is supposed to be preceded by thickening of the internal coat—an endarteritis. This thickening may remain as such, with a tendency to increase, and so to obliterate the lumen of the vessel, at the same time causing an atrophy of the middle coat, a condition resembling syphilitic endarteritis obliterans. The effect of this is to stiffen the wall, and to dispose rather to thrombosis than to rupture.

But in other cases, and in the majority, this thickening tends to caseous degeneration and softening, so producing the characteristic yellow patches above referred to. The change may stop here, and again tend to favour thrombosis ; or the caseation may proceed to the deposit of lime salts with great impairment to the elasticity of the wall, though not necessarily so weakening it as to lead to rupture. Such a state of affairs, as Charcot and Bouchard have suggested, may however tend to raise the pressure in the small arterioles beyond, and so to aid in the formation of miliary aneurysms in these arterioles. But the caseation frequently takes a different course ; it softens and breaks down ; and then there results an actual weakening of the wall with, sooner or later, the formation of an aneurysmal dilatation, the bulging of which is favoured to some extent by the comparative want of support of the cerebral vessels by surrounding tissues. These aneurysms vary in size from that of a pin's head to that of a pea, and sometimes much larger—as large as a small chestnut in one case (Bastian (2)). They are found most commonly on the middle cerebral or its large branches ; and frequently in the fork formed by the division of the vessel. Out of 108 cases of persons dead of cerebral haemorrhage, they were found in 18 ; and of these 11 were on the middle cerebral. It should be stated, however, that Gull found aneurysm of the basilar more common than of any other artery, thus :—basilar, 20 ; middle cerebral, 15. These aneurysms are frequently multiple, and no doubt will account, by their rupture, for many of the arachnoid haemorrhages ; though they are often impossible to find on account of the large quantity of surrounding clot. It must not be supposed, however, that aneurysms of this size are common causes of intracerebral haemorrhage. Atheroma of the cerebral vessels is found in an overwhelming percentage of cases of haemorrhage. Out of 108 cases in which this point was definitely noted, more or less atheroma was found in 92 (85·1 per cent).

Conversely, however, atheroma is a fairly common lesion, whether associated with cerebral haemorrhage or not. Out of 675 necropsies during two consecutive years the cerebral arteries presented more or less atheroma in 46 ; and during the same period there occurred 25 cases of cerebral haemorrhage of which, say, 84 per cent presented atheroma. We may conclude, then, from these figures that atheroma without cerebral haemorrhage is at least as common again as atheroma with it.

Miliary Aneurysms.—In 1868 Charcot and Bouchard made their memorable observations on miliary aneurysms (*vide* Vol. VI. p. 598). These take the form of minute dilatations of the arteriolar wall, sometimes fusiform and elongated, sometimes distinctly saccular. They can be seen with the naked eye, as minute red grains on the vascular twigs. These writers considered the lesion to be essentially a periarteritis. They admitted that atheroma may coexist in the larger vessels, but did not consider that atheroma has any direct relation with these aneurysms or with cerebral haemorrhage. The part they assign to atheroma is the stiffening and want of elasticity of vessels affected by it, and the consequent transmission of the shock of the heart-beat to vessels less able to bear it and already weakened by this periarteritis.

Subsequent observers have placed the initial lesion in the internal coat. Eichler and others have looked upon it as a combined lesion, a peri-endarteritis. The common-sense view is to look upon atheroma and miliary aneurysm as manifestations of the same process. No doubt miliary aneurysms are found in some cases without atheroma, and the reverse is true ; but is it necessary on that account to suppose a different morbid process in the two cases ?

The great central fact in the chain of events is the rise of arterial tension or stress on the vascular wall. Hypertrophy of the left ventricle is a part of this process, and thus the abnormal shock of the pulse-wave becomes an accessory factor. As the vascular walls become more and more rigid, the pulse-wave is less and less taken up in its transmission ; and it arrives at the small twigs with a force much less modified than is normally the case. Why in some cases there should be miliary aneurysms without apparent intervention of atheroma is a difficulty of the same nature as the frequent want of proportion between the amount of hypertrophy of the left ventricle and the extent of an interstitial fibrosis of the kidney.

For a more detailed account of these arterial lesions see the article on "Arterial Degenerations and Diseases," Vol. VI. p. 582.

Infective Endocarditis.—Before leaving the consideration of the vessels, notice should be taken of a not uncommon termination of infective endocarditis ; namely, rupture of an embolic aneurysm. Aneurysms of this nature are caused by the blocking of a vessel by an embolus—usually a small tag of fibrin from the valve—loaded with microbes. By their proliferation the microbes give rise to an acute inflammation of the wall of the blocked vessel, which results in the rapid formation of an aneurysm, which may rupture. Out of 134 cases of cerebral haemorrhage 6 were ascertained to be of this nature ; but it is probable that this estimate is considerably under the true proportion (*vide* art. "Embolism," Vol. VI. p. 785). Aneurysms of the cerebral vessels in young persons are generally of this description. Sir W. Church, some time before the microbic origin of such aneurysms was understood, shewed that they are generally associated with disease of the valves of the heart ; and he gives a list of 13 cases, all between the ages of thirteen and twenty.

(B) *Associated Lesions of Heart, Kidney, and General Circulation.*—The correlation of these factors, and their relative importance in cerebral haemorrhage, are matters about which there is still much diversity of opinion. An attempt has been made in the following table to arrange these lesions so as to shew the frequency of their association. The figures are made up from a total number of 110 cases of cerebral haemorrhage :—

[Table

Lesions associated with Cerebral Haemorrhage.	Males.	Females.	Total.	Percentage.
1. Atheroma of cerebrals and aorta. Hypertrophy of left ventricle. Chronic interstitial nephritis.	55	17	72	65·4
2. Atheroma of cerebrals and aorta. No hypertrophy of left ventricle. Chronic interstitial nephritis.	4	2	6	5·5
3. Atheroma of cerebrals and aorta. Hypertrophy of heart. No disease of kidney.	4	4	8	7·3
4. Atheroma of cerebrals and aorta only.	4	2	6	5·5
5. No atheroma. Heart hypertrophied. Chronic interstitial nephritis.	4	2	6	5·5
6. Chronic interstitial nephritis only.	2	2	4	3·6
7. No lesion of vessels, heart, or kidney found.	2	1	3	2·7
8. Aneurysm of cerebral vessels without any other lesion.	5		5	4·5

It will be seen at once how commonly granular kidney, hypertrophy of the left ventricle and atheroma are found in association. Atheroma may exist alone, or without one or other of the usually associated lesions, as shewn by Nos. 4, 2, and 3. On the other hand, we find no atheroma in 5, 6, and 7, making a total of 13 cases, or 11·8 per cent.

Great emphasis has properly been laid upon the evidences of chronic high tension or stress of the coats of the arteries, and its probable causal relation to arterial degeneration; but we still find a considerable percentage of cases of cerebral haemorrhage without such evidences, as above shewn. According to Charcot and Bouchard, and other continental observers, this percentage is still higher. To explain such cases it seems almost necessary to look for some inherent tendency to arterial degeneration; especially in cases in which the rupture takes place in young and apparently healthy persons who presented no evidence of high blood-pressure either before death or after. In this same category must be included that form characterised by early death and a tendency to run in families.

Nothnagel drew attention to the observation of Frerichs that out of 241 cases of Bright's disease, 6 only suffered from cerebral haemorrhage; that is, 2 per cent. This observation is not borne out by experience at a London hospital; thus, it was found that, during ten years, out of 675 necropsies there were 116 cases of all diseases in which the kidneys were more or less granular, or 17·2 per cent; and this number included 20 out of 25 cases of cerebral haemorrhage; thus the coexistence of the two conditions in this country is apparently much higher than on the Continent.

(C) *Mechanical Effects of Haemorrhage.*—When a considerable vessel bursts in the soft yielding brain-substance there is formed at first a more or less oval focus, with its long axis parallel to the line of least resistance; that is, in the length of the fibres if the rupture be in the white matter. If it is in the common position between the lenticular nucleus and the external capsule, it will tend to separate these two structures. If the leak be a gradual one the effused blood may clot quickly, and so stop any further leaking for a time, if not permanently. In this case the clot gradually changes colour, turns brown, softens, and slowly becomes absorbed. The cerebral substance round the focus is generally stained, sometimes for a distance of a quarter of an inch. This stain is at first a claret-red, but soon alters to brown, and finally to yellow. At the same time the lacerated brain-substance undergoes fatty change, and eventually disappears; then a cavity is formed, often with a distinct cyst-wall (Charcot), containing a yellowish fluid, with at one period masses of brown amorphous pigment in it, or even crystals of haematoidin. Where the extravasation of blood is but small, absorption is so complete that only a yellow stain may be left behind—the so-called ochreous patches.

But very frequently the effusion of blood is too rapid for such a favourable course. The blood is then pumped into the soft brain-tissue, and with each beat of the heart its power for destruction and laceration increases, on the principle of the hydraulic press, by virtue of which, as the cavity enlarges, the power exerted by the blood-pressure and pulse becomes enormously multiplied[1]: in fact it is difficult to see how a haemorrhage into the brain ever ceases, unless by pressure on the artery itself. The brain itself now becomes compressed, so that on opening the cranium the convolutions are found to be flattened; at first chiefly on the side of the haemorrhage, and later on both sides.

The advance of the destructive haemorrhage is practically always towards the centre; so that, unless stopped, rupture into the ventricles follows sooner or later. The lateral ventricle on the same side is first filled with blood, which then oozes across through the foramen of Monro into the opposite ventricle, and downwards into the third ventricle so as to distend the infundibulum, which, being thin, may rupture, and blood appear in the arachnoid space about the base. At this stage the corpus callosum will be found to bulge upwards, and to fluctuate. The fornix and lower aspect of the corpus callosum may be softened and eroded. The blood may find its way down the descending cornu of the lateral ventricle and reappear at the base; but this passage is generally stopped by compression. In haemorrhages of this size the blood will often be found along the aqueduct of Sylvius and in the fourth ventricle, from which it may find its way upwards and round the peduncles of the cerebellum, coating the latter with a thin layer of clot.

Rupture of vessels at the base gives rise to an effusion of blood beneath the arachnoid. In such haemorrhages the arachnoid cavity of the base will be filled with clot which, owing to its entanglement with the numberless fine fibrils of the arachnoid, is generally very firm; it may also spread up from the base so as to form a thin coat of clot over the whole brain.

---

[1] This power can be rather strikingly demonstrated by directing a stream of water at a very low pressure, say 1 mm. of mercury, into a thin india-rubber air-ball. Even at this low pressure the ball will inevitably burst, after distension to its utmost. If a small piece of the same rubber be tied over the mouth of the delivery tube it will sustain a great pressure of mercury, say 100 mm. or more.

Haemorrhages into the cortex rarely open externally (Charcot), but follow the general rule of thrusting towards the centre. When, however, they do rupture externally, the blood tends to spread all over the surface in the arachnoid space.

(D) *Situation of the Focus of Haemorrhage.*—By far the commonest seat of haemorrhage is in the region of the lenticular nucleus; and, as before mentioned, the ruptured vessel is most commonly that branch of the middle cerebral which runs between that nucleus and the external capsule. However, in the larger number of cases the destruction of tissue is so extensive as to render the discovery of the primary vascular lesion impossible; yet the centre of the mass of clot may be taken, approximately, as the site of the initial lesion; and in the following cases this point has been taken as the guide to its situation.

Out of 118 cases in which the seat was noted we find 76 (64·4 per cent) in which the focus lay external to the lenticular nucleus and the anterior part of the optic thalamus; of these, 32 were on the right side and 44 on the left. The pons Varolii is the next and most common situation; in 19 cases the lesion was primary and in the pons only; in 11 others there were small haemorrhages in the pons, but the main lesion elsewhere. So that in respect of vulnerability of the vessels of the pons the figure should stand at 30 (25·4 per cent).

The corona radiata, or white matter of the frontal region, comes next, but with a considerable drop in the numbers. The vessels here concerned will probably be the anterior cerebral, or again the deep branches of the middle cerebral. Of these we find 6 only, 5 of which were on the left side. Foci confined more to the optic thalamus, and due to rupture of branches of the posterior cerebral, occurred in 5 instances only, of which 4 were on the right side. The corona radiata of the posterior lobe was the seat of haemorrhage in 3 cases. The cerebellum presented foci of haemorrhage in 2 cases only; Sir W. Gowers says this haemorrhage most frequently follows rupture of a branch of the superior cerebellar to the dentate nucleus. The crus cerebri was affected in one case, but a large haemorrhage in the thalamic region may not infrequently extend backwards down the crus of the same side.

In comparing the liability to rupture of the two systems of vessels —the carotid and vertebral—we find that the accident is just twice as frequent in the former as in the latter; namely, 84 of the carotid system and 41 of the vertebral (the 11 pons cases included above come on both sides of the account).

Haemorrhage into the lateral ventricles is rarely primary. It was believed to be so in 2 of the cases. According to Duret, the walls of the ventricles are mostly supplied by the posterior cerebrals. The choroid plexus receives blood from the carotid as well as from the vertebral system. Miliary aneurysms have been found on the vessels of the choroid plexus. But secondary haemorrhage into the ventricles is very common, as may be judged by its occurrence in 56 of the above cases (47·4 per cent).

Lastly, haemorrhage may take place into the arachnoid space, and this occurred in 4 instances, in which the ruptured vessel was not found. This form of haemorrhage is generally named meningeal haemorrhage; but obviously it is due to lesion of the cerebral rather than of the meningeal system of vessels.

It has sometimes been stated that cerebral haemorrhage is commoner on the left side, but this does not appear to be so; in 876 collected cases of cerebral haemorrhage E. Jones found 438 on each side of the brain.

True meningeal haemorrhage, due to rupture of branches of the meningeal arteries, is most often traumatic. The effusion of blood may be on either side of the dura mater—that is, extradural, or subdural. Subdural haemorrhages of considerable size may occur in some blood diseases; such as purpura, scurvy, and pernicious anaemia. The extravasated blood forms a thin layer of clot over the hemisphere, sometimes on both sides symmetrically. The clot is sometimes laminated, the various layers indicating definite extravasations; the most recent being next the dura mater. Moreover, Dr. Herringham has shewn that the haemorrhage can actually be seen in the substance of the dura mater, in the form of ecchymoses sometimes of considerable size.

It was formerly considered that subdural haemorrhages were very common in asylum cases, more particularly in general paralytic dementia. Virchow maintained that these haemorrhages were of inflammatory origin, hence the term Pachymeningitis haemorrhagica, a view not accepted generally in this country (Wynne, Wigglesworth). The term, however, has been retained by Dr. Mott, but not quite in the sense held by Virchow. He recognises a simple subdural haemorrhage or haematoma, which may be due to direct injury, and a slow repeated leaking from small vessels, producing a laminated clot, the appearance seen in cases of general paralytic dementia. In either case there is a weakening of the vascular wall of a subacute chronic inflammatory nature, part of the degenerative process, which may be found in cases other than of general paralysis. Dr. Mott finds that in 461 necropsies in general paralysis there were no intracerebral haemorrhages, and only 20, or 4·3 per cent, of pachymeningitis haemorrhagica. Another interesting point is that the heart, far from shewing the enlargement of arteriosclerosis, was usually wasted in these cases (Vol. VI. p. 593).

Haemorrhage is not uncommon in the course of the growth of an intracranial tumour, more especially in angiosarcomas. The haemorrhage may so plough up the growth, and alter appearances, as at the necropsy to mask the original disease.

The traumatic haemorrhagic lesions sustained at birth are generally meningeal or subarachnoid (*vide* art. "Cerebral Palsies of Children," p. 454).

(E) *Secondary Degenerations.*—If the haemorrhage do not prove fatal, and the destruction of tissue is complete and involves the motor fibres, there will follow secondary degeneration of the pyramidal fibres in the medulla and cord. The great bulk of the pyramidal fibres cross, at the decussation of the pyramids, to become the crossed pyramidal tract in the lateral column of the cord. A considerable number, however, do not cross, but run down in the anterior column of the cord next the anterior fissure. A number of uncrossed fibres are found in the "mixed zone" of Flechsig, that is, in the neighbourhood of the anterior roots. All these fibres degenerate completely, and finally become absorbed and disappear, leaving at first a mass of disorganised myelin; their place is ultimately taken by fibrous or sclerotic tissue, which eventually contracts into a patch of scar tissue.

Symptoms.—In this article we are mainly concerned with such symptoms as can be referred to extravasations of blood into or upon the brain, and therefore with the symptoms peculiar to the earlier or acute stages of the attack. The chronic or enduring symptoms, being common both to haemorrhage and vascular occlusion, are fully treated of under the latter head.

Prodromes, in the ordinary sense of the word, can scarcely be said to exist. Any premonitory symptoms must be attributed to circulatory disturbance, or to small and gradual haemorrhage; in the latter case such symptoms are practically part of the attack. Atheroma is the commonest cause of vascular occlusion, and it is possible that some of the symptoms called prodromal may be due to it. With this reservation we may mention, among the so-called prodromal symptoms of the older writers, giddiness or dizziness, mental irritability, numbness or tingling of the extremities, and headache. Nothnagel remarks on the importance of a sudden loss of the power of speech, as suddenly regained after a few hours.

The onset of the attack may be sudden or gradual; with or without loss of consciousness. A sudden and complete loss of consciousness is implied in the name "apoplexy." Apoplexy has become so associated with haemorrhages into the brain that sudden haemorrhages in other parts of the body are called also apoplectic; notably those in the lungs. Nevertheless the general opinion is that the symptoms of haemorrhage into the brain are more often gradual than sudden, and that the term "apoplectic" applies less often to haemorrhage than to embolism (Trousseau). It is, however, usual to speak, somewhat loosely, of the early comatose stage as the apoplectic stage.

The explanation of the apoplectic onset is still rather obscure. It appears to bear an uncertain relation to the amount of blood extravasated; for, as Nothnagel argues, a great haemorrhage may present a gradual development of symptoms, whilst a small one may exhibit the picture of apoplexy. The terms "cerebral surprise" (Trousseau) and "cerebral shock" (Hughlings Jackson) serve to convey to the mind the idea of the suddenness rather than of the extent of the lesion. Whether the loss of consciousness is to be referred to sudden rise of intracranial pressure, as the experiments of Pagenstecher would seem to prove, or to interference with blood-supply (anaemia), again in its turn due to pressure on vessels (Abercrombie), or to a condition allied to concussion, must at present be a matter of opinion. Astley Cooper produced a temporary loss of consciousness by pressure of the finger on the brain through a trephine hole; and Bergmann rendered a child comatose by compressing a meningocele; but neither of these observers says how long the loss of consciousness lasted under pressure, or if consciousness returned without relief of pressure (*vide* p. 30).

The onset may be quite painless; but a sudden sharp pain in the head is not uncommon (Abercrombie), especially when the extravasation is into the arachnoid space: such a pain, followed immediately by coma, suggests also a large haemorrhage into the ventricles.

Convulsions, generally speaking, are not common. When they occur they usually appear in the early stage. Where they are local they suggest pressure on cortical areas, and therefore extravasation into the arachnoid space. They are also common in haemorrhage into the pons.

The patient may recover consciousness, partially or completely; or the loss of consciousness of the initial attack may deepen, from a state in which he can be partially aroused, into the deepest coma or carus, which may last for many hours or sometimes for days before death ("Ingravescent apoplexy").

At this period the skin may be found moist with sweat, sometimes so excessively as to saturate the clothes. The veins of the face and neck may be turgid, and the countenance generally cyanosed, but this state will vary with the amount of respiratory embarrassment. On the other hand, in the gravest cases the countenance may be placid and pale, or of natural colour, as in sleep.

The breathing is often stertorous, with noisy rattlings in the throat, due to accumulations within the trachea of secretions, and often of food and drink; the natural and imperious reflex cough being diminished, in common with the other reflexes, by the depth of the coma.

Owing to the complete muscular "resolution" the cheeks will be flaccid, and may be drawn in and puffed out by the respiratory movements. A similar condition of the soft palate may give rise to loud snoring and stertor.

The respiratory rhythm is frequently unaltered, but any alteration is a symptom of grave import. It may be irregular or deep and slow. The Cheyne-Stokes rhythm is generally of fatal augury.

The state of the pulse is variable; more commonly it is slow and deliberate, sometimes it is rapid. An irregular pulse is a sign of grave importance. The blood-pressure may be naturally high in persons liable to haemorrhage. But in large haemorrhages it often rises to a great height, 300 mm. Hg or more. A steady rise of pressure indicates continued haemorrhage, and is therefore of very serious, probably of fatal, import (Janeway). The affection of respiration depends largely upon intracranial pressure; this has been shewn by the experiments of Spencer and Horsley, who find that a rise of intracranial pressure is followed by slowing of the respiration and of the heart, even to complete temporary arrest.

Vomiting may occur early in the attack. It is, as Sir William Gowers remarks, especially common in haemorrhage into the cerebellum.

The position and movements of the eyes and head often give important evidence of the unilateral situation of the lesion. There is a tendency, frequently well marked, for the head to turn to one side; and with this may be seen a conjugate deviation of the eyes in the same direction. This seems to be due rather to unopposed action of the muscles on the side of the lesion than to definite spasm. The patient is then said to look towards the lesion. The importance of this sign was insisted upon by Prevost, who affirms that it always indicates an organic lesion of the brain. The symptom tends to disappear in two or three days. It may sometimes appear in severe one-sided convulsions of an epileptic nature. It must be remembered, however, that during a convulsion the deviation may be in the opposite direction, when it is due to spasm. Moreover, in haemorrhage into the pons with lesion of the sixth nucleus, the eyes look away from the lesion.

The condition of the pupils is too variable to give much help in diagnosis. They may present every degree of dilatation or contraction or they may be unequal.

Cerebral haemorrhage never gives rise to optic neuritis, but important indications of the antecedent condition of the patient may be gathered by examination of the fundus, where may also be seen sometimes haemorrhages recent and old, and perhaps albuminuric retinitis.

The temperature has received much attention from Bourneville. In large rapidly-fatal haemorrhages there is generally a fall of the general temperature at first. It may fall to 96° F., or even 94·4° (Bastian (4)), and the patient may die before reaction sets in. But more often this fall is followed by a rise; gradual in some cases, rapid in others. In slight cases it may rise to little above the normal; but in severe cases it may rise steadily to 108° F. or even higher. In large ventricular extravasations, and in haemorrhage involving the pons and medulla, this rise is often extremely rapid. In unilateral lesions, even in the apoplectic stage, there is a difference in the temperature of the two sides of the body, so marked sometimes as to be recognised by touch (Lépine). Dr. Bastian (3) points out that this difference is caused by a fall on the non-paralysed, rather than by a rise on the paralysed side, as is stated by most writers.

In cases of large haemorrhages the reflexes are all abolished, superficial, deep, and conjunctival. The sphincters of the bladder and rectum are relaxed, and urine and faeces passed. The urine may be plentiful in quantity and contain albumin, and also sugar.

In the state of deepest coma the limbs lie completely flaccid, so that it is impossible to decide which side is affected. But as the coma becomes less deep, slight movements may be seen in the limbs of the sound side; and when lifted the arm on this side falls less heavily than that of the other. Sometimes there is a marked rigidity of the limbs on the paralysed side, a rigidity attributed by some observers to cerebral irritation. Such early rigidity is of course to be distinguished from the rigidity of a later stage of paralysis.

The extreme difficulty in the way of diagnosis between haemorrhage and vascular occlusion makes it impossible to say how many of the slighter cases of hemiplegia are due to the one cause or the other. There can be little doubt, however, that small haemorrhages, like thrombosis, may be unaccompanied by loss of consciousness. The frequent discovery of old foci of haemorrhage, without any history of an apoplectic attack, confirms this impression. This subject is further discussed on p. 329.

Duration.—Sudden death is very rare. One case is quoted by Fagge; in it the haemorrhage was at the base. It is probable that the explanation of such cases must lie in a profound interference with vital functions due to pressure on the medulla.

There are several recorded instances of death in five to ten minutes; but more commonly life is prolonged from two to twelve hours, and in some cases the patient may remain in a state of complete unconsciousness for a week or ten days before death. In fatal cases the respiration ceases before the pulse.

The initial apoplectic coma may pass off in a few hours, the patient may recover consciousness, and then suddenly relapse into fatal coma; an indication of secondary rupture of the focus of haemorrhage into the ventricles. In cases which recover, the coma may pass off in any time from half an hour to six hours. The earliest evidence of a change for the better is a return of the reflexes. There is generally some headache, dulness, apathy, very often also some alteration of speech; these symptoms may all disappear, leaving only the paralysis or localising sign behind.

Localising Signs and Symptoms.—Even during the apoplectic stage certain hints may be taken as to the seat of the lesion. When the coma becomes less deep the patient may exhibit uneasy movements of the limbs which, being absent on one side, point to the unilateral site of the lesion. An early rigidity of the paralysed limbs is sometimes found.

The deviation of the eyes and head in the direction of the side of the lesion has already been mentioned.

The mouth is often drawn away from the paralysed side, and the tongue, if protruded, deviates in the opposite direction—that is, towards the paralysed side. The upper facial muscles are not affected. The explanation of this, given by the late Sir W. Broadbent and generally accepted, is that muscles like the occipito-frontalis, though morphologically bilateral, have through long association become actuated equally well by either centre in either hemisphere. In this respect this group of muscles resembles the respiratory thoracic muscles and diaphragm, which are never affected unilaterally in hemiplegia. Occasionally, however, we see cases in which, for a time at least, there is a well-marked facial paresis on the paralysed side, the explanation of which is obscure. Though in hemiplegia the upper facial muscles are rarely affected, for the reasons above given, yet there is evidence of the bilateral representation of their movements in the want of power of independent closure of the eye on the paralysed side—the "orbicularis symptom" (L. Revilliod). A person who, before the attack, could shut either eye independently is found after it to be unable to close that on the paralysed side alone; he can of course close both together without any sign of paresis. This symptom is considered here for convenience, but it belongs more properly to the hemiplegic stage.

The above description applies to the commonest seat of haemorrhage, that is, the region of the corpus striatum. Here the unilateral signs are due to pressure upon the great mass of white fibres converging to form the internal capsule, or to actual destruction of them. As the apoplectic symptoms pass off, the features of a hemiplegia become more defined.

The researches of Beevor and Horsley tend to confirm the view of Charcot and others that the fibres of the capsule are arranged in an orderly manner from before backwards. Thus they find that the excitable fibres of the capsule are arranged in the same order as the foci of representation in the excitable cortex; also that a considerable part of the posterior limb of the capsule, amounting in the lowest plane to a third, is inexcitable. That this inexcitable region of the capsule contains afferent or sensory fibres is practically certain from the results of clinical and experimental observation (Charcot, Veyssière).

Motor symptoms due to lesions in the region of the corpora striata must be attributed to pressure upon or destruction of the fibres of the internal capsules, for the so-called basic ganglia are found to be inexcitable. Moreover, from the above experimental researches we may infer that the fibres derived from the anterior lobes of the brain will be found in the anterior, and those from the Rolandic area in the middle part of the capsule; whilst sensory fibres lie in the posterior or thalamic region. We have therefore the elements of a more or less certain diagnosis of the seat of the lesion. If a patient survive a haemorrhage of such extent as to destroy the whole of the internal capsule on the left side, we may expect grave speech-defects (probably not amounting to true aphasia), complete right hemiplegia, and hemianaesthesia. A temporary hemianaesthesia is extremely common, but in haemorrhage it is less commonly permanent. This temporary loss of sensation is probably due to compression by a clot which eventually diminishes in bulk by absorption.

Permanent hemiplegia is unfortunately very common. The defects of speech are also very common, but happily often transitory. They consist generally of little more than defective articulation, hesitation, or stammering. True aphasia can scarcely be possible from destruction of fibres so low down. Defects of speech are common in lesions of both sides.

For further details concerning the symptomatology of the chronic condition of hemiplegia, the reader is referred to the article on "Occlusion of Cerebral Vessels" (p. 293 *et seq.*).

Corona Radiata.—Any part of the corona radiata may be the seat of haemorrhage. The symptoms from the fan-like arrangement of the fibres will be much more restricted than in lesions of the same extent in the capsule. These lesions are also rarer, for the vessels supplying the centrum ovale are the small terminations of both the cerebral and cortical systems of arterioles, and therefore less liable to the formation of aneurysms.

The anterior lobe may be the seat of a haemorrhage large enough to hollow it out to a mere shell. In the apoplectic stage it is doubtful if this would give any localising sign at all; but as the coma clears away there may be very definite speech-defects, even true aphasia (Pitres), without any definite motor paralysis.

Lesions of the white matter of the posterior lobes, again, produce no motor signs, but Pitres describes considerable mental weakness in such cases. The white fibres of the occipital region converge to form the optic radiations which stream into the optic thalamus. These may be largely injured by a haemorrhagic focus, and important symptoms connected with vision may result. Ablation experiments (Munk, Brown and Schäfer) on the occipital lobes shew that homonymous hemianopsia of the opposite side follows extensive lesion of one lobe, and that removal of both lobes produces complete blindness. It is practically certain that the same effects are produced by destructive lesions of the optic radiations. Dr. Ferrier finds that temporary amblyopia of the opposite eye follows ablation of the angular gyrus. Clinical experience, however, tends more and more to confirm the view that each occipital lobe receives the sight fibres from the half of the two retinae on its own side.

Cortex.—Haemorrhages of the grey matter of the cortex are generally small, often capillary; but, on the whole, rare. Here the whole field of localisation is open, to which chapter the reader must be referred (p. 37).

Optic Thalamus.—Observations by various authors on lesions of the optic thalamus have been brought to a focus by Dejerine, who sums up the clinical features in the term Thalamic syndrome as follows: (1) Slight transient hemiplegia without contractures. (2) Persistent hemianaesthesia, cutaneous hyperaesthesia, and affection of deep sensibility. (3) Slight hemiataxia and more or less complete astereognosis. (4) Paroxysmal pains on the affected side, often very severe and intractable. (5) Choreo-athetotic movements. (6) The reflexes generally may be normal or exaggerated. A striking feature is the absence of the extensor plantar response of Babinski, in spite of other evidence of pyramidal degeneration and even its presence as shewn after death (*vide* also p. 110).

Corpus Callosum.—As shewn by Beevor the corpus callosum receives its blood exclusively from cortical vessels, namely, the anterior cerebral which supplies the genu and most of the body, and the posterior cerebral which nourishes the splenium and forceps major. Local lesions therefore are more liable to arise from thrombosis than haemorrhage. Nevertheless a few words of reference to recent works on the symptomatology of the corpus callosum may not be out of place here. That it is a great connecting commissure between the two hemispheres is shewn by the experiments of Dr. Mott and Prof. Schäfer. But the work of Liepmann and others as summarised by Dr. S. A. K. Wilson shews that the motor effects of lesion of the callosal fibres come into line with the motor phenomena of aphasia, and are included under the general designation of "Apraxia" (*vide* p. 447).

Crus Cerebri.—The origin of the third nerve from the crus cerebri lends a special interest to haemorrhages of this part. If the third nerve or its nucleus be destroyed or compressed, paralysis of the ocular muscles supplied by that nerve will follow on the same side as the lesion, with hemiplegia of the opposite side of the body (Weber).

Isolated lesions of the crus are rare, but it is not uncommon to find large haemorrhages of the thalamic region encroaching backwards down the crus of the same side. In such cases, however, the coma is probably too deep to allow the ocular symptoms to be made out. Sensation will frequently be affected on the paralysed side.

Pons Varolii.—Haemorrhagic lesions of the pons present certain fairly distinctive features. The texture of this part is more resistant than in any other region of the brain, owing largely to the interlacing of fibres in all directions; haemorrhage here is usually therefore small and circumscribed. Nevertheless the pons sometimes becomes hollowed into a cavity full of clot. The loss of consciousness is generally profound. Convulsions are described by most authors, and these, as a rule, are general, or affect the lower limbs only; they are rarely unilateral (Gowers). From the point of view of the present hypothesis concerning the invariable association of convulsive discharge with nerve-cells and cortical grey matter, these convulsions are very difficult to explain. Nothnagel has shewn that irritation of the pons in rabbits gives rise to general convulsions. The pupils are frequently strongly contracted, but not invariably so. In these circumstances the diagnosis between opium poisoning and haemorrhage into the pons may be extremely difficult, and yet very important in view of the widely different methods of treatment in the two cases.

The more sure indications of haemorrhage into the pons consist in the effects of lesions of cranial nerves, the most important being the sixth and seventh. When the seventh is involved we have the association of paralysis of the face on the same side as the lesion with hemiplegia and hemianaesthesia of the opposite side of the body. This is the case when the lesion is low down in the pons. When the lesion is high in the pons the facial paralysis, if it exist at all, may be on the same side as the paralysis, owing to the crossing of the supranuclear connexions of the nerve (Nothnagel). Very interesting results follow lesion of the sixth nerve or nucleus. If the nerve only be affected, there is paralysis of the external rectus on the same side as the lesion, with consequent internal strabismus of that eye. If, however, the nucleus only be involved, there will be defect of conjugate deviation of the eyes on the same side; that is, the eyes will be drawn over by the paralysed side, or away from the lesion, the exact opposite to the effect of lesions higher up. This is explained by the associated action of the external rectus of one eye with the internal rectus of the other, the nucleus of the sixth nerve being connected with that of the opposite third by the commissural fibres of the posterior longitudinal bundle. Vomiting is not uncommon in haemorrhage into the pons. Polyuria, albuminuria, and sometimes temporary glycosuria may occur.

The medulla oblongata may be considered with the pons, and may be disposed of in few words. Theoretically paralyses might be numerous, from small local haemorrhages; but practically haemorrhages into the medulla are generally large enough to cause death, and that rapidly—even instantaneously. The possibility of a localised haemorrhage with symptoms of bulbar paralysis must be admitted; but hitherto the recorded cases of pseudo-bulbar paralysis (Dixon Mann) have been cases of vascular occlusion.

Cerebellum.—The exact diagnosis of haemorrhage in this part of the brain is very difficult. Generally speaking, the apoplectic stage presents no distinguishing symptoms. The onset is very frequently marked by a sudden pain in the back of the head or neck. Vomiting is also an almost invariable symptom. An extensive haemorrhage almost always bursts into the fourth ventricle, or into the arachnoid space spreading up under the tentorium, both of which conditions are rapidly fatal. It is possible that if the patient survive a haemorrhage into one hemisphere of the cerebellum the proper localising signs might be manifested, though a recorded case has not been found. These are, according to Dr. Risien Russell, motor paresis of the limbs on the same side as the lesion, incoordination of movement, and, sometimes, exaggeration of the knee-jerk on the same side (*vide* also p. 134).

Ventricular Haemorrhage.—By this is meant a haemorrhage, primary or secondary, into the lateral ventricle, which may spread to fill the opposite ventricle, and finally all the ventricles of the brain.

The coma is rapid and deep, and the issue generally fatal. As indicated above, this accident is generally secondary to the rupture of a haemorrhagic focus in the region of the basic ganglia. Its occurrence may be suspected when, in a case of obvious cerebral haemorrhage, there has been a partial or complete recovery of consciousness followed by a sudden relapse into deep and lasting coma, with complete muscular resolution and abolition of reflexes. The temperature, which may have begun to rise again, falls, to rise subsequently to a great height. The pupils may be dilated, immobile, or contracted to a pin's point size, in which case the picture resembles closely that of haemorrhage into the pons.

That haemorrhage into the ventricles is not necessarily always fatal is evidenced by a case of Charcot's, a woman, in whom was found an old focus of haemorrhage occupying the corpus striatum and thalamus, and communicating freely with the lateral ventricle.

Meningeal Haemorrhage.—Under this head may be included traumatic lesions of the meningeal arteries, and subdural haemorrhage, both of which may be strictly termed meningeal; and also haemorrhage into the arachnoid space, which would be more correctly named arachnoid haemorrhage. Small subdural extravasations are often found after death without any history of symptoms. But an extensive haemorrhage, by its disposition, causes great rise of intracranial pressure, especially directed upon the cortex, and therefore gives rise to fairly well-marked symptoms. There is generally great pain in the head, due perhaps to pressure on the dura mater; coma rapidly follows. There are generally also convulsions, sometimes localised at first. The pain and the convulsions are more common in this than in any other form of haemorrhage.

Prognosis.—That the gravity of the prognosis is directly proportional to the size of the haemorrhage is almost a truism. And yet this statement must be qualified to a certain extent on consideration of the situation of the lesion; for it is evident that a clot which, if in the pons or medulla, would be large and rapidly fatal would be of comparatively little importance if in the corona radiata.

Where the coma is very deep, even when the patient is seen quite early, a very cautious prognosis should be given; and if the coma persist without any lessening in intensity over twelve hours, the outlook is very grave. In favourable cases signs of return to consciousness begin to shew themselves in two or three hours; but, in some cases, recovery may take place after a deep coma of twenty-four hours; after this time, however, a fatal termination is most usual. A comparatively rapid return to consciousness with a sudden relapse into coma suggests ventricular haemorrhage, and is practically of fatal import.

When there is a considerable initial fall of temperature, with a rapid rise to a point much above the normal, a large haemorrhage may be suspected, and the prognosis is proportionally grave. Convulsions indicate a meningeal effusion, a great and sudden increase in the intracranial pressure, or a haemorrhage into the pons; all conditions of great gravity. An abnormally slow, or quick, or irregular pulse is an unfavourable sign; so also is a progressive rise of blood-pressure. Equally unfavourable are alterations of the respiratory movements, especially Cheyne-Stokes respiration. The appearance of albumin or sugar in the urine adds greatly to the probability of a fatal issue.

On the other hand, a slight degree of coma, or, if deep, a coma of short duration, is a favourable indication. One-sided symptoms, discovering themselves early, indicate a focal lesion of moderate extent. The prognosis then is rather of the extent and duration of the paralysis. It is remarkable how complete in some cases is the recovery of motion in the paralysed limbs, probably on account of the rapid shrinking of the clot by absorption, with relief of pressure on the motor fibres. No rules, of much use for guidance, on this point can be given. If, however, an improvement is to take place, it generally begins within a week or ten days. It is generally the rule that improvement takes place first in the leg, and that when it begins first in the arm the ultimate result will be less satisfactory than in the former case (Trousseau).

The appearance of rigidity early, that is within the first week, is a very unfavourable sign. The same may be said of increased reflexes. The cases most likely to do well are those in which there is no rigidity, with equal knee-jerks on the two sides, without any sign of ankle-clonus, and with a not too lively plantar reflex on the paralysed side. The appearance of these signs, especially rigidity, indicates a secondary descending degeneration, and therefore destruction of fibres in the motor tract.

There is frequently considerable mental deterioration, and emotional instabilities, with especial tendency to uncontrollable fits of weeping or laughter, symptoms perhaps more common when the lesion is on the left side of the brain, but in any case unfavourable symptoms as regards complete recovery.

Speech-defects, such as hesitation, stammering, thickness of utterance, or even a tendency to misname persons or things, are very common and frequently disappear early; they do not influence prognosis seriously. But true amnesic aphasia, word-deafness, or word-blindness are very serious signs, and rarely pass away completely. For the further discussion of prognosis in hemiplegia, see p. 296.

Differential Diagnosis.—*From Thrombotic Vascular Occlusion.*—The diagnosis of cerebral haemorrhage is often one of extreme difficulty, so much so that, in many cases, it may be absolutely impossible to arrive at a certain conclusion. This uncertainty is all the more to be regretted inasmuch as treatment, to be of value, can only avail in the very earliest stages. The physician then frequently finds his beneficent intentions reduced to comparative impotence at the stage in which he might well hope to influence the progress of the disease.

The early stages of haemorrhage and softening may closely resemble each other in their symptoms, a resemblance which is explained rather by the diversity of symptoms in haemorrhage than by a similar want of definiteness in those of thrombosis. To put this point somewhat crudely, haemorrhage tends to simulate thrombosis rather than the reverse.

A consideration of the physical conditions in the two lesions affords some clue to the differential diagnosis, and may explain its difficulties. Haemorrhage into the brain or cranial cavity must of necessity be accompanied by rise of intracranial pressure; yet in small haemorrhages this pressure may be so slight as to give rise to no direct symptoms, and herein lies one great difficulty in diagnosis. There is no such rise of pressure in thrombosis, and small haemorrhages therefore, in their symptoms, may exactly resemble thrombosis.

The main symptoms of cerebral compression are coma, convulsions, alterations of pulse, blood-pressure, and temperature. These may be absent in cases in which the haemorrhage is small, and are generally absent in cases of thrombotic softening. Trousseau, following Récamier, sums up the matter in these words: "Whenever complete and absolute hemiplegia occurs suddenly, without loss of consciousness, softening of the brain may be diagnosed. Whenever, on the contrary, the complete loss of motor power is attended by loss of consciousness . . . haemorrhage may be diagnosed." To this broad rule there are many exceptions.

Authors differ widely as to the relative frequency of the two lesions. Some consider that thrombosis is the commoner lesion (Gowers), and I am inclined to take the same view. But others look upon haemorrhage as the most frequent cause of hemiplegia (Nothnagel). This difference of opinion naturally arises out of the difficulty in distinguishing the slighter haemorrhages from thrombosis. There can be no doubt that in the post-mortem rooms of general hospitals haemorrhage is seen vastly more often than thrombosis.

Embolism.—When a large vessel, such as the middle cerebral, is plugged, there is generally a sudden loss of consciousness. The patient then presents the picture of true apoplexy. This is due, however, not to a sudden rise of pressure within the skull, but to the interference with the blood-supply of a large area of brain. The recovery of consciousness usually takes place in a comparatively short time, and sometimes almost suddenly.

A source from which an embolus may be derived may be detected; and undoubtedly the commonest source is the valves of the heart in the course of rheumatic or infective endocarditis. Emboli may occur also in pyaemia, with foci in any part of the body. They are not uncommon in a septic condition of the uterus after childbirth. Sometimes a clot may come from the wall of the aorta, or from an aneurysm (Gowers).

Apoplexy in young persons, though it may be haemorrhagic, is more likely to be due to embolism.

Epilepsy.—The epileptic seizure may be marked by a short convulsive stage and a long period of coma. This may lead, in the absence of history, to a temporary error in diagnosis.

Poisons.—Alcohol.—Acute alcoholic poisoning may present great difficulties in diagnosis. In such a case the pupils may be tightly contracted or dilated. The alcoholic odour of the breath is no sure guide, for alcohol is a universal remedy with the public. From the point of view of the welfare of the patient it would be less disastrous to mistake alcoholism for cerebral haemorrhage than the reverse. Even where grave doubt in the diagnosis exists, it cannot do much harm to use the stomach-tube carefully; but of course the more active general measures indicated in alcoholism should not be used unless there be a clear history of alcoholic poisoning.

Opium.—Still more difficult, in the absence of history, is the diagnosis from opium and other narcotic poisons. Here prompt treatment is essential, and yet certainty in diagnosis may be impossible. Large ventricular or pontine haemorrhages present symptoms much resembling those of opium poisoning, such as deep coma, stertorous breathing, absence of reflexes, and pin-point pupils. The temperature is generally normal, or but slightly lowered, whilst in haemorrhage it is certain to rise, and in most cases rapidly. Initial convulsions constitute rather strong presumptive evidence against opium poisoning, though they are described as occurring sometimes before death, especially in children (Taylor).

Uraemia.—Where, as is often the case, there are repeated epileptoid convulsions, with albuminuria, uraemia is not likely to be confounded with cerebral haemorrhage. But uraemia is sometimes marked by a sudden onset of coma, with rise of temperature, and then the diagnosis is more difficult. Neither is the discovery of albumin in the urine a sure guide, for it may be found in cerebral haemorrhage, either as an antecedent condition or as an effect of haemorrhage.

The other toxaemias, such as cholaemia and diabetic coma, are less likely to be confounded with apoplexy.

General Paralysis.—Reference may here be made to the pseudo-apoplectic attacks occurring in the course of general paralytic dementia. These patients are liable to sudden seizures, which are quite indistinguishable in their symptomatology from true apoplexy, and which may even prove fatal, without, however, presenting any post-mortem evidence of a gross haemorrhagic lesion.

Treatment. — Beyond attention to symptoms, there are two main indications for treatment, namely, (i.) to stop the haemorrhage; (ii.) to relieve intracranial pressure.

Broadly, there may be said to be two extremes of severity of the lesion—those in which the haemorrhage is so large as to be quite beyond the influence of treatment, and those in which the focus is so small as to require no treatment. Between these two extremes, however, there must be a number of cases in which treatment may be of great effect.

(i.) There are two ways in which the cessation of the haemorrhage may be promoted—(*a*) by increasing the coagulability of the blood; and (*b*) by lowering the vascular pressure, even if only for a short time.

(*a*) The time taken by the blood to clot, and the subsequent firmness of the coagulum, vary much in different states of general health. Certain drugs are supposed to affect the clotting power of the blood, and there is a long list of so-called haemostatics, the potency of which in internal haemorrhage is, however, very doubtful. Sir A. E. Wright introduced the use of calcium salts as a means of shortening the coagulation-time of the blood and so arresting haemorrhage.

(*b*) To lower the arterial pressure two means are at our disposal, local and general.

Local.—Sir V. Horsley and Mr. Spencer have shewn, by experiment on monkeys, that haemorrhage from the middle cerebral and its branches may be controlled for a considerable time by compression or ligature of the carotid on the same side; and this in spite of the anastomosis of the circle of Willis. This would suggest the expediency of trying compression of the carotid, provided the diagnosis be reasonably certain, for it is obvious that such a treatment must be directly contra-indicated in thrombosis. Moreover, haemorrhage from the posterior cerebrals would, of course, be unaffected by compression of the carotid.

General.—To bring about a diminution in the arterial pressure the most potent agent, by far, is venesection—or arteriotomy of the temporal artery. One effect of rapid abstraction of blood must be mechanical, with an immediate effect on the intracranial lesion. Both these factors tend in the direction of lowering the arterial pressure, and so, even if temporarily only, of lessening the haemorrhagic effusion, and allowing coagulation to take place.

(ii.) High intracranial pressure is the main cause of coma. In deep coma the reflexes are lowered if not abolished. Hence saliva, secretions, and often food and drink collect in the trachea, and, not being expelled by reflex cough, add greatly to the respiratory embarrassment, and lead to venous engorgement, circumstances which aggravate the already high intracranial pressure.

One effect of abstraction of blood is to lower intracranial pressure, as above stated, often with immediate return to consciousness.

The stertor can be much diminished by attention to the position of the tongue (Bowles), and by placing the patient on his side with the head raised so as to allow fluids to run out. Another very important precaution, while the coma is deep, is to feed only by the nasal tube.

Bleeding in cerebral haemorrhage, as in other conditions, has passed through a long period of disrepute, brought about by its indiscriminate use. Trousseau was among the first to inveigh against it, but it is doubtful whether its discontinuance as a mode of treatment is not almost as discreditable as its abuse. On physiological grounds it is difficult to see that it can do anything but good. It must of course be used with discrimination. A full pulse, a labouring heart, and an engorged condition of the vessels of the neck are among the indications for its employment. It is quite obvious also that the diagnosis of haemorrhage should be fairly well established, for in thrombosis it is distinctly contra-indicated.

Another time-honoured method of lowering intracranial pressure is by purgatives. These probably act thus by dilating the splanchnic vessels. Those most commonly used are croton oil and calomel, which are eminently suitable on account of the ease with which they may be administered and the rapidity of their action.

HOWARD H. TOOTH.

REFERENCES

1. ABERCROMBIE, J. *Pathological and Practical Researches on Diseases of the Brain and Spinal Cord*, 8vo, Edinburgh, 1828. — 2. BASTIAN, H. C. *Paralyses, Cerebral, Bulbar, and Spinal*, London, 1886, 45, 8vo.—3. *Idem. Op. cit.* 155.—4. *Idem.* "Rupture of a Large Aneurism in the Right Corpus Striatum, with Intra-Ventricular Haemorrhage and Extreme Lowering of Rectal Temperature," *Trans. Clin. Soc.*, London, 1884, xvii. 18.—5. BAYLISS, W. M., and L. HILL. "On Intracranial Pressure and the Cerebral Circulation," *Journ. Physiol.*, London, 1895, xviii. 334.—6. BEEVOR, C. E. "On the Distribution of the Different Arteries Supplying the Human Brain" (8 plates), *Phil. Trans.*, London, 1908, B. cc. 1-55.—7. BEEVOR, C. E., and Sir V. HORSLEY. "On Experimental Investigation into the Arrangement of the Excitable Fibres of the Internal Capsule of the Bonnet Monkey (*Macacus sinicus*)," *ibid.*, 1891, B. clxxxi. 49. — 8. BERGMANN, E. "Die Lehre von den Kopfverletzungen," *Deutsche Chirurg.*, Lief. 30, 8vo, Stuttgart, 1880.—9. BISCONS, J. H. A. *De quelques recherches anatomiques et physiologiques sur les artères cérébrales*, 8vo, Bordeaux, 1890 ; also, *Nouveau Montpellier méd.*, 1890, 985.—10. BOURNEVILLE, D. M. B. *Études cliniques et thermométriques sur les maladies du système nerveux*, 8vo, Paris, 1872.—11. BOWLES, R. L. "Observations on Stertor and on the Varying Con-

ditions upon which it is Dependent, with the Treatment Necessary to its Relief," *Med.-Chir. Trans.*, London, 1860, xliii. 41.—12. Brown, S., and E. A. Schäfer. "An Investigation into the Functions of the Occipital and Temporal Lobes of the Monkey's Brain," *Phil. Trans.*, London, 1889, B. clxxix. 303.—13. Charcot, J. M. *Clinical Lectures on Senile and Chronic Diseases.* Transl. by W. S. Tuke, 279, 8vo, London, N.S.S., 1881. — 14. *Idem. Op. cit.* 272. — 15. Charcot et Bouchard. "Nouvelles recherches sur la pathogénie de l'hémorrhagie cérébrale," *Arch. de physiol.*, Paris, 1868, i. 110, 643.—16. Church, Sir W. S. "On the Formation of Aneurysms, and especially Intracranial Aneurysms in Early Life," *St. Barth. Hosp. Rep.*, London, 1870, vi. 99.—17. Cooper, Sir A. *Lectures on the Principles and Practice of Surgery*, 8vo, London, 1824, i. 300.—17*a*. Cushing, H. "The Blood-pressure Reaction of Acute Cerebral Compression, illustrated by cases of Intracranial Haemorrhage," *Amer. Journ. Med. Sc.*, Phila., 1903, cxxv.—18. Dejerine, J., et G. Rousey. "Le Syndrome thalamique," *Rev. neurol.*, Paris, 1906, xiv. 521.—19. Drummond, D. "Abscess in the Right Cerebellar Hemisphere, associated with Paralysis and Fits limited to the same Side," *Lancet*, London, 1894, ii. 190.—20. Duret, H. "Sur la distribution des artères nourricières du bulbe rachidien," *Arch. de physiol.*, Paris, 1873, v. 97.—21. *Idem.* "Recherches anatomiques sur la circulation de l'encéphale," *ibid.*, Paris, 1874, vi. 60, 316, 664, 919. — 22. Eichler, G. "Zur Pathogenese der Gehirnhämorrhagie," *Deutsch. Arch. f. klin. Med.*, Leipzig, 1878, xxii. 1.—23. Fagge, C. H., and P. H. Pye-Smith. *The Principles and Practice of Medicine*, London, 1891, 3rd edit., i. 560, 8vo. —24. Ferrier, D. *The Croonian Lectures on Cerebral Localisation*, 54, 8vo, London, 1890.—25. Gintrac. *Maladies de l'appareil nerveux*, tome ii. 423, 8vo, Paris, 1869. —26. Gowers, Sir W. R. *Manual of Diseases of the Nervous System*, 2nd edit. ii. 423, 8vo, London, 1893.—27. Gull, Sir W. W. "Case of Aneurism of the Cerebral Vessels," *Guy's Hosp. Rep.*, London, 1859, 3rd ser., v. 281.—28. Gulland, G. L. "The Occurrence of Nerves on Intracranial Blood-Vessels," *Brit. Med. Journ.*, 1898, ii. 781.—29. Herringham. "Haematoma of the Dura Mater," *Trans. Path. Soc.*, London, 1895, xlvi. 4.—30. Heubner. "Zur Topographie der Ernährungsgebiete der einzelnen Hirnarterien," *Centralbl. f. med. Wissensch.*, Berlin, 1872, x. 817.—30*a*. Janeway. *Clinical Study of Blood-pressure*, 1904, 189, 248. — 30*b*. Jones, E. *Quart. Journ. Med.*, Oxford, 1910, iii. 233.—31. Kolisko. *Über die Beziehung der Arteria Choroidea Anterior zum hinteren Schenkel der inneren Kapsel des Gehirns*, 1891.—32. Lépine, R. "Note sur les variations de température des membres paralysés, relativement aux membres sains," *Gaz. méd. de Paris*, 1868, 3me ser., xxiii. 501.—33. Mann, J. D. "Notes on a Case of Pure Apoplectic Bulbar Paralysis," *Brain*, London, 1885, vii. 244. — 34. Mendel, E. "Über die Apoplexia Cerebri Sanguinea," *Berlin. klin. Wchnschr.*, 1891, xxviii. 577.—35. Morison, A. "On the Innervation of Intracranial Vessels," *Edinb. Med. Journ.*, 1898, N.S. iv. 413.—36. Mott, F. W., and E. A. Schäfer. "On Movements resulting from Faradic Excitation of the Corpus Callosum in Monkeys," *Brain*, London, 1890, xiii. 174.—37. Munk. "Die Funktionen der Grosshirnrinde," *Gesammelte Mitteilungen*, Berlin, 1890.—38. Nothnagel, H. "Anaemia, Hyperaemia, Haemorrhage, Thrombosis, and Embolism of the Brain," *Cyclopaedia of the Practice of Medicine*, edit. by H. von Ziemssen, transl. by A. H. Buck, xii. 1, 8vo, London, 1877.—39. Obersteiner, H. "Die Innervation der Gehirngefässe," *Neurol. Centralbl.*, Leipzig, 1897, xvi. 356.—40. Pagenstecher, F. *Experimente und Studien über Gehirndruck*, 8vo, Heidelberg, 1871.—41. Pitres, J. A. *Recherches sur les lésions du centre ovale des hémisphères cérébraux étudiées au point de vue des localisations cérébrales*, 4to, Versailles, 1877.—42. Prevost, J. L. *De la déviation conjuguée des yeux et de la rotation de la tête dans certains cas d'hémiplégie*, 4to, Paris, 1868.—43. Revilliod, L. "Hémiplégie gauche chez une gauchère. Embolie du tronc brachio-céphalique, signe de l'orbiculaire," *Rev. méd. de la Suisse Rom.*, Genève, 1889, ix. 595.—44. Russell, J. S. R. "The Value of Experimental Evidence in the Diagnosis of Diseases of the Cerebellum," *Brit. Med. Journ.*, 1895, i. 1079, 1240.—45. Speil, E. "De la respiration du sang dans l'encéphale ; expériences faites au laboratoire de physiologie de l'université de Bruxelle," *Encéphale*, Paris, 1887, vii. 55.—46. Spencer, W. G., and Sir V. Horsley. "On the Changes produced in the Circulation and Respiration by Increase of the Intracranial Pressure or Tension," *Phil. Trans.*, London, 1892, B. clxxxii. 201. — 47. *Idem.* "Report on the Control of Haemorrhage from the Middle Cerebral Artery and its Branches by Compression of the Common Carotid," *Brit. Med. Journ.*, 1889, i. 457.—48. Tedeschi, A. "Contributo allo studio della circolazione cerebrale," *Neurol. Centralbl.*, Leipzig, 1891, x. 142 [Abstr.].—49. Trousseau, A. *Lectures on Clinical Medicine*, i. 7, 15, 8vo, London, N.S.S., 1867.—50. Veyssière, R. *Recherches cliniques et expérimentales sur l'hémianesthésie de cause cérébrale*, 8vo, Paris, 1874.—51. Weber, Sir H. "A Contribution to the Pathology of the Crura Cerebri," *Med.-Chir. Trans.*, London, 1863, xlvi. 121.—52. Wigglesworth, J. "Remarks on the Pathology of So-called Pachymeningitis Interna Haemorrhagica," *Brain*, London, 1892, xv. 431.—53. Wilson, S. A. K. "A Study of Apraxia," *ibid.*, 1908, xxxi. 164.—54. Wright, Sir A. E. "Upon a New Styptic, and upon the Possibility of Increasing the Coagulability of the Blood in the Vessels in Cases of Haemophilia and Aneurism and Internal Haemorrhage," *Brit. Med. Journ.*, 1891, ii. 1306.—55. Wynne, E. T. "Subdural Haemorrhage," *Trans. Path. Soc.*, London, 1892, xliii. 1.

H. H. T.

INTRACRANIAL SYPHILIS

By William Aldren Turner, M.D., F.R.C.P.

One effect of the syphilitic poison in the causation of nervous disease is the production of a specific inflammatory lesion of the blood-vessels and membranes of the brain and spinal cord. These changes present the histological features exhibited by syphilis elsewhere in the body. The nervous tissues are interfered with through diminution or arrest of the blood supply, and by the effects of pressure from gummatous formations. As the morbid anatomy and microscopical appearances of syphilitic disease of the blood-vessels have been fully described by Dr. Mott in Vol. VI. p. 562, attention will, as far as possible, be confined to the clinical symptoms of intracranial syphilitic affections. As it is impossible to adopt an arrangement of description, independent of the underlying pathological condition, the two series of phenomena have been given in a table on pp. 337, 338.

Etiology.—*Time of Onset.*—From a large number of observations, the general statement may be made that the effects of syphilis upon the nervous system may appear within a comparatively short period after infection.

Table shewing the Time of Onset of the Disease of the Nervous System after Infection (copied from Ogilvie, *Lancet*, 1895, i. 1368).

During 1st half-year.	During 2nd half-year.	During 2nd and 3rd years.	During 4th and 5th years.	From 6th to 10th years.	From 11th to 15th years.	From 16th to 20th years.	After 20th year.	Summary.
per cent.	per cent.	per cent.	per cent.	per cent.	per cent.	per cent.	per cent.	
11	14·4	18·6	15·7	24·6	8·6	4·4	2·8	70 necropsies.
11·4	8·6	19·7	15	24·9	10·7	5·2	4·3	325 cases, including necropsies.

From these data it is clear that of 70 cases of cerebral syphilis submitted to post-mortem examination, 44 per cent occurred within the first three years and 59·7 per cent within the first five years following infection.

Sir W. Gowers has stated that of 50 cases of cerebral endarteritis causing hemiplegia, present in persons from twenty-five to forty-five years of age, 25 per cent occurred during the first two years after syphilitic infection; whilst the remainder were spread over the next twelve years. Rumpf gives 23 per cent of the cases of syphilitic affections of the membranes of the brain and spinal cord as occurring within the first year.

The following TABLE shews the time of Onset of Nervous Symptoms after Infection in 34 cases of Cerebrospinal Syphilis, personally observed.

Years after Infection.	1-2	2-4	4-6	6-8	8-10	Over 10.
Number of Cases.	7	4	3	3	3	14

It is clear therefore that, so far from being an indication of the deferred action of the syphilitic virus, symptoms pointing to intracranial disease are in at least half the cases evidence of an early affection of the blood-vessels and membranes of the brain.

The *age* of the patient at the time of onset of the nervous phenomena is also a point of interest. It is stated that the older the patient the shorter the interval between infection and the onset of cerebral symptoms. In 4 cases out of the original series, in which syphilis was contracted after the age of thirty-five years, in two instances symptoms pointing to a cerebral affection came on two years after infection, and in the other two five years afterwards. Of the 34 cases, the onset of the nervous symptoms occurred between the ages of thirty and forty years in 17; 12 of them being between thirty-five and forty years of age. Of the 7 cases in which cerebral symptoms ensued before the age of thirty, the period between this and infection ranged from eighteen months to three years; so that the view just expressed does not receive confirmation from this series.

As regards other points of causation, data have been brought forward by some authors to shew that injury plays an important part in exciting the cerebral affections; and it is a common observation that a circumscribed patch of syphilitic pachymeningitis is not infrequently situated in the neighbourhood of an old injury to the skull. The disease has also been stated to occur more commonly in brain-workers.

It would appear that in the majority of cases of intracranial syphilis the immediate causes cannot be ascertained; the meningeal and vascular affections being idiopathic and spontaneous; but the most likely cause is neglect of or insufficient treatment in the early stages of the disease.

We have not enough evidence to shew how far a predisposition to mental and nervous diseases favours the occurrence of cerebral syphilis.

It is sometimes stated that those who suffer from nervous symptoms in the later stages of syphilis have passed through a mild form of the disease in the earlier; so mild in some cases that the primary sore and the cutaneous and other secondary symptoms have been overlooked. Thus in many tabetics and general paralytics it is often difficult to obtain a definite history of the early syphilitic symptoms. But it does not seem that this observation holds good in the cases of "specific" affections of the nervous system, for in these persons a clear history of antecedent syphilis is usually obtained. Thus, in 7 of the 34 original cases special mention was made of the character of the secondary phenomena, and of the nature and duration of the treatment. In these the secondary symptoms appeared to be of average severity, and treatment by mercury and iodide of potassium, or by a combination of these, was continued for six months—the shortest period—up to twenty-one months. Yet, notwithstanding this antisyphilitic treatment, symptoms indicative of a vascular lesion declared themselves; and in one case hemiplegia ensued during the administration of iodide of potassium. Sir J. Hutchinson records a case in which the prolonged administration of antisyphilitic remedies did not prevent the onset of paraplegia, which took place indeed during the administration of the drugs.

Symptomatology.—*General Characters.*—Although the name "cerebral syphilis" is used to denote a series of symptoms arising from the action of the specific virus upon the blood-vessels and membranes of the brain, yet it can be laid down as a general principle that the phenomena thus produced do not differ materially from similar effects arising from other causes. Thus, hemiplegia occasioned by syphilitic arteritis and thrombosis is, in essential features, similar to that arising from non-specific vascular occlusion; localised convulsions, arising from the presence of a gumma, are in nowise different from those caused by a glioma or tuberculous tumour in a similar situation; and the oculomotor palsies due to syphilitic meningitis agree in character with those originating in causes of a non-specific nature.

Although the clinical features of paralysis, or other symptoms arising from syphilitic vascular or meningeal lesions, do not differ from those due to other causes, they frequently present two characteristics suggesting their syphilitic nature; the one being a tendency to multiplicity or "random association" of symptoms, the other a tendency to remission and relapse. Thus, an incomplete hemiplegia of more or less gradual onset may disappear, to be followed within a longer or shorter time by a similar attack either upon the same or opposite side, which in its turn resolves and may for a third time return; and the same applies to convulsive symptoms.

In this category also may be placed the fleeting and variable oculomotor palsies—sometimes in one eye, sometimes in the other—which give rise to diplopia and not uncommonly herald the approach of more serious intracranial disease.

The more pronounced symptoms of cerebral syphilis are usually preceded for a longer or shorter period by headache and insomnia; and, as these are ordinarily the danger-signals in the majority of such cases, the early recognition of their cause is of the utmost value.

(*a*) *Headache.*—This is an early, if not the earliest, indication of syphilitic cerebral affection. Such headache is characterised by its great intensity, and by its tendency to increase towards evening and in the early morning, whereas during the day the patient is relatively free from pain. The headache may be general, or it may be more or less limited to one side of the skull; in some cases it is more pronounced in the frontal, in others in the parietal or occipital regions; and there is often a tender area on cranial percussion. It may precede the onset of hemiplegia, or aphasia, or a sudden attack of mental confusion, or an epileptiform attack, by a few weeks or even by several months. It is usually present, but well-marked cases of cerebral syphilis are met in which neither headache nor other prodromal phenomena are noted. In many cases with the onset of graver symptoms the headache disappears, or materially lessens in severity. The pathological cause of this headache is indefinite. In many cases it is probably of a neuralgic nature, but in others is due to an inflammation of the cranial periosteum.

(*b*) Commonly associated with headache, and largely due to it, is *insomnia.* The former is rarely present without the latter, but sleeplessness in some cases may exist without headache.

(*c*) Of other and rarer prodromal symptoms may be mentioned *vertigo* and *mental apathy.* The latter, characterised chiefly by lowered cerebral activity and want of attention, may, however, be so pronounced as to indicate a state of mental stupor. In some instances various further psychical conditions precede cerebral syphilis, or may be the actual onset of it.

Although such prodromal signs may be inconstant and of varying intensity, it is rare for all of them to be absent. It is, therefore, unusual to find a case in which severe cerebral implication of syphilitic nature is primarily manifested by hemiplegia, aphasia, or an epileptiform attack.

Clinical Forms.—In attempting to describe different clinical types of syphilis as it affects the cranial contents, and the relation of such types to several specific morbid states, a difficulty at once presents itself; for although certain phenomena point, more or less precisely, to the predominance of this or that pathological condition, on necropsy it is rare to find the condition so limited. For example, a case presenting the clinical features of gumma may shew after death an extensive associated meningeal affection; or a case in which the symptoms pointed to vascular obstruction may shew more or less gummatous infiltration. The following is a suitable basis for description, founded upon both clinical and pathological data:—

1. Symptoms arising from vascular lesions:—

Hemiplegia and aphasia.	Perivascular infiltration and endarteritis obliterans.

2. Symptoms due to gummas :—

Focal epilepsy and hemiplegia.	Gummas of cortex and subcortical tissues.

3. Symptoms arising from meningeal lesions :—

Focal epilepsy. Monoplegias, aphasia. Palsy of the cranial nerves.	Leptomeningitis and pachymeningitis of cortex and base of brain.

4. Symptoms arising from diffuse lesions :—

Syphilitic dementia.	Meningitis and vascular occlusion.

(i.) *Symptoms arising from Vascular Lesions.*—The two symptoms pointing most conclusively to arterial obstruction are *hemiplegia* and *aphasia.*

Hemiplegia of syphilitic origin presents the features seen in that resulting from vascular disease due to other causes, and shews all varieties, from the slight and incomplete to the severe and complete forms. Its onset is rarely attended by unconsciousness, and is seldom without warning, not uncommonly it is preceded by headache, it is usually of a temporary character, and, after recovery, it may be followed by hemiplegia either upon the same or the opposite side of the body.

Arterial occlusion arising from syphilitic periarteritis and endarteritis is productive of two series of phenomena: those of a temporary or transient nature, arising from ischaemia (local anaemia) of the cerebral centres; and those of a permanent and enduring character, caused by more or less complete obliteration of the vascular lumen, with consequent softening. Amongst the former are placed transient and incomplete hemiplegia, or aphasia, or confusion of mind lasting from a few minutes to as many hours, with complete recovery; temporary attacks of vertigo, of loss of consciousness, and of unilateral numbness or paraesthesia.

On the other hand, one-sided attacks of numbness and tingling may precede for some days the onset of a transient, or a permanent, loss of power on the same or on the opposite side. Or, again, symptoms of arterial ischaemia may occur synchronously with those of basal meningitis.

Many end in permanent paralysis from a cerebral softening which differs in no respect from that seen in other conditions.

All forms of aphasia may occur from vascular occlusion, not only of the motor type, but also the sensory forms—word-deafness and word-blindness. As with loss of voluntary power, aphasia may be either temporary or permanent, depending upon the completeness of the vascular lesion.

The symptoms above described are indicative of disease of the middle cerebral artery and its branches, this being the vessel most commonly affected by syphilitic arteritis; next in frequency come the basilar artery and its branches. The effect of vascular lesions therefore depends upon their degree and situation in the affected blood-vessel, the symptoms being referred to any part of the central nervous system. Syphilis is the common cause of vascular disease in the young, and in any doubtful case a cytological examination of the cerebrospinal fluid should be made.

In order to establish the syphilitic nature of the symptoms of vascular lesions, besides obtaining a history of syphilitic infection, the presence of chronic disease of the kidneys should be eliminated, although this complication is not uncommon in old-standing cases, and in cases arising late in life.

Cerebral haemorrhage arising directly from syphilitic causes is rare, unless the arterial degeneration has led to the formation of an intracranial aneurysm, in which case the basilar artery is the one most commonly affected. In this event rupture, with consequent extravasation of blood, either into the brain substance or subdural space, is the usual mode of death. Intracranial aneurysms are, according to statistics, commoner in the degenerative period of life; yet they are not uncommonly met with in young adults, both male and female. In such cases, if heart disease can be excluded with confidence, syphilis is usually the cause.

(ii.) *Symptoms arising from Gummas of the Cerebral Cortex, Cortical Membranes, and Arteries.*—Under this heading are considered symptoms of convulsive and paralytic nature such as partial, localised, or Jacksonian attacks, hemiplegia and monoplegia of cortical nature, and aphasia.

A gummatous new growth is productive of symptoms similar to those of any other cerebral tumour in a like locality. These are the general symptoms of intracranial new growth with focal or localising phenomena. Thus, an epileptiform attack may involve the arm, or leg, or the face and arm, or the whole of one side of the body. These symptoms are sometimes accompanied by the mental apathy and loss of memory and attention observed in cerebral syphilis, and are not uncommonly associated with optic neuritis.

As in Jacksonian epilepsy arising from non-specific lesion, a partial or completely unilateral convulsion is usually followed by temporary paralysis. Such palsies differ from those caused by arteritis—with local anaemia—in their gradual incidence, in their longer duration, and, if complete, in heir more certain amelioration under antisyphilitic treatment.

Syphiloma arises most commonly from the cerebral meninges, and is consequently found to occupy a superficial position. If found deeply in the cerebral substance, such as the centrum ovale or the basal ganglia, the growths have arisen from an ingrowth of the pia-arachnoid, or from the adventitia of a blood-vessel.

Although *gumma of the cerebellum* is rarely found on post-mortem examination, symptoms pointing to a lesion of the cerebellum are not uncommon in cerebral syphilis. The cerebellar arteries also may be the seat of obliterative endarteritis, and from their occlusion give rise to softening of the cerebellar hemispheres.

This is a convenient place to refer briefly to the connexion, if any, between syphilis and the production of epilepsy. It is generally acknowledged that epilepsy may occur in the subjects of inherited syphilis without any other obvious reason, although this association is remarkably rare. Fournier is of opinion that true epilepsy may occur in the so-called secondary stage of syphilis, as a direct result of the syphilitic intoxication; just as cases of syphilitic insanity have been described during the eruptive stage of the disease: but evidence on this point is extremely meagre. Whether syphilitic infection may of itself lead to the establishment of epileptic fits, or of insanity, without organic injury to a part or parts of the brain, is doubtful; but convulsions indistinguishable from those of epilepsy not uncommonly follow the damage to the brain consequent upon cerebral syphilitic lesions.

(iii.) *Symptoms arising from Meningeal Lesions.*—When the lesions are cortical in situation, the symptoms are those of focal epilepsy, corresponding to the cortical region irritated. The chief symptoms of syphilitic basal meningitis are paralyses of the cranial nerves.

If the motor region is the seat of irritation, focal fits occur in the opposite limbs; if the occipital region is the seat of lesion, visual spectra and subjective visual sensations are described in the opposite half-fields.

One of the commonest positions for a basal pachymeningitis is the interpeduncular space and the region of the optic chiasma.

The nerves most commonly affected are the optic, third, and sixth. All degrees of impaired visual acuteness may be met with up to blindness. Oculomotor palsies present all degrees from complete ophthalmoplegia to palsy of a single branch. A common symptom is palsy of a branch or branches of the third nerve, or of the trunk of the sixth nerve on one side. Such palsies are usually associated with headache, and occasionally also with graver symptoms of cerebral syphilis, such as vertigo, hemiplegia, partial convulsions, and optic neuritis.

Common also is an incomplete or partial affection of a nerve-trunk; thus, in one case the patient complained of double vision, in another unilateral mydriasis with palsy of accommodation summed up the extent of the affection; whilst in others slight ptosis, with or without palsy of accommodation, was the only indication of an affection of the third nerve. Such palsies commonly resolve under antisyphilitic treatment, and leave no trace of their existence. They should, however, be carefully distinguished from the temporary and often fleeting oculomotor palsies met with as early phenomena in tabes dorsalis and disseminated sclerosis. It is clear, therefore, that palsy of a branch or branches of the third nerve, occurring in a syphilitic subject, is a danger-signal of the utmost value, as it may be a forerunner of more serious organic disease, whether of a syphilitic or of a degenerative nature.

It is hardly necessary in this place to do more than refer to the *ophthalmoplegias*, which have been fully described elsewhere (Vol. VII. p. 512). Chronic progressive ophthalmoplegia is in some cases of syphilitic origin, although in numerous instances such a cause may be definitely excluded. A form of subacute ophthalmoplegia is not infrequently met with in consequence of syphilitic vascular disease with thrombosis of the Sylvian branches of the basilar artery.

Palsy of the *trigeminal* nerve, as an isolated symptom, is found in cerebral syphilis. Here the lesion is of meningeal origin, and is to be found involving the trunk of the nerve as it leaves the side of the pons Varolii, or implicating the Gasserian ganglion in the middle fossa. It is also met with in association with other symptoms, such as hemiplegia, indicative of a central lesion either gummatous or vascular in nature. More commonly, however, this nerve is found paralysed, in association with other adjacent cranial nerves, from meningeal lesions of the middle and posterior cranial fossae; thus, the fifth and the seventh nerves may be paralysed together on one side; if the disease be limited to the middle fossa, palsy of the fifth and sixth nerves is observed; or when the disease involves the posterior fossa trigeminal palsy may be found associated with palsy of the bulbar nerves.

Palsy of the trigeminal nerve is indicated by the usual phenomena of anaesthesia over the cornea, face, and mucous membranes supplied by this nerve; and, if the motor branch be involved, by paralysis of the masticatory muscles. In the earlier stages, however, numbness and neuralgic pains may be noted; and also neuro-paralytic keratitis as an irritative phenomenon.

Paralysis of the *seventh nerve*, as a result of basal syphilitic meningitis, is not uncommon; and, in proportion to the number of purely peripheral facial palsies of syphilitic nature, is relatively frequent. A basal syphilitic palsy of the facial nerve may be associated with palsy of the *auditory* nerve on the same side.

A unilateral palsy of the *bulbar nerves*—vago-glossopharyngeal and hypoglossal—although not frequently met with, is suggestive of a syphilitic basal pachymeningitis. Palsy of these nerves gives rise to the characteristic symptoms of palsy of the soft palate, of the vocal cord, and of the tongue (with hemiatrophy) on the side of the lesion. Not uncommonly also, owing to the intracranial course of the spinal accessory nerve, paralysis of the sterno-mastoid and of the upper part of the trapezius muscles is found in association with the other phenomena.

(iv.) *Symptoms arising from Diffuse Lesions—Syphilitic Dementia; Pseudo-general Paralysis.*—Clinical experience shews that the mental symptoms occurring in cases of cerebral syphilis are not necessarily due to any one particular form of specific lesion. Hence, this subdivision of the phenomena of cerebral syphilis is one of much difficulty and some obscurity; for, apart from the general mental symptoms, a certain morbid state has been described under the term of syphilitic pseudo-general paralysis or syphilitic dementia.

The clinical features of the diffuse cerebral type are the following: the early appearance of cranial nerve paralyses, especially of the third and sixth nerves, characteristic pupillary changes (inequality, irregularity, and impaired light-reaction), the occurrence of general or localised epileptic seizures, often followed by paralysis with definite signs of organic disease. Headache, vomiting, and optic neuritis may be superadded in some cases.

The symptoms which characterise the syphilitic form may be distinguished from those of the ordinary disease by the absence of ambitious conceptions; and tremor, so essential a feature in the diagnosis of general paralysis proper, may be absent, or only slight, in the form under consideration. Again, the presence of local palsies, such as ptosis, hemiplegia, or aphasia, may lead one to suspect a syphilitic meningeal or vascular condition, rather than a purely degenerative process in the cerebral cortex. As regards the mode of onset, in the specific form it is sudden, of an epileptiform or apoplectiform character; its duration and course are ill-defined, the prognosis is often good up to a certain point, and the disease is not progressively fatal.

But notwithstanding such points of distinction, it is impossible, in the great majority of cases, clearly to define the process underlying the symptoms; for many cases of true general paralysis present in a marked degree all those phenomena which are said to be characteristic of the diffuse form.

The methods of cytological examination of the cerebrospinal fluid and the serum diagnosis of Wassermann have to some extent rendered the differential diagnosis of these two conditions more certain. In the diffuse cerebral form there is a definitely lesser degree of lymphocytosis of the cerebrospinal fluid than in true general paralysis of the insane, and the absence of a Wassermann reaction of this fluid (Mott).

But a point of importance is that we may meet with cases presenting mental and motor phenomena, in which on necropsy the cerebral cortex is relatively free, whilst the observed lesions are vascular and meningeal in nature, and basal in position. Thus, in one case the symptoms were chiefly mental throughout the whole course of the disease, which lasted fifteen months. The onset was sudden and characterised by a series of epileptiform convulsions; these were succeeded by an attack of acute mania, with incoherence of speech, restlessness, great excitability, and loss of memory; but there was no marked headache, and the other prodromal symptoms were not apparent. After this sudden onset the patient became mildly demented, rambling in speech, and defective in memory; he slept badly, and there was occasional muscular twitching. One year after the onset ptosis of the right upper eyelid appeared, the gait became staggering, the articulation defective, and the mental impairment progressed towards fuller dementia. The necropsy revealed basal meningitis and softening of the right cerebellar hemisphere from vascular obstruction, whilst the cortex cerebri was practically normal.

From this and similar cases the conclusions may be drawn:—

(*a*) That the mental symptoms occurring in cerebral syphilis arise from diffuse meningeal and vascular changes, and are not associated with any one particular lesion.

(*b*) That in many cases of cerebral syphilis the mental symptoms are pronounced and suggest the onset of general paralysis, but shew a marked tendency to resolve under general antisyphilitic treatment.

(*c*) That it is difficult to distinguish the false from the true general paralysis, for some cases presenting early symptoms, which suggest the syphilitic variety, eventually pass into the true form, and conversely.

(*d*) In all cases presenting difficulty in diagnosis, a cytological examination of the cerebrospinal fluid should be made, and the Wassermann reaction taken.

Incidental mention has already been made of the *mental* phenomena occurring in cases of cerebral syphilis; and it is from the coexistence of such symptoms with others of a more purely motor or sensory nature that this condition is often recognised. Loss of memory, failure of the power of attention, with its associated impaired capacity for work, often accompanied by attacks of confusion of thought, verbal amnesia, and even temporary lapses of consciousness, with or without subsequent transient aphasia, point in a suggestive manner to some specific affection. With such symptoms there is commonly a change in character and disposition, and a curious tendency to somnolence, so that the person drops off to sleep while at work—a condition which may be so pronounced as to be classified under the name *stupor*.

Prognosis.—Although the physician may be guided in the prognosis of cerebral syphilitic affections by certain general facts, gleaned from the statistics and personal observations of other workers, he will find eventually that each case coming under his notice must be studied and judged upon its own characters. For, as Sir W. Gowers has insisted, much depends on the nature of the pathological change which has already taken place; an inference which can only be determined by a careful consideration of all the details of individual cases. Thus, the tissue hyperplasia which occurs in the early stages of the disease is removed with comparative ease; caseation may undergo absorption, either directly or indirectly as the result of treatment; but fibroid transformation and necrosed tissue, the results of vascular obstruction, are irremediable.

Hence we find that cortical lesions of the nature of gummatous new growth, or meningitis of a gummatous nature, occasioning symptoms of Jacksonian epilepsy and localised paresis, present an especially favourable prognostic outlook. But it is a matter of some importance to note that should the cranial vault also be largely involved in the specific process the prognosis is distinctly less favourable (Rumpf).

Relatively favourable also are basal specific lesions, more especially of the meninges, the majority of the cranial nerve palsies of syphilitic nature being included in this category. Of essentially unfavourable prognosis are vascular lesions, in whatsoever part of the brain they may occur. Hence the permanent effects of cerebral syphilis are obviously of vascular nature.

But it will not be out of place in this relation to give some statistical data for general guidance. From 90 cases observed by Fournier, and 34 observed by Rumpf, the following facts as regards recovery appear :—

	Fournier.	Rumpf.
Total	90	34 cases
Died	14	5 ,,
Cured	30	12 ,,
Incomplete cures	13	6 ,,
Permanently maimed	33	8 ,, (result unknown in 3)
Percentage of recoveries	33·3	35·2

Of the 34 original cases, to which reference has previously been made, 14 were observed over a sufficiently long time to be of service from the prognostic point of view. Of these, 8, presenting the features of hemiplegia, aphasia, and oculomotor palsy, shewed no symptoms of the original disorder at periods varying from six months to five years after the onset of cerebral symptoms. Hence, for statistical purposes, they are regarded as "cures." One patient died sixteen months after the onset of the symptoms, and four were permanently maimed. The percentage of recovery in these cases is 57·1 per cent—a somewhat higher figure than that given by Fournier and Rumpf, which probably depends on the fact that they were not observed over a sufficiently long period. From Naunyn's tables, quoted by Dr. G. Ogilvie, it does not appear that there is any material difference in the percentage of cured and uncured cases, whether considered from the age at which the symptoms manifest themselves, or whether they occur within a short interval or after an interval of ten years from first infection.

In estimating the prognosis of cerebral syphilis the tendency to relapse and remission is to be borne in mind. As already explained, this is a characteristic feature of the condition, and as such plays an important part in prognosis.

Treatment.—There are no rules for the treatment of syphilitic affections of the nervous system which are not applicable to the treatment of syphilis generally. Attention is here directed to a few points concerning the methods of administration of the antisyphilitic remedies.

Of the first importance is placed *mercury* and its preparations. It may be given in various ways: by the mouth, by inunction, or by intramuscular injection. Each plan is lauded by its advocates, who readily find defects in alternative methods. It would appear to be near the truth to say that each method has its special advantage and use in certain cases, and each has its disadvantages. The chief disadvantages of the ingestion method are the uncertainty in the action of the drug and a tendency to troublesome forms of intestinal derangement. The two preparations used for this purpose are, corrosive sublimate (gr. $\frac{1}{12}$-$\frac{1}{6}$), or as the liquor hydrarg. perchlor. (30-60 m.), and the green iodide of mercury (gr. $\frac{1}{6}$-1).

Inunction is largely practised at the spas to which syphilitic patients resort. Fournier recommends that from 5 to 10 grams of mercurial ointment be rubbed in daily, preferably after a hot bath. Different portions of the body should be selected on consecutive days for the inunction. It is difficult to fix a period for the duration of this treatment; but fifty to sixty inunctions are usually sufficient. At Aachen a course of treatment consists usually of sixty immersions in the hot sulphur water, followed by sixty inunctions, each of 5 grams of mercurial ointment. *Intramuscular injections of mercurial salts* are strongly advocated by some writers. The chief advantage of this method is that a known quantity of the drug is given, all of which is absorbed; hence smaller doses are sufficient, and a less frequent application is necessary (one injection per week being counted sufficient by those who use the method).

Col. Lambkin has strongly recommended a preparation of metallic mercury in doses of 1 grain; but other insoluble mercurial preparations which may be used in this way are: salicylate of mercury (gr. $\frac{1}{2}$) and calomel (gr. $\frac{1}{2}$). The soluble preparations of mercury used for intramuscular injections are the perchloride (gr. $\frac{1}{8}$), succinimide (gr. $\frac{1}{6}$), and cyanide (gr. $\frac{1}{4}$).

Next in importance come the *iodides of potassium, sodium, and ammonium.* They are of special value in syphilitic affections of the nervous system and in the later stages of the disease. The dosage of these drugs varies considerably in different countries. Thus, in America enormous doses are administered, 120 to 150 grains of iodide of potassium thrice daily being no uncommon amount. In France, doses of considerably less magnitude are given, but on the whole larger than is customary in this country, for example, 2 to 10 or 12 grams per diem (30-180 grains). In own our country the dosage also varies within considerable limits. Some physicians are content with 5 to 7 grains thrice daily; others consider that 15 to 20 or 30 grains of potassium iodide given thrice daily, or, better still, every six hours, will produce as marked a benefit as the larger doses already mentioned. There is one important point to be noted, namely, that *iodism* is much more likely to occur from the use of small than of large doses; and the addition of an ammonium salt is recommended to counteract the depressing effect, as well as to permit, according to some, the administration of smaller doses of the iodides. Probably the most satisfactory method of treating cerebrospinal syphilis is by the internal administration of the iodides along with mercurial inunctions.

A number of new preparations, having arsenic as their chief ingredient, have lately been introduced. Atoxyl, arsacetin, and soamin may be mentioned. It is too soon to express a definite opinion upon their action, but toxic effects have been recorded as following their administration in some cases.

A word may be said as to the use of *natural baths* in the treatment of cerebral syphilis. It appears that the sulphur baths hasten the elimination of mercury and its products from the system, in this way enabling smaller doses of mercury to be given. Owing to the sulphurous nature of the waters the spas most popular for this condition are: in America, the hot springs of Arkansas; in Russia, Abastuman and Piatigorsk; in

Germany, Aachen (Aix-la-Chapelle); and in France, Luchon and the Pyrenean spas.

In conjunction with the general therapeutic measures just described, *local treatment* of paralysis, by means of massage, passive movements, and the faradic currents, should be carried out over long periods of time.

William Aldren Turner.

REFERENCES

1. Fournier. *Syphilis du cerveau*, Paris, 1879.—2. Gowers, Sir W. *Syphilis of the Nervous System*, London, 1890.—3. Hutchinson, Sir J. *Syphilis*, London, 1889.—4. Lambkin. *System of Syphilis*, London, 1908.—5. Mott. "Morison Lectures," *Arch. Path. Lab. Lond. County Asyl. Claybury*, London, 1909, iv. 13.—6. Ogilvie, G. *Lancet*, London, 1895, i. 1368.—7. Rumpf. *Die syph. Erkrank. des Centralnervensystems*, 1887.

W. A. T.

GENERAL PARALYSIS OF THE INSANE

Synonyms.—*Dementia paralytica; Progressive allgemeine Paralyse der Irren.*

By G. H. Savage, M.D., F.R.C.P.
Pathological Section by E. Goodall, M.D., F.R.C.P.

General description — Etiology — Morbid Anatomy — Pathogeny — Warnings (prodromes) — Invasion — Course — Clinical groups — Duration—General and special symptoms—Remissions—Arrests—Terminations—Diagnosis—Treatment: general and special—Moral responsibility and testamentary capacity.

General Description.—General paralysis of the insane is a progressive disease or degeneration which occurs most commonly in married men of middle age, living in cities, taking both meat and alcohol, and exercising their reproductive functions. It is marked by progressive degradation of all the mental and bodily functions, beginning with the most highly developed and specialised, and ending on the one part in complete mental defect (dementia), and in paralysis on the other. It must be considered incurable, though during its course, which averages three years, there may be remissions more or less complete. It consists of several more or less definite stages. It may exhibit any form of mental disorder, though mania with wild extravagance of ideas is the most common. During its course fits of various kinds may occur. The characteristic bodily symptoms may exist for long periods without any evident mental affection; but I do not recognise a general paralysis apart from that of the insane: we meet indeed with many cases in the public hospitals, presenting all the physical signs of general paralysis, in which there is no sign of insanity; yet if these patients live long enough they will pass into some degree of dementia.

Nevertheless, we may ask ourselves if the group of symptoms usually designated by the name "General paralysis of the insane" is sufficiently uniform to deserve a specific name. I own that I cannot look upon "general paralysis" as a very definite disease; but as other degenerations in their advance follow certain lines (the lines of least resistance), so it is in general paralysis. All diseases vary in their individual instances, though the instances bear a general similarity to each other. In my opinion, however, more than one distinct kind of degeneration is classed under the present name, besides the numberless varieties which arise from special conditions in the cause or environment. As normal individuals vary as the result of heredity, education, and general surroundings, so the symptoms and course of cases of general paralysis vary with the hereditary qualities of the patient, the proximate causes of the disease, and the conditions under which the degeneration has taken place. For instance, I believe that the general paralytics who own a neurotic heredity differ somewhat in the course of their disease from those who have no such heredity; and again that the course of the disease which depends on syphilis alone differs from that which results from syphilis and injury, or syphilis and alcohol.

No precise description of the ruin of a house can be laid down beforehand, save that the weakest parts will go first; the paths of destruction will vary with the adverse circumstances; the cottage which on a lone moor fails first in the roof would, if near a town, have its windows first destroyed. In the same way the decay of the general paralytic who passes a quiet life will differ greatly from that of one who lives at large, subject to many social and moral exigencies.

I look upon general paralysis of the insane as a degeneration of all parts of the nervous system, though certain portions suffer more than others; and in the various cases the degeneration starts in different parts and proceeds at different rates. The disease usually begins either as the result of local injury or local strain, and has a preference for the last developed and most specialised parts of the nervous system.

I submit a table from the Report for 1908 of the Commissioners of Lunacy shewing the proportion of general paralytics compared with the other forms of mental disorder.

TABLE shewing by yearly average the number of Pauper Lunatics admitted during the five years 1902 to 1906 into County and Borough Asylums, Registered Hospitals, etc., according to sex and forms of Insanity.

Forms of Mental Disorder.	Total.		
	M.	F.	T.
Congenital or infantile mental deficiency	677	501	1,178
Mania, either acute, chronic, or recurrent	3616	4166	7,783
Melancholia, either acute, chronic, or recurrent	2394	3524	5,918
Delusional Insanity	356	349	705
General Paralysis of the Insane	1099	231	1,331
Primary Dementia	252	219	471
Secondary Dementia	410	333	743
Senile Dementia	489	555	1,043
Organic Dementia	170	92	262
Total	9464	9970	19,434
Proportion per cent of Pauper Patients to Total Number of Admissions			
Congenital or infantile mental deficiency	7·1	5·0	6·1
Mania, either acute, chronic, or recurrent	38·2	41·8	40·0
Melancholia, either acute, chronic, or recurrent	25·3	35·4	30·5
Delusional Insanity	3·8	3·5	3·6
General Paralysis of the Insane	11·6	2·3	6·9
Primary Dementia	2·7	2·2	2·4
Secondary Dementia	4·3	3·3	3·8
Senile Dementia	5·2	5·6	5·4
Organic Dementia	1·8	·9	1·3
Total	100	100	100

I shall point out later that other forms of degeneration, such as that connected with senile changes, run very similar courses, but yet there are differences.

Special vital conditions dispose to this disease, which is one associated directly with the modes of life met with in highly civilised states. It is most common in men of middle age, living in cities, who have lived freely; that is, who have taken a good deal of meat, have used stimulants, and have exercised their sexual functions freely: but beside all this there must, in the majority of cases, have been a peculiar determining cause of the breakdown. There is a pretty general consent of opinion at present that syphilis, if not the immediate cause, is a proximate one of great power. General paralysis has been described as a toxic disease depending on syphilis, but though in my experience syphilis plays a very important part, yet it has not been proved to be an invariable antecedent.

Etiology.—The causes of general paralysis are divided into *proximate* and *remote*, and these again may be separated into *physical* and *moral*. The disease occurs in England (among the middle and upper classes at any rate) ten times as frequently among men as among women. In the female patients who have come under my observation, syphilis has rarely been excluded. It occurs in middle age, the period from thirty-eight to forty-five containing the largest number of my cases. It is rare in a purely agricultural population: thus, it is hardly ever met with in the Highlands of Scotland, in Ireland, and in the wilder parts of Wales. It is common among those who dwell in or frequent cities; it is most common in married persons; it occurs chiefly in the well-grown and vigorous who "enjoy" life and do not control their desires, though it is rarely, in my opinion, directly due to sexual excess. Neurotic heredity plays a much smaller part in the origin of this disease than it does in the production of other kinds of insanity. In the ancestors of the general paralytic, however, we often meet with such evidences of arterial degeneration as apoplexy, Bright's disease, and gout. General paralysis occurs occasionally in both parent and child, in brothers, and even in twins; but such occurrences are very rare. It seems to be rare among vegetable feeders, such as certain Oriental races, though these may give way to excessive sexual indulgence and have had syphilis. In Abyssinia no case of general paralysis was noted in 12,000 who had syphilis (Paton). I understand that certain nomadic tribes, though saturated with syphilis, not being addicted to alcohol or meat, do not get general paralysis. Syphilis is the chief cause, however, and alcohol is only of secondary importance.

I consider that almost anything which interferes with the normal restoration of the nervous tissues may predispose to general paralysis in certain persons; thus worry, which prevents sleep, will bring about in one person what syphilitic arterial degeneration will do in another, and impaired nutrition, due to toxic blood, brain injury, or febrile illnesses, such as influenza, may predispose to or precipitate the attack of general paralysis. As I have said, I agree with the majority of observers in thinking that among general paralytics there is a smaller direct neurotic inheritance than among the other inhabitants of our asylums. Worry, when added to other unfavourable conditions, is one of the most common causes of general paralysis. I do not recognise sexual excess alone as an important factor in the disease: general paralysis is not common in polygamous races; it does not occur at the age of greatest excess; it is not a sequel of masturbation, yet in masturbators there is more excess than in any other persons; and, finally, general paralysis is not, in my opinion, prevalent in the mentally indolent classes, in which sexual indulgence is most common. Excess is, after all, a relative word, and that which would be moderate in the countryman, with little drain on his nervous powers, would be great excess in a city dweller living by his brain. At least half my patients suffering from general paralysis have had syphilis, and have told me so; of the other half a large proportion were too weak in mind when first seen by me to reply to such inquiries, and I do not think it is justifiable, for the sake of science, to put questions of this kind to the patient's near relations. My own belief is that in at least 80 per cent of the cases syphilis may be established, and I submit herewith a table of the last 500 cases seen in private :—

Syphilis admitted	259
Doubtful	89
No history obtained	142
Excluded	10
	500

There are several classes of general paralysis with syphilitic histories to be considered : first, those in whom syphilis had been recognised, medically treated, and all but forgotten by the patient till some special inquiry recalled the old disease to the memory. Thus, a patient who had had syphilis twenty years before, during a period of pecuniary trouble manifested ptosis and external strabismus which rapidly passed off under specific treatment. From that time, however, some very well-marked change was noticed in the character of the patient, and, later, symptoms of general paralysis became evident. In some other patients years after the syphilis, and without any marked recrudescence of symptoms or particular cause for exhaustion, motor or sensory troubles arise. Thus, one will have attacks of temporary aphasia, another a monoplegia, a third a sensory perversion or defect, a fourth slight attacks of an epileptiform character; and any of these may be the starting-point of general paralysis. In a large group, tabes dorsalis, associated with syphilis if not dependent on it, precedes the ordinary symptoms of general paralysis by many years. I have met with several cases in medical men formerly infected with syphilis who fell into syphilophobia, and the worry of this was the immediate cause of the general paralysis.

Though general paralysis is most common among the races who take alcohol, yet in my experience alcohol alone is not a common cause of the disease. As alcoholic excess and sexual excess are common early symptoms they are often mistaken for causes. I believe that as alcohol produces a temporary reduction of mental power which resembles general paralysis, so the frequent repetition of alcohol in excess may dispose to degeneration along the same lines ; still it is noteworthy that other toxic influences (such as lead), which are more stable, produce more lasting effects on the nervous system ; and other nerve-poisons which stand intermediate in fixity between lead and alcohol seem to have an intermediate influence in the production of general paralysis. Thus, it has not been rare for me to meet with general paralysis started in suitable cases by influenza, although in the same cases alcohol alone had failed to produce the effect.

I believe that injury is a very common starting-point of general paralysis, but only in predisposed persons. I am in the habit of comparing the nervous systems of those who have had constitutional syphilis, and who have taken excess of stimulants or who have led exciting and exhausting lives, to fruit which is mature, and which will keep sound till some accident starts the degenerative changes which rapidly spread, "and so from hour to hour we ripe and ripe, and then from hour to hour we rot and rot." I cannot recall a single case of insanity due to injury pure and simple which turned out to be general paralysis.

Sunstroke likewise plays a very subordinate part in the production of general paralysis. In dwellers in tropical climates who have indulged in excess of alcohol, and who have spent unhealthy lives, sunstroke may act as the "last straw"; but it is much more common for the sun to produce symptoms which are like those of general paralysis, but which can be distinguished from them, and are much more curable.

To sum up: the causes of general paralysis are complex, though simpler in some cases than in others. The causes may be physical or moral: syphilis, lead, alcohol, fevers, injury, and sunstroke are the most common physical causes; whilst worry, anxiety, and strain are the more frequent moral causes.

G. H. S.

Morbid Anatomy.—In considering the morbid appearances of the nervous system in general paralysis we note at the outset that there are none, so far as our present knowledge goes, which can be regarded as peculiar to this disease. In the very great majority of cases, however, which have been regarded clinically as general paralysis, the necropsy reveals a complex of morbid conditions which, whilst perhaps scarcely so suggestive as the group of symptoms which go to make up the clinical picture, is nevertheless sufficiently characteristic to bear out the diagnosis founded upon the latter. As regards the brain, it may be affirmed that certain morbid conditions are found there in a more pronounced form in general paralysis than in any other disease in which this organ is affected.

The naked-eye appearances of the cerebrospinal axis and its enveloping parts, with which we are familiar at the necropsy of a case of general paralysis in which the disease has lasted the ordinary period, are as follows: The skull-cap (sawn through at a standard level) is commonly heavier than normal. In some 25 per cent of cases hyperostosis is noted. The diploe is obliterated or deficient in about three-fourths of the cases; the sutures are more or less obliterated or covered with calcareous deposit, and exostoses occur. The dura mater exhibits changes in about one-half of the cases, is then thickened, congested, and adherent to the skull-cap, especially along the sagittal and coronal sutures and over the frontal bone. The Pacchionian bodies are unusually prominent, causing marked pits in the bone above them. Calcareous deposit occurs in the falx cerebri. The inner aspect of the dura shews rusty discoloration, or localised blood-clot, which may be surrounded by membrane, or cystic formations may be present ("arachnoid cyst"), or membrane of various degrees of consistence, extent, and vascularity may be stripped off, especially from the most prominent part of the convexity of one or both hemispheres or the temporal fossae ("pachymeningitis haemorrhagica," "haematoma of the dura mater").

The serous fluid in the subdural and subarachnoid spaces and in the ventricles is turbid and increased in amount, 4 to 6 ounces escaping. The arachnoid, where it bridges over sulci, and over the space between the crura cerebri and optic tracts, is thickened and milky, or opaque and swollen, watery or gelatinous; and the leptomeninges generally are thickened, oedematous, hyperaemic, and shew milky streaks along the course of the main veins, these conditions being most marked over the convexity of the parietal and frontal lobes. The frontal lobes frequently adhere to each other, the adhesions being easily broken down. The pia, on stripping, is found to adhere to the cerebral cortex along the summits of the gyri, though for short distances only, and at this stage not strongly. Indeed, at the usual date of death there is frequently no adhesion, the leptomeninges stripping, on the contrary, with undue ease. Adhesions are commonly present over the frontal and parietal lobes, at their convexity, and are especially noted over the central gyri and the bases of the frontal gyri. The occipital lobe does not usually present adhesions. After removal of the pia, the cortex at the site of adhesion presents a worm-eaten appearance. Summing up in respect to the meninges, there is evidence of diffuse chronic meningitis in over 80 per cent of the cases.

There is a general atrophy of the brain; the gyri are shrunken, ribbon-like where most affected, the sulci and lateral ventricles are widened, and there is a loss of weight (1250 to 1270 grams would represent the weight commonly found in the male). The organ is flaccid; it collapses when placed upon the table, the hemispheres separating posteriorly, and the posterior extremity of the corpus callosum becoming torn—an indication of softening. Atrophy, though general, is especially noted at certain parts, such as the central and precentral gyri, the basal ganglia, the pons, and medulla. Softening is also general, with foci of special intensity; it occurs irregularly in the grey and white matter, especially in the parietal, central, and temporal gyri, in the island of Reil, in the external and internal capsule, and lenticular nucleus; it is also met with in the basal ganglia. The focal softening is ascribed to blockage of minute arteries. There is often considerable atheroma of the main arterial trunks, and haemorrhagic foci exist, especially in the centrum ovale, the basal ganglia, and the pons and medulla. These are due to various kinds of degeneration of the walls of the arterioles (hyaline, fatty, atheromatous) in conjunction with increase of the arterial blood-pressure. Frequently also there are traces of former haemorrhages, in the form of cysts and scars. The cerebellum participates in the above degenerative changes, but not in a marked degree; here softening—general and local—and hyperaemia are the most marked naked-eye alterations.

The cortex cerebri exhibits foci of softening, general hyperaemia, or patchy discoloration. It is also atrophied, especially in the frontal and parietal regions. In the white matter similar conditions exist, and the perivascular spaces are dilated. The ventricles are dilated, and the ependyma of the fourth ventricle is congested, thickened, softened, sometimes gelatinous, sometimes tough; its surface is granular in varying degree. The same conditions of the ependyma are found in a lesser degree in the other ventricles.

The cranial nerve-roots shew grey degeneration and atrophy. These conditions have been noted in the olfactory bulbs and tracts, the optic tracts, the third, fifth, sixth, and seventh nerves.

In contradistinction to some of the conditions noted above we find exceptional states, chiefly affecting the covering of the brain; of such is atrophy of the skull-cap, which is therefore lighter than normal. The dura mater may appear quite normal—this is less unusual than might at first be supposed—or there may be evidence of pachymeningitis externa, and rusty deposits may be present on the outer aspect of the dura. Granulations occasionally occur on the other aspect of the arachnoid, and adhesions between the dura and arachnoid. Adhesions between the pia and cortex may be very scanty, or unusually situated; they may be absent, even though the disease be not advanced. The ependyma may appear normal, or at most slightly granular; in about 20 per cent of the cases naked-eye evidence of disease in this structure is but slight.

Turning to the *spinal cord* and its investing membranes, there is naked-eye evidence of disease in the membranes in about 39 per cent of the cases. Adhesions exist between the vertebrae and dura mater; and the membranes of the cord are thickened, opaque, and hyperaemic, chiefly posteriorly. There may be blood-extravasation upon the inner aspect of the dura, but deposits, whether haemorrhagic or calcareous, are much rarer upon the membranes of the cord than upon those of the brain. Occasionally there are adhesions between the dura and the pia. The cord is chiefly softened, but sometimes indurated. The different columns, chiefly the lateral and posterior, may shew grey degeneration; there is evidence in some cases of disseminated sclerosis. Lesions of the columns, manifest to the naked eye, are less commonly observed in general paralysis than in the ordinary degenerations of these parts. In some cases the entire cord is diminished in size, or there is diffuse softening. The changes in the cord, as will be seen when the histological conditions are referred to, are very various.

In acute cases, and in those which succumb in the earlier stages of the disorder, the naked-eye appearances, as might be expected, are less indicative of disintegration and degeneration. In place of these are exhibited the signs of acute disease: the blood-vessels are everywhere congested, whether in the diploe, dura mater, leptomeninges, or brain substance; flakes of lymph occur in the cerebrospinal fluid, and at points there may even be pus-formation—purulent meningitis (Kaes). The membranes are tense, and on cutting them the full-volumed vascular brain projects, and its diminished consistence is noted. Adhesions between the pia and cortex are strong and extensive. Such adhesions are present in at least 70 per cent of necropsies of general paralysis. There may be recent deposit of blood in the subdural space.

Histological Changes—The Brain.—Seeing that when the disease has lasted its customary period, end-products only, of no particular importance, are met with in the brain, cases should be selected for the purpose of histological examination, in which death had taken place in an earlier stage; preferably those in which the duration of the disease had not exceeded one year. In such a case we find in the cerebral pia mater that the vessels are prominent, with thickened walls and abundance of nuclei. There is round-celled infiltration of the adventitia and thickening of the various coats of the vessels of the pia. The septa passing from the pia to the cortex are thickened and increased. There is free exudation into the meshes of the pia. The trabeculae between the arachnoid and pia, as seen in a sulcus, are bathed in inflammatory exudate. The various structures of the cerebral cortex give indications of disease. Thus, the blood-vessels are exceedingly numerous, distended with corpuscles, and tortuous. At points along their course they exhibit dilatations, at which points the lumen, on cross section, is found occluded. Newly-formed vessels are observed. The nuclei of the vessel-wall are greatly increased, and about the vessel-walls are plasma-cells in abundance. Mast-cells are also seen and extravasated leucocytes. The coats of the vessels are thickened, and there is proliferation of the endothelial nuclei. Hyaline degeneration or fatty change of the vessel-wall may be present. The subadventitial lymph-spaces and the perivascular spaces shew collections of lymph-corpuscles and blockage by cells (especially plasma-cells), coagulated lymph, and altered blood-pigment. The perivascular sheaths become much distorted by the contraction of the vascular processes of the enlarged glial cells. The conditions described indicate obstruction to the circulation of blood and lymph, by which the integrity of the nerve-structures must be impaired.

The supporting structure (neuroglia) exhibits deep staining in many parts of a section; this is especially marked in fresh sections stained with aniline-blue-black. Instead of a ground-substance scarcely stained, there is found a mottled, patchy blue staining between the nerve-cells, a granular or fibrillar condition. Modern selective methods of staining the neuroglia shew a meshwork of neuroglial fibrils, especially prominent in the outermost layer of the cortex. With these conditions, deeply-stained round bodies, apparently nuclei, are found in abundance; and glial cells ("spider," "lymph-connective") are numerous and prominent, with large deeply-stained bodies and numerous processes. These spider-cells are especially noteworthy in the outer layer of the cortex, where their increase is earliest noted, and also in connexion with the vascular sheaths. A strong "vascular" process passes from the cell to the vessel-wall, ending there in an enlarged extremity, a nucleated mass of protoplasm; and from the body of the cell other processes radiate. But these cells also form prominent objects between and around the nerve-cells. The cell-bodies shew absorbed detritus. The hypertrophy of the vascular glia-cells is associated with degeneration of the neurons and obliteration of the lymph-channels.

As regards the nerve-cells, the normal distribution of the cell-layers is disturbed in places, these layers being ill-defined and the cells wanting. The cell-bodies in the earlier stages shew acute changes, being swollen and staining irregularly or diffusely; the chromophil granules and neuro-fibrils disappear. Later, granular degeneration or fatty hyaline changes occur, or vacuolation. Ultimately the cells become sclerosed, deformed, shrunken, staining deeply; the axis-cylinder becomes visible, the processes sinuous, varicose, and shrunken. The medullated sheath and axis-cylinder processes disappear. The pericellular spaces appear to be widened owing to cell shrinkage. The cell-nucleus is altered in shape; displaced, its membrane shrunken and irregular, its substance deeply stained. The nucleolus disappears. In advanced cases areas of considerable size can be seen without any cell-elements left.

The medullated fibres of the cortex are wasted and degenerated, especially the tangential fibres of the outermost layer, but likewise the super-radial and radial fibres. They ultimately disappear in large measure.

Ljubimow has described the degeneration, atrophy, and—in the later stages—disappearance of the "association fibres" of the cortex; this degeneration proceeding from before backwards over the cortex. The nerve-fibre degeneration is regarded as a primary degeneration, independent of any inflammatory process. Colloid bodies are seen, especially in the outermost layer, which are ascribed to degeneration of the nerve-fibres.

In summing up the above histological changes, it may be said that they consist in atrophy of the nerve-elements and hypertrophy and hyperplasia of the vascular and connective tissues.

Changes similar in character to those existing in the cortex are found in the area immediately beneath the cortex, in the medullary substance, the basal ganglia, corpora quadrigemina, cerebellum, pons, and medulla; but these are less marked, and have not claimed the attention which the condition of the cortex has naturally received (see also Vol. VII. p. 227, and Figs. 46, 48, 49, 52).

Degenerative changes are present in the nuclei and roots of the cranial nerves, as in the hypoglossal and facial, and the vagi (with which may be associated the cardiac and pulmonary disturbances and lesions which occur), with evidence of vascular implication (pronounced hyperaemia, with thickening of the walls of the vessels).

The Spinal Cord and Peripheral Nerves.—Changes of very different kinds occur in the cord, and are found in the majority of cases. Out of 145 cases Fürstner failed to find morbid alteration in 16 only. The tunics shew chronic inflammatory changes; more especially there is evidence of chronic leptomeningitis; the pial septa are involved in this. In the cord itself there may be a diffuse degeneration, or irregularly-occurring patches of degeneration (softening, sclerosis), more commonly the latter, in combination with changes in the columns. Degeneration is usually found in combination in the posterior and lateral columns, and most notably in the former, but sometimes singly, and then most frequently in the posterior columns.

Fürstner finds that one side is regularly more affected than the other. The lateral columns are especially affected in the dorso-lumbar region; in the posterior columns degeneration is often seen along the whole length of the column, with degrees of local intensity. The anterior columns are but rarely affected, and never without disease in the posterior and lateral columns. The degeneration may be more intense in certain streaks, as in the posterior-root zones, or in Goll's tracts (rarely), or in the lateral pyramidal tracts. These degenerative changes consist in hyperaemia; the vessels being engorged, their walls thickened, and the nuclei in the latter increased. These vascular changes are especially prominent in the posterior columns. The connective tissue is increased, with nuclear proliferation and enlargement and proliferation of the spider-cells, especially along the vessels. The medullated nerve-fibres shew loss or swelling of myelin, the latter taking up the stain; or the myelin is granular, the axis-cylinders being interrupted or distorted. The cornua also exhibit changes in varying degree; there is degeneration and atrophy of nerve-cells, the conditions found being the same as in the brain, though less marked. The spinal nerve-roots (posterior and anterior) are also degenerate; their connective tissue is hypertrophied from chronic inflammatory change; the nerve-fibres are atrophied. The spinal ganglia are in like manner degenerate. The lumbar and sacral nerve-roots are believed to shew the most marked changes.

The peripheral nerves shew, in varying degree, parenchymatous degeneration (degeneration of the medullary sheath, swelling and atrophy of the axis-cylinder) and atrophy, and overgrowth of the connective tissue. The degenerative changes are more marked in the nerves of the lower than of the upper extremities.

The ganglia of the sympathetic system have been found to exhibit changes indicative of degeneration of the nerve-cells and chronic inflammation of the connective tissue.

The cerebrospinal fluid, in addition to being frequently increased in amount and turbid, shews increase in albumin and also lymphocytosis; plasma-cells are also found.

Degenerative changes are also found in the muscles, including the heart and diaphragm (Mott, Campbell). There is fatty change with disappearance of the muscle-fibres and increase of connective tissue.

Diseased conditions, mainly of a chronic inflammatory kind, with arteriosclerosis, have been described (Klippel, Mickle, and others) in various viscera, as in the lungs, stomach, intestine, liver, spleen, kidneys.

Pathogeny.—The disease is one attacking the whole nervous system, though the cerebrum, and more particularly the cerebral cortex with the investing pia-arachnoid, is in the vast majority of cases the earliest seat of disease, and the locality in which its effects are most pronounced. The morbid process is usually first manifest and most active in the frontal and parietal areas. In some cases the encephalon alone shews indications of disease. Occasionally, to judge by symptoms, the cord or bulb would appear to be attacked first, and the brain later. As regards structures outside the nervous system, it may well be that these are attacked independently of this system, and not merely secondarily. Thus, the fatty changes which occur in various muscles cannot be summarily ascribed to degeneration in the nerves passing to them; on the contrary, examination of these nerves in some cases has failed to shew changes which would account for degeneration in the muscles (Mott). It is not possible at present to affirm how far the various degenerative states of the non-nervous tissues are secondary, and due to nerve-degeneration (impaired nutrition), or to vasomotor disturbance, dependent on degeneration of the central nerve-tissues, especially the cortex cerebri, or are primary and independent, ascribable—like the muscular degeneration—to the influence of a toxin.

The nature of the pathological process remains obscure. It has long been, and still is, a subject of debate whether the primary change is inflammatory, affecting the vessels and interstitial tissue—a meningo-encephalitis—or a parenchymatous degeneration, the specific tissue, the nerve-elements, being primarily attacked. According to the first view the degeneration and atrophy which the nerve cells and fibres exhibit are due to impairment of nutrition consequent upon diminished blood-supply, following upon obstruction of the blood-vessels by waste-products and upon pressure of increasing connective-tissue. The second view is that the inflammatory changes manifested by the supporting framework of the nerve-elements are merely secondary in nature, "the result of decay of the neurons, and irritation of the products of their degeneration" (Mott). Observations are adduced to shew that changes in the blood-vessels are obvious when the nerve-elements shew few or none, and vice versa. Thus, on the one hand, we find described (Mendel, Bevan Lewis) changes in the vessel-walls, such as accumulation of nuclei in them, cell-accumulation in the lymph-spaces, thickening of the coats of the vessels, without alterations in the nerve-elements; on the other hand, Zacher, investigating the brain in an early stage (fourth to eighth week) of the disease, found a high degree of atrophy of the nerve-fibres of the cortex, with very slight vascular lesion. Others (Collela, Awtowkratow) have described alteration in the processes of the cerebral cells of the nuclei in the bulb, where the blood-vessels have been simply dilated. Parenchymatous degeneration of the peripheral nerves, independent of the vascular lesion, is described by d'Abundo, Collela, and Bianchi. There is further the fact that the frontal lobe—the highest-level centre—is the part earliest affected, there being no apparent reason why, were the change primarily inflammatory, the temporal and occipital lobes should not be equally attacked; yet these parts exhibit lesions to a comparatively slight extent.

The widespread character of the disease, affecting as it does not only the brain, but the entire organism, would point to the action of a toxin. Recently Dr. Ford Robertson has insisted that general paralysis is due to a bacterial toxin acting upon a prepared soil, the normal defences having been weakened by predisposing causes, such as syphilis and alcohol. In a number of cases he has found an organism resembling the Klebs-Loeffler bacillus in the digestive and respiratory passages, and in the brain (in recent cases this has been traced continuously from the nasal mucous membrane to the brain). Rats experimented on with this organism have shewn certain clinical symptoms and post-mortem brain-changes analogous to those seen in general paralysis. These views have not met with support; the general opinion is that the part played by these bacteria in this disease if any, is secondary only.

Edwin Goodall.

REFERENCES

1. *Allg. Ztschr. f. Psychiat.*, Bericht über die psychiatrische Literatur für 1906 und 1907, and preceding years (gives list of the year's literature on the subject in periodicals of different countries, with abstracts and references).—2. Kaes. "Statistische Zusammenstellung der makroskopischen Veränderungen des Centralnervensystems bei allgemeiner Paralyse," *Allg. Ztschr. f. Psychiat.*, 1895, li.—3. Ljubimow. "Über das Verhalten der Associationsfasern der Hirnrinde bei Paralysis progressiva," ref. in *Allg. Ztschr. f. Psychiat.*, 1894, l.—4. Mickle, J. "General Paralysis," and "Critical Digest on General Paralysis," *Brain*, 1894, xvii. 50.—5. Mott's *Archives of Neurology* (from the Pathological Laboratory of the London County Council).—6. Nissl, F. u. Alzheimer, A. *Histologische u. histopathologische Arbeiten über die Grosshirnrinde*, Erster Band (deals exhaustively with the subject and gives very full references).—7. *Rev. Neurol. and Psychiat.*, Edin. (see monthly papers for the past year or two).—8. *Textbooks on Mental Diseases* (Psychiatry) of Bevan Lewis, Berkley, Bianchi, Kraepelin, Ballet, Th. Ziehen, Weygandt, Clouston, Stoddart, Savage and Goodall, M. Craig, Tanzi.—9. Tuczek. *Beiträge zur pathologischen Anatomie u. zur Pathologie der Dementia paralytica*, 1884.

E. G.

Prodromes.—General paralysis may appear without any warning; but certain nervous changes may already have taken place in the higher centres, which, however, were not what I may call indicating areas; thus, the appearance of the disease is sudden, though the morbid change may have been long advancing slowly. After the full recognition of the disease, we commonly learn from the friends of symptoms (now appreciated for the first time) which push back the duration of the malady. Such symptoms are the warnings, or prodromes, of general paralysis. These warnings may affect various parts of the nervous system, causing loss of power or loss of control. I will consider some of the more prominent under the heads of mental, sensory, and motor warnings.

On the *mental side* is noticed some impairment of the most specialised accomplishments of the individual; thus, the musician ceases to please his trained auditors, the artist's work no longer commands a market, the artisan gets out of work, the soldier loses his good-character badge. There is, in fact, a loss of the highest power of adjustment, a kind of social ataxia which interferes with delicate social intercourse.

Loss or defect in power of attention is very common. Defect of will power, lack of decision, shewn in hesitancy and doubt, increased irritability, change in temper, abnormal susceptibility to the influence of stimulants of all kinds are of very frequent occurrence. Hysteria and "nervousness" may also occur long before the danger is suspected.

Among the *sensory warnings* we may meet with defect in smell, sudden loss of sight of a temporary kind, similar loss of hearing, temporary and local anaesthesias, formications, flashes of light, tinnitus aurium, and hallucinations or illusions of one or more of the senses; though in my experience these last sensory warnings are not very common. Giddiness and so-called congestive attacks may occur, or bilious attacks ending in vomiting similar in some ways to migraine or fainting fits. If such symptoms appear in patients who seem in other respects likely to suffer from general paralysis, they may be of value as warnings. On the *motor side* there is often well-marked restlessness, or, occasionally, stupor or undue torpor with constant sleepiness; slight and temporary aphasia, or loss of power of expression by speech, or by writing, or other defect of the kind, may occur, with some alteration in gait (ataxic or spastic), inequality or fixity of the pupils, or change in muscular electrical reactions. There may be convulsive seizures or partial palsies, such as ptosis, external strabismus, or loss of power in one limb. Besides these local or special troubles, there is often also a general indescribable change; the person is "not himself," he is tending away from himself. There may be loss of recent memory, inability to transact business, confusion of thought, loss of animal spirits, or, on the other hand, an undue buoyancy with sleepless restlessness. I believe the character of the coming general paralysis is generally foreshadowed by the nature of these earlier warnings, though this is not always the case.

Lastly, I would call special attention to a group of warnings which might be considered with the symptoms of social ataxia, but as they have especially important relations I prefer to place them apart. I refer to the *moral perversions* which in not a few cases precede the recognition of general paralysis. Crimes against morality and propriety are the most common, such as criminal assaults, indecent exposure, and bigamous marriages; or, again, stealing, getting money under false pretences, and reckless conduct of business, with apparent fraudulent intent, may be the kind of backsliding.

The recognised invasion may be quite sudden, and it may appear first on the motor, the sensory, or the intellectual side. The first symptoms of the disease may be but the sudden increase of any one of the warnings. A fit of one kind or another may be followed by all the mental and physical signs of general paralysis. An attack of mania, whether this be of the simple emotional or of the delirious form, may pass into general paralysis. Melancholia, or mere silliness, may be the earliest feature of the disease. In rarer cases "folie circulaire" or alternating insanity, systematised hallucinational or delusional insanity seem to initiate the disease; the fact being that the degeneration varies in the part of the brain which is chiefly and earliest affected, and consequently the earliest signs of the disease vary. To sum up: the disease may be recognised suddenly, and any form of mental, motor, or sensory disorder may appear as its first symptom. In some cases the patients have been odd from youth and the symptoms of general paralysis are accentuations of the earlier oddness. It is therefore very common for the disease to exist for long periods before its true nature is recognised, and what later is a matter of easy diagnosis was not recognisable in the earlier stages.

Forms of General Paralysis.—Though various authors, including Dr. Mickle, have described certain special varieties of disease in which specific changes in the nervous centres are connected with definite mental disorders, I have not been able to satisfy myself that these divisions can be definitely made. It is convenient, however, to recognise that the symptoms generally combine in one or other of the following clinical groups: (i.) Acute or galloping general paralysis; (ii.) ordinary general paralysis, with mania and exaltation of ideas; (iii.) melancholic-hypochondriacal or stuporose general paralysis; (iv.) progressive dementia with general paralysis; (v.) general paralysis of the double form; (vi.) spinal general paralysis, that is, beginning with ataxic or spastic symptoms; (vii.) general paralysis in woman; (viii.) developmental or adolescent general paralysis; (ix.) senile general paralysis.

(i.) *Acute or Galloping General Paralysis.*—The chief characteristic of this disease is the rapidity with which the whole of the symptoms, both physical and mental, may run through their course, and end fatally. The causes of death may have been long at work: signs of mental decay, indeed, may have been passed over as unimportant till the catastrophe revealed them in their true light. The tinder was dry for the spark, and destruction rapidly followed. In some cases death has taken place in six weeks, in others, in from three to six months. In one group of such cases acute delirium or acute delirious mania ushers in the disease, and probably some of the fatal cases of delirious mania are really cases of acute general paralysis. A febrile illness, a sudden moral or physical blow, or a fit may start acute general paralysis. At first there is acute delirious mania with an unusual degree of restless activity, there is also some rise of temperature, there is very well marked tremor of voluntary muscles, especially those about the mouth. Speech is greatly masked, often reduced to muttering; the face is flushed, the expression anxious, and excitement is rapidly followed by mental weakness, loss of vesical and rectal control, bed-sores, rapid exhaustion, and death. Fits are not, in my experience, common in this form of the disease, though they may initiate it.

It is interesting to note here that a certain number of patients who have acute delirious mania recover; certain others are left permanently weakened in mind, and a few pass from acute delirious mania into general paralysis. The general paralysis thus started by delirious mania is not always of this acute and rapid kind, but may belong to either of the other forms.

(ii.) *The Ordinary Maniacal General Paralysis.*—It is almost always preceded by a slight period of depression, which often assumes a hypochondriacal aspect. It resembles ordinary mania, and is nearly always associated with expansive benevolence and with a general exaltation of ideas. The exaltation is all but universal, so that the patient believes himself to be wealthy, beautiful, a poet, an author, an athlete, and an immortal. Restlessness is also well marked in these cases, but next to expansiveness probably "facility" (as it is called by Dr. Clouston) is most characteristic. The patient, though believing himself to be so mighty, yet, like a child, is very readily diverted from one subject or purpose to another. The mania assumes the emotional or hysterical aspect, and the more marked this is, the greater the chance of remission. The second and third stages are alike in all the various forms.

Typical Case of General Paralysis.—The following is a specimen of an ordinary case of general paralysis. A married man of about forty-five, of active habits and with plenty of ability, not belonging to a neurotic family, who in early youth had been a free liver, fifteen years before had contracted syphilis, of which he thought very little, as he had few or no constitutional symptoms. He married when the supposed danger of infection had passed, and for some time he indulged very freely in sexual connexion ; but he steadied down and was a thoroughly good and respected man of business. Certain severe money losses, due to no fault of his own, worried him, broke down his sleep and appetite, and he sought relief in alcohol. He complained of headache and loss of power, and was regarded as neurasthenic. At this time rest and travel were advised. He was very emotional, even hysterical ; at other times he was apprehensive and hypochondriacal. Change and rest, however, seemed to set him up, and in two or three months he returned to work fairly well. He had an epileptic seizure, but from this attack he recovered in a few days, but some defect remained in his speech ; he hesitated, and did not articulate clearly. He now took more prolonged rest, and when he came back to town all his friends were struck with his buoyant, exuberant health ; and he said he felt better than he had felt for years. This feeling of buoyancy led to amorousness, and not content with home he got into bad company and scandal arose. He rapidly became intolerant of interference, irritable, and passionate. This state of unrest ended in a sudden outburst of mania, with violence and extravagance, during which endless telegrams and letters were sent to distinguished persons to whom he was unknown. Sent to an asylum, he fell at once into all the ways of the place, making himself the agreeable friend of one and all, and never seeming to tire in his work and play. By this time, however, the change in his expression and in his aspect was well marked : his gait was altered, his pupils unequal and sluggish, his writing changed, and speech hesitating and clipped. Every available piece of paper was seized for voluminous scribbling. Still little or no change was seen in his temperature or in his general circulation, though after exercise his breathing seemed to get shorter. He had lost all his finer acquirements, and though he would sing or play on the piano, his performances were feeble and faulty. Memory, though failing, was still fairly good ; he had no power of appreciating his own condition, and still thought himself in perfect health. Sleep was profound and appetite good. This period of excitement lasted for about four months, to be replaced by a phase of general dissatisfaction with his position and surroundings. He complained incessantly that he ought never to have been brought to an asylum, and he appealed so constantly for release that at last this was granted at the wish of his wife. At home he was exacting, irritable, and unstable. A change into the quiet country was tried with marked gain, but a return to London was followed by another but much slighter fit which left the mind much weaker, and the patient now exhibited the characteristic "facility." He would read childish novels and cry over them, go out for the same walk daily, and be perfectly satisfied with himself and his surroundings and excursions. Sleep being very good and appetite greedy, he got very fat, his face lost expression, and his skin became sallow and greasy. This calm continued for six months, when a severe fit of an apoplectiform character left him for some days hemiplegic and aphasic. These symptoms slowly passed off, but the man now began rapidly to lose mental and bodily strength ; he laughed to himself, was neglectful of cleanliness, occasionally lost control of rectum or bladder, and ate ravenously without masticating ; at times he was irritable, but too weak to give much trouble. He got thinner, and bed-sores developed over the sacrum. While trying to get out of his chair he fell, and a rib was fractured, but no complaint of pain was made. Soon after, other fits occurred, and for some days he was in a "status epilepticus." One arm was now noticed to be contracting, and he was too helpless to sit up.

The second and third stages had occupied a year ; the bed-ridden paralytic state continued for six or seven months, during which his limbs contracted. He seemed quite unconscious of his surroundings ; still the organic reflexes persisted, so that anything placed on his lips caused a swallowing movement. Thus paralysed, emaciating, and incontinent of urine and stools, recognising no one, he remained till the temperature rose, with pneumonia at the base of one lung; this rapidly spread, and with a temporary gleam of reason before his death he died quietly at the end of about three years from the time the disease was first fully recognised.

(iii.) *The melancholic form of general paralysis* is not always easy of detection. It is generally thought that there are now many more cases of general paralysis with symptoms of depression than formerly. In many cases, as already said, there is a period of depression which may be of very short duration. General paralysis may be associated with any of the various forms of melancholia. Thus, active resistive melancholia, and melancholia with stupor, may be met with. I have seen melancholic patients with extreme enlargement of ideas—as, for example, to imagine such an occlusion of the bowels that if they burst—as they must—the world would be flooded by the eruption. One such patient believed himself to be a mass of syphilis, and that he would give the disease to hundreds of men in the city. In my experience hypochondriacal notions of bowel obstruction are common in the melancholic forms of general paralysis.

In many cases of melancholia there is great difficulty in getting the patient to speak or to put out his tongue, and consequently it is often hard in the earlier stages to form a diagnosis. Not infrequently I have been misled into thinking the case to be one of simple melancholia till I found the patient, though making constant complaints, was getting fat ; then I have found that his pupils were unequal, his speech was defective, and his reflexes abnormal ; or perhaps the onset of fits has cleared up the diagnosis. Not only marked melancholic symptoms, but even "persecution mania" may be an early symptom of general paralysis. I have known one man who with all the physical signs of general paralysis only exhibited simple weakness of mind and the delusion that he had scabies, which he was always anxious to shew to all visitors. Another man was very dangerously suicidal because he thought he was infecting the city, and that therefore crowds were waiting to lynch him outside his house. Though suicide is not common in the ordinary general paralytics it may occur in the melancholic and persecuted patients.

It is of great importance in this group to recognise the want of proportion between the bodily and mental symptoms; thus, if a man complaining of being miserable, of being obstructed in his bowels, or of being persecuted by his enemies, yet eats well, sleeps well, and gains flesh, he is either a chronic melancholic or possibly a general paralytic passing into dementia. Remissions are less common in the melancholic than in the maniacal form of general paralysis; fits of various kinds may occur, and there is generally a tendency to fatness and dementia.

(iv.) *Demented Form.*—Whereas most general paralytics pass ultimately into dementia, some of them pass into weakness of mind from the outset, being from the first dull, heavy, indolent, and sleepy. Although they get weaker as the disease advances, yet they rapidly fatten and are very liable to fits. In some the first stage is one rather of mental confusion than of mental defect, but dementia comes on later. Some of these demented cases begin with fits. It has been pointed out elsewhere that the progressive stages of general paralysis are summarily represented in the stages of drunkenness; for alcohol will make one man emotional, amorous, or pugnacious, another sentimental and lacrymose, and a third merely stupid.

It is common to meet with cases of general paralysis of the simple progressive demented type in general hospitals, and many such are treated at home as cases of "softening of the brain." It must not be forgotten, however, that the most placid and docile of patients may suddenly become violent and maniacal.

(v.) *Circular Form of General Paralysis.*—This is to my mind the most difficult of all forms to detect and to define. It is among those suffering from this form of general paralysis that so many "cures" are recorded; and it was not till I had been misled several times by such cases that the nature of the disease dawned upon me. This variety may be described in a very few words, but the description will not prevent mistakes being made over and over again. A patient (all mine have been men), of the age and with the ordinary history of general paralysis, becomes very extravagant and exalted; he will boast, rush about the town, give presents, make love, drink, and get into the hands of the police; in jail or in an asylum a diagnosis of general paralysis is made, but he slowly improves, expresses gratitude and contrition, and is sent home "well," the doctor believing he has made a mistake in diagnosis. A year later the patient is seen again, but now is in a state of profound melancholia with refusal of food, and the physician feels sure that his original diagnosis must have been wrong. Slowly this melancholia passes off again, to be followed, after an interval of varying duration, by acute mania, resembling the previous attack even in its minutest details: this gives way again to calm, and perhaps to a second "recovery." After the lapse of several years the patient, however, presents the physical and mental signs of general paralysis, of which disease he dies. I do not know any way of avoiding mistakes in these cases beyond careful observation of the physical signs.

(vi.) *Spinal General Paralysis.*—General paralysis may follow symptoms of tabes dorsalis which may have lasted for some years without any apparent mental defect; or it may be preceded by marked signs of spastic paraplegia. It is possible that general paralysis may follow disseminated sclerosis; I have seen such cases, but I am in doubt about the affinity of the two diseases. It is thought by some authors that peripheral neuritis —whether due to injury, lead, or alcohol—may give rise to general paralysis; and accordingly a form of general paralysis of an ascending propagated course is described.

(vii.) *General Paralysis in Women.*—It used to be doubted whether general paralysis ever occurred in women, and it is certain that women of the educated classes rarely suffer from it, but there is no longer any doubt that gentlewomen suffer, though to a much less degree than men. There are no special symptoms to separate the general paralysis of men from that of women. It usually occurs rather earlier in life, it has a tendency to last longer, fits are not so common, and dementia sets in earlier, the patient rapidly passing into a quiet childish state. I think the reflexes, as a rule, are much more exaggerated in women, and it is rare to meet with ataxic symptoms; this is the more interesting when we remember the rarity of tabes in women. Women may menstruate up to a very advanced stage of general paralysis; even in the second stage they may become pregnant, and be delivered normally. Children born of general paralytic parents (of either sex) will probably shew signs of degeneracy; they may be idiotic, imbecile, or vicious.

(viii.) *Developmental or Adolescent General Paralysis.*—I have seen several such cases, the majority having been taken at first to be cases of disseminated sclerosis, and it was not till later that suspicions of general paralysis arose. This form may occur in children from twelve years upwards; I see no reason why infants should not also suffer. The progress of the disease, in my experience, is not rapid; but dementia appears early, and there is rather gradual decay than a disorder of the intellect. Paralysis soon occurs, fits are not uncommon, and the patients slowly die exhausted.

I have never yet met with such a case in which there was not evidence of parental syphilis—a very interesting point in connexion with the causation of the disease in the adult.

(ix.) *Senile General Paralysis.*—This variety is not recognised by many writers, and I am not inclined to regard it as a very distinct form of the disease. I have already pointed out that progressive decay of bodily and mental functions is the essence of general paralysis, and the same may be said of senile dementia; but the latter is rather a normal extinction of the higher functions, whilst the former is a premature and unnatural decay. It is recognised that some senile persons, mostly men, exhibit a wild, excited, and extravagant train of symptoms, during which they waste their money, go in for sexual debauches, drink, and behave like general paralytics; yet in most of these there is not the universal benevolence which is common in general paralysis, nor is there the marked defect of articulation; the physical weakness is rather paralytic from the first than mere enfeeblement. The memory, too, is more affected in dementia than in general paralysis, and if fits occur they are more probably apoplectic than epileptic. The presence of Korsakow's syndrome is common in senility, not in general paralysis. There are cases, however, in which all the symptoms of general paralysis occur in patients over sixty, and such cases must be called cases of senile general paralysis; and I know no limit to the age when true general paralysis may occur, for as age is a relative term, so the diseases of old age must be relative also.

AGES OF ONSET OF GENERAL PARALYSIS

	20-30.	30-40.	40-50.	50-60.	Over 60.	Total.
Bethlem	2	49	38	10	1	100
Virginia Water . .	5	37	46	8	4	100
Ticehurst	4	9	7	5	...	25
Private	42	119	27	12	...	200
	53	214	118	35	5	425

Duration of the disease and of its stages. This may vary from a few weeks in the acute form, to many years in some of the chronic forms, or in those in which prolonged remissions have occurred. I have found the average duration in private cases to be about three years from the time the disease was recognised, but taking the history of the warnings and the prodromal stages of the disease, I should estimate its duration in private patients of the upper class at about five years from the very beginning to the end. I have known of one or two undoubted cases which lasted from twelve to twenty years. I am inclined to think some well-marked cases never advance beyond the prodromal stage, that indeed there may be an early arrest of the morbid process; and I believe that in some cases arrest may occur during either of the other stages: at any rate complete remissions may occur during the prodromal stage, the stage of excitement, or the stage of paresis. I know of no guide to the probable duration of the various stages, and I do not think there is any definite proportion between the length of the several stages. Cases which begin with very acute mania, excitement, and exaltation more frequently present remissions; cases beginning with steady loss of mental power rarely, if ever, have remissions; but the disease may be arrested. In a remission not only is there no progress of the disease, but also a gain of power; in arrest the progress is stopped, but no gain is made.

Symptoms.—First, I shall take those which may be called the more characteristic, and refer later to those which are the less common. The only absolutely characteristic symptoms are progressive paralysis and progressive dementia, states which scarcely need any description. On the mental side we find loss of the highest self-control, which is shewn in different ways in different persons; there is a loss or defect in the highest and most special acquirements—loss of power of self-judgment, and of judgment in affairs, often engendering extravagant notions of personal worth and power. Emotional instability is also a result of defect of control. Will-power is in defect, so that the patient is facile and easily moved; there is some defect, too, in the registration of recent impressions (loss of recent memory).

Loss of the highest mental control permits many impulsive acts and emotional outbreaks. An external impression may lead directly to an impulsive act or to a sudden emotional outburst—resulting in uncontrolled violent reflex acts; most of the earlier symptoms depend on defect of control, which shews itself also in a loss of delicacy in performing the most refined and specialised acts. Tact and accomplishments are lost; the artist is no longer exact in drawing or sensitive in colour; the musician loses sense of time and harmony; the arithmetician is no longer nimble in computation; the clerk is slow and awkward in his writing; the actor loses the versatility and mobility of his features; the artisan is discharged for careless work; the domestic servant gets into disgrace for frequent breakages. The finer defects may run together in several lines: thus, the clerk may write badly, speak thickly, and walk awkwardly; and the artist may be deficient not only in his own special art, but in related acquirements also.

The defects in the highest control may be progressive, or they may be well marked up to a certain point and then seem to be arrested. There may be remissions also in the individual symptoms. The loss of self-knowledge is a very important symptom; for, although it is common for persons of unsound mind to fail to realise that they are unlike themselves and unlike others, this lack of self-knowledge is most marked among the general paralytics. The general paralytic seems incapable of knowing himself, and of recognising or comparing his past and present states. No amount of experience enables even a medical man to recognise the disease in himself, though he may indeed have been an asylum medical officer. Such a one may be hypochondriacal at the onset of the disease, and in dread of it; but, when he has once passed into the buoyant stage, he is quite incapable of realising his state, indeed may take a visitor round the wards and point out other patients who have similar symptoms to his own, without being able to recognise them in himself. This want of self-knowledge leads also to the grandiose ideas, the egotism, and to the change of notions as to personality which partly depend on them. Patients may lose the feeling of self, and this may go farther, so that they have confused ideas of their own identity, and may even lose themselves altogether. I have met with patients who sought for themselves, and even took off their clothes to see if they were really there.

Megalomania.—Exaltation of ideas may depend on loss of thought-control, each idea, as it passes through the mind, being seized as real and appropriate. The mind is thus coloured by every passing wave of thought, and naturally assumes the happy guises of the great.

Exaltation, though common in general paralysis, is not invariable, nor is it confined to this disease. It may be present in ordinary and in alcoholic mania, in delusional insanity, and in paranoia ; the exaltation of general paralysis differs, however, from the other forms in its universality and benevolence. The general paralytic at one moment is an actor, at another a bishop ; he sees no inconsistency in being at once a god and a jockey, a poet and a millionaire ; as a rule he is benevolent in his exaltation, being willing to grant power and wealth to those about him. The patient with monomania of grandeur as a part of delusional insanity is usually content with his one great idea, and is not in the least disposed to give up his power or to share it with others.

Exaltation is not only associated with the maniacal or expansive form of general paralysis, but may occur also in the other forms of mental disorder which may form part of it. I have met with melancholic general paralytics who have thought that millions of devils were torturing them ; and I may refer again to more than one patient who supposed his bowels to be obstructed by millions of tons of faeces. In other cases delusions with ideas of persecution have been associated with notions that all the world was in league against them.

It is noteworthy that in general paralysis there may be a dual consciousness, so that while the patient talks of his millions of gold and diamonds he may yet remember the state of his balance at the bank, and act consistently therewith.

Micromania is a name used in contrast to megalomania and to indicate what Dr. Mickle calls "belittlement." This occurs more frequently in general paralysis than in any other form of mental disorder ; but it is rare even in this. The patients say they feel so small they can go under doors, or they can get through cracks and keyholes. This feeling of belittlement may be associated with some cutaneous anaesthesia ; as the opposite idea of greatness or swelling of body may depend on hyper-aesthesia. Micromania is met with in certain other cases, but chiefly in senile degeneration, or mental degeneration of one kind or another. I have met with senile patients who spoke of themselves, and of all about them, as "little things."

Emotional instability is very rarely absent in general paralysis, and is often one of the earliest symptoms ; there is a tendency to exaggerate both the joys and the sorrows of life ; usually it is marked by increased irritability, so that the formerly docile, good-tempered man becomes hard to live with. In rare instances the change is of the opposite kind, the bad-tempered man becoming more easy to manage. Tearfulness and passionate outbursts are common in general paralysis, and it is well to remember that hysteria occurring in a middle-aged man is very frequently an early symptom of general paralysis.

The general paralytic is pleased with a feather and tickled with a straw; he will sob over the penny novel and weep over the most vulgar drama. The ease with which men are affected by emotional causes is a gauge to their stability, and is a good indication of the decay produced by age or disease.

The defect of will-power, already described as one of the well-marked features of the disease, and the *facility* (of Clouston) are seen not only in emotional weakness, but in the want of will which makes it often easy to guide these patients for their welfare; determined and obstinate perhaps if argued with, they may be diverted by changing the direction of their thoughts. Want of will may shew itself in some cases as true *folie de doute*.

Memory is affected in very various ways: one of the earliest complaints made by patients themselves is that their memory is not what it was; but, as this statement is made by most men over forty-five, it does not attract much attention. Often the memory seems to be more seriously affected at first than later, probably because fewer calls are made upon it; the loss of memory is slow and progressive, but in some cases the defects suddenly appear after fits. It begins with failure as to more recent and more isolated facts; then it leads to a strange jumble of ideas and a confusion of the past with the present, causing general incoherence. The memory may partly recover itself during remissions. Generally speaking, the loss of memory is not nearly so well marked as in alcoholic paralysis and in senility.

On the *sensory side* there is usually some loss of acuity in one or more of the senses, but it is not common in general paralysis to come across cases of progressive failure ending in complete blindness or deafness; though I have often met with cases of more or less complete blindness, or deafness of one ear, which has proved transitory. There is no special change in the discs associated with the disease; in some cases there is an excess of vascularity in the early stages, in others atrophic changes; and in some the vessels seem to be placed in channels which have rather dark lines along their edges. Sometimes, towards the close of the disease, there is marked optic degeneration. It is probable that in the optic nerves, and in other peripheral nerves, progressive degenerative changes may take place, if the patient live sufficiently long.

The pupils are generally unequal; the inequality varying, even from day to day. The pupils are frequently irregular in outline, but the most important point, in my opinion, is to ascertain whether they react to light and accommodation. There may be paradoxical dilatation. We often meet with ataxic symptoms in which some reaction to light or accommodation is retained, though this is much less than normal; and there is little proportion between the spinal and the pupillary symptoms. It is common, however, to meet with small pupils in cases which began with locomotor ataxia; and it is also common to see dilated pupils in acute cases with spastic symptoms and in the later stages of other cases. There is no direct relation between the mental symptoms and the dilatation of the pupils; nor, in my experience, is there any direct connexion between the dilatation of the pupil on the left side and the exaltation of ideas as some have suggested. The pupils, then, are generally abnormal, they vary from time to time, they generally react defectively to both forms of stimulation, and they are often irregular and eccentric in form.

It has been taught in France that in general paralysis there is loss of power to detect the smell of pepper. I have failed to find this symptom, and I do not attribute any value to it. Many cases of hyperaesthesia have been described, and I have met with a few. In some there is only a local sensitiveness, which in most cases passes off; in other cases patches of anaesthesia occur, but as yet we cannot attach any special value to these symptoms.

Headache or head tenderness, neuralgia, and other nerve-pains have been described by Sir J. Batty Tuke and others; but it is not a common experience for me to meet with these.

Hallucinations and illusions of the various senses may be present at any time of the earlier stages of the disease. In my experience they are not so common as in other forms of mental disorder; but it is a mistake to think that they are rare. Dr. Mickle says that more than half his patients had hallucinations during one period or another. Those of sight and hearing were the most frequent, and were about equally common. Many general paralytics say they see and hear things as the outcome of their boastful delusions; they think they are God's messengers, and must therefore hear from Him. There is nothing special in the character of the hallucinations, which may be single or multiple, pleasant or painful, constant or occasional. In some few of my own cases the hallucinations followed the same course as in systematised delusional insanity, and were associated with ideas of persecution, of spies, or detectives, of whisperings, and annoyance.

Common sensibility may be reduced or almost annihilated; I have known patients rub holes in their bodies or their limbs; and one man slept placidly while his hand was being roasted. It would seem that there may also be deficient reflex sensibility, so that there is an especial danger of choking by the food collecting in the throat, which is not duly stimulated to swallow.

The *disorders of muscular or motor control* are many and characteristic; they are of two classes: the passive, as seen in defective reaction and defective expression; and the active, as seen in disorders of articulation, writing, and gesture. The face wears a mask, the cheeks are flabby without lines or folds; in some cases the patients look as if they had blown out their cheeks, in others the frontal muscles have to be fixed before the rest of the face can be moved. Besides loss of expression there is loss of control, as seen in the very tremulous overaction or ill-directed action. In many cases in which the knee-jerks are exaggerated there is also great excitability about the facial muscles; the tremor is usually most pronounced about the muscles of the lips, the tongue also being very greatly affected; for a time indeed it is ataxic, so that its movements are irregular and awkward. There may or may not be fibrillar tremor, but I do not think this is very common. The tongue from being ataxic becomes more paralysed and rests on the teeth, taking an impression from them along its sides. Certain words are not readily pronounced. I believe it will be found that some general paralytics fail in the labials, others in sibilants or dentals. Tremor of the tongue and difficulty in speech are greatest after mental or moral excitement, and after eating. There is often loss of control over the cheeks and lips, so that saliva flows over the chin, or food collects about the gums.

Various forms of disorder are associated with the function of mastication; thus, there is often a tendency to smack the lips, or to movements like sucking, grinding of teeth, or swallowing. The grinding of the teeth may go so far as to fracture them.

The *handwriting* in general paralysis is almost pathognomonic. In the earliest stage the patient often writes voluminously in a rather shaky hand, and it is noteworthy that the words are often left incomplete; syllables are omitted and the letters are not attached one to another. In some cases, however, the handwriting improves in legibility, for the patient, finding his inability to write quickly, begins to write large text like a child; yet even then the letters are often separate and the words shortened. Although the writing has much the character of that of an old man, in this latter case the shakiness is not associated with the separation and dropping of the letters. In alcoholism, however, the shakiness and the elision or dropping of letters may sometimes occur together.

Muscular defect in gesture is seen in the orator and the actor, and may give way to ludicrous exaggeration of the natural expression. The gait varies greatly, there being many grades between the extreme ataxic and extreme spastic walk. In some cases the symptoms may vary, so that a patient who at one time seems hopelessly ataxic, at another may lose this defect. In many cases the symptoms differ on the two sides. There is very often great restlessness, which on the muscular side is seen in incessant movement or perpetual writing of letters.

The knee-jerks may be absent, normal, or exaggerated; or they may differ on the two sides: they may also vary from time to time.

Fits and Palsies.—During the progress of general paralysis fits occur commonly, but by no means universally. These may be the earliest symptom, though more frequently they occur in the later stages of the disease. The attacks have been called, according to their severity, congestive, epileptiform, or apoplectiform seizures.

I have already pointed out that the occurrence of hysterical fits in middle-aged men is of serious import; I need only repeat that it is not uncommon to meet with cases in which hysteria was the first symptom of general paralysis. A hard-worked man who has had a great deal of worry and anxiety, when consulting the doctor, bursts into tears, sobs, then laughs, and for a moment cannot speak. Such emotional instability may precede the other symptoms by a year or more.

A slight attack of giddiness, some temporary loss of consciousness similar to that of *petit mal*, sudden transient loss of speech, blindness, or deafness may mark the onset of the disease; such symptoms pass off and recur. They may be present, under stress of mind or body, at irregular intervals several years before the other, and more easily recognised, symptoms of general paralysis are noticed.

Congestive or other seizures may start the active mental disorder: thus, directly after a fit a man may shew great mental excitement; but in the great majority of cases the fits first appear after the patient has passed into the second stage; so that some have spoken of this as the "fat, fatuous, and fitty stage." It is a common experience that patients grow fat before the onset of the fits. There may be slight warnings, resembling attacks of *petit mal*, before severe apoplectiform fits occur; but this is by no means constant, as the first fit may be very severe, or even fatal. It is common to meet with some gastric disorder immediately before the fit, and increase of temperature is not an unusual warning of this danger. The fits are very irregular in their recurrence; and though recurring fits generally resemble one another, this is not constantly so. I have known a fit cause right hemiplegia and aphasia, followed later by one affecting the left side and not affecting speech. The seizures may continue for days together, the patient remaining in the *status epilepticus*. Fits are most common towards the end of the disease, and are often the immediate cause of death. As a rule, marked mental degradation follows the onset of the fits, but in a few cases I have met with temporary improvement after epileptiform seizures.

The fits may affect the sensory, motor, or vasomotor centres equally or partially. Temporary loss of power in one limb, or temporary strabismus, ptosis, or aphasia may occur.

It is not uncommon to meet with so-called "bilious attacks," which may have a vasomotor origin.

The fits vary greatly in form and in degree. I have already spoken of the slight passing attacks which occur most frequently in the earlier stages.

Next in order are the epileptiform fits, which cannot by inspection be distinguished from true epilepsy. They are most commonly associated with complete loss of consciousness, though I have seen a patient convulsed while at the same time he was conscious, and was swearing at his disorderly limbs.

As in epilepsy, these fits may start in a definite spot and follow a definite line of development; but this is not often the case.

The epileptiform fits may pass off rapidly, leaving the patient tired, but ignorant of what has happened; as a rule, however, the epileptiform fits of general paralysis leave more physical and mental weakness behind them than a similar nervous discharge in epilepsy would do.

Whilst the result of an epileptiform seizure is more marked in general paralytics than that of a similar fit in an ordinary epileptic, it will be seen, on the other hand, that the apoplectiform seizures in them are followed by less marked and less permanent disorder than is a true apoplectic fit.

I need not describe the character of the epileptiform fits themselves, as they start from the same centres as do those of true epilepsy, and generally run a similar course. In the apoplectiform fits we have usually one-sided convulsions, the fits often starting with a distinct deviation of the head and eyes to one side, and with rigidity and convulsive movements. These tonic and clonic convulsions may recur for hours or days together, as a result of external stimulation or spontaneously. During or after the convulsive seizure there is most profuse sweating; and there may be involuntary actions of bowels and bladder. The convulsions may be so severe as to fracture the limbs or the teeth. There is frequently pallor at the onset, but this is replaced by congestion of the face and neck, which may pass into lividity. The pupils may be equal, but are commonly dilated; there is complete insensitiveness of the conjunctiva. The fits of this description may leave a patient comatose, or he may remain for hours in a very unstable state; so that the slightest external stimulus will lead to a fresh outburst of convulsions. As a rule, the hemiplegia which follows such apoplectiform seizures resembles in all details that of true apoplexy; thus, with right-sided hemiplegia aphasia will probably occur. This hemiplegia, however, is of comparatively short duration, passing off much more quickly, as a rule, than that following a coarser brain-lesion. I have seen an apoplectiform seizure, leaving temporary paralysis, followed by a seizure of the opposite side, associated with recovery of power in the side first affected. After a severe apoplectiform fit there may be coma, followed by maniacal excitement, which, passing off, leaves the patient hemiplegic. In some patients such fits recur for many months or even for a few years; but, as a rule, frequent fits mean speedy exhaustion and death.

In all cases of fits the treatment must be purely expectant: bleeding or severe purgation is not to be thought of; chloral hydrate, in 20-grain doses, administered by enema, has seemed in some cases to shorten the fit and reduce the excitability. During the attack the patient should have his clothes loosened, and he should be kept as free from all sensory stimulation as possible. Unless very weak, it is well not to trouble about feeding by the mouth for a while, as the attempt will probably give rise to new convulsions. I do not think any good is to be derived from sinapisms or the like. In some of my cases amyl nitrite was given at the onset of the fits, but I cannot report any favourable result from it. Under the head of apoplectic or congestive seizures some observers class the paralytic seizures that may come on during general paralysis, in which a limb or part of the body may suddenly lose power for a time without any convulsions. These cases resemble apoplexies due to brain-softening, and may have similar pathological causes.

The paralysis generally increases, and may become associated with contraction of the limbs, even to an extreme degree. The contraction may have a hemiplegic or a paraplegic distribution. With great amount of disablement muscular strength may be maintained. I have met with men who could register a very high power on the dynamometer, yet who could not direct a pen.

In some of the cases of general paralysis with ataxic symptoms I have met with various changes in the joints and the skin. In one of my cases the joints were affected by Charcot's disease, and symmetrical bullae formed about the feet; there was also a marked and persistent *cutis anserina* and an abnormal growth of hair on the legs. I know of no special odour, such as has been described, associated with the disease. Herpetic eruptions may occur, more particularly in the earlier stages of the disease (*vide* Vol. VII. p. 475). Carbuncles may play an important part in general paralysis; they may usher in the disease or they may bring it to an end; yet I have seen very prolonged remissions follow serious carbuncles. There is nothing characteristic about the temperature of general paralysis; many observations have been taken, and these shew that in some cases the disease is associated with a slight persistent rise in temperature, this rise being higher, as a rule, at night. During the middle and part of the later stages the temperature is often normal or subnormal; and I have known a chronic patient have a subnormal temperature for many months. Towards the end, when there are secondary complications, the temperature varies greatly and may be very important as a warning of complications such as pneumonia. Before the onset of convulsions, and during and after these, a rapid and marked rise may occur. The rise is associated with or followed by profuse sweating.

General Nutrition.—Usually in the early stages of the disease there is, with the restless excitement, some loss of weight; later, there is a tendency to fatness, which, however, passes off in the last stage. There is a great tendency to the development of haematomas. Brittleness of bones and defective power of resistance are common, but at the same time the power of repair of injuries is often maintained at a high standard. I have no doubt that in advanced cases marked arterial changes occur; but I cannot find any evidence of early or widespread arterio-capillary fibrosis: nor do I find grounds for believing that changes similar to those met with in Bright's disease are common. Yet I have often found evidence of syphilitic changes in the arteries of the brain. There are not infrequently local haemorrhages—haematomas and pachymeningitis being among the results.

Capillary congestions are frequently seen over the malar bones, and in some of the hollow viscera—such as the bladder—which may lead to haemorrhage. There is some change in the blood itself; and recent investigations confirm the relationship of the disease to syphilis by the almost constant positive reaction given by Wassermann's test (*vide* Vol. VII. p. 281). The skin becomes sallow and waxy, erythematous disorders occasionally appear, and if the nursing be inefficient bed-sores will form. There is a great tendency to profuse sweating after the fits, and this may lead to sudaminal rashes. "Tache cérébrale" does not seem to me to be of any value as a symptom, at any stage of the disease; it is common to many and various diseases, and may indeed be found in the normal state.

The *pulse* in general paralysis varies greatly; often in the earlier stage it is above the normal in tension and frequency; it soon loses power, often remains rapid, small, and compressible, and may become very slow, pointing to increasing weakness of the heart. The pulse-rate increases, as a rule, with the temperature. There is no characteristic sphygmographic tracing of general paralysis.

Respiration varies, as a rule, with the pulse. After fits it is common to meet with Cheyne-Stokes breathing, which may be of short duration or may persist; in a few cases respiration becomes very slow.

Sleep in the first stage of general paralysis is often good, but the patient does not appear to need so much as usual; he will sleep profoundly, wake early, and begin his restless day betimes. In some cases this will lead the patient to the extravagant notion that he has discovered the root of all evil to be too long a rest in bed. Sleepiness is, however, no uncommon symptom of the earliest stage; patients may sleep almost as soon as they sit down; they sleep over a meal, and sleep well at night also. One teacher has gone so far as to assert that sleeping after meals is a cause of general paralysis; it is certainly a common symptom of it. In the second stage sleep is generally more normal; but towards the end there is restlessness, associated with the bodily discomfort of incontinence of urine and, in neglected cases, of bed-sores.

The *urine* of the general paralytic presents no constant abnormal quality; I have met with sugar, and also with albumin, and not infrequently I have noted a high specific gravity, with abundance of lithates, which corresponds with the rapid tissue-changes of the body. Moreover urea and uric acid in excess, or sulphates in excess, are common. Occasionally, after very severe convulsions, I have met with albumin in the urine.

Remissions of the symptoms, both mental and physical, may occur in most of the periods of the disease. These are most common in the earlier stages, and they are more frequently met with in patients who have begun with symptoms of maniacal excitement. In part this is due to the fact that such patients have accentuated their symptoms by giving way to alcoholic and other excesses. The remissions may be complete or partial; thus, as I have said, both mental and physical symptoms may disappear for a time, but it is much more common to see cases in which the mental symptoms subside while the physical ones are progressing.

I have seen one patient who, while excited shewed little or no ataxia in his gait, but who, as soon as his mind was more clear, was quite unable to stand alone; in this case there were several remissions, each associated with similar symptoms. I think that the symptoms which are most persistent are those affecting the finer adjustments, such as speech and writing. During the remissions the patients may resume their professional work for a time, and may rejoin their families; though, as a rule, the return to old ways leads to rapid recurrence of the symptoms. It is rare to meet with patients who have had more than one complete remission. Acute stages may be followed by intervals of quiescence, and these again may be followed by excitement; but after each period of excitement there is a marked degradation in the patient's state of mind. These cases with recurring attacks resemble in many ways the general paralysis of the double form.

Arrest in progress of the disease without return of sanity may occur even when the patient has passed into the paretic stage; and, in one case at least, I have seen the disease arrested for many years after the symptoms had gone so far as to lead me to think the patient was dying of fits in the last stage. Arrest or remissions may follow injuries or acute diseases, such as pneumonia; or they may follow suppuration, or some skin affection, or very rarely after slight fits.

The *termination* of general paralysis may be natural or accidental. The natural end depends on the progressive disease affecting the whole system; thus exhaustion and slow death by heart or lungs may occur; or the exhaustion may depend on inability to assimilate proper food: besides the weakness there may be diarrhoea, bed-sores, and vesical catarrh. Implication of the bulb may lead to irregular breathing, and to defect of power in respiration; and this may cause clogging of the lungs, pneumonia, and death. Phthisis, or at any rate degenerative lung disease, may end the case. Whether the phthisis be more often fibroid than tuberculous, I cannot say. The fits of one kind or another, which may come on at any stage, may cause death either directly by their frequency, or be associated with some brain lesion or vascular lesion, such as haemorrhage, which may lead to death. Such are the terminations which I have called natural.

The accidental terminations of general paralysis may come by fracture of ribs leading to pleurisy or pneumonia; and by fractures of other bones leading to other complications. Bruising may lead to haematoma, which may exhaust the strength, or give rise to blood-poisoning or to abscesses. Local haemorrhages may follow slight bruises on the surface, or be shed into the membranes of the brain. Exposure to heat or cold may cause death. Impaction of food in the air-passages or in the oesophagus, or the inhalation of fluid during a fit or as a result of paralysis about the throat, may cause immediate or more distant death; suffocation may occur during a fit if the patient turn on his face. Suicide may be the result of such a delusion as that the patient can fly; or it may be the result of mental depression.

Diagnosis.—It is not always possible to distinguish between general paralysis and some other forms of mental disorder; each stage of the disorder has its difficulties. No single symptom can be considered as pathognomonic; to establish the existence of the disease we must prove the presence of both bodily and mental symptoms, which on the whole are progressive. In forming a judgment it is well to remember that there is no form of insanity which may not be associated with general paralysis. In examining male patients of middle age the question of general paralysis must always be taken into consideration. General paralysis is rarely detected in its earliest stages, which stages are not recognised until the disease is fully established. Neurasthenia may be the starting-point of general paralysis; therefore in every case of extreme nervous weakness it is well to examine the state of the pupils and the knee-jerks, and to look for any evidence of the emotional instability and change in temper which are common in this disease. If hysteria occur in middle-aged men it is well to look out for the above signs of degeneration; and in both neurasthenia and hysteria any changes in articulation and in writing must be closely observed.

Convulsive or paralytic seizures of a transient kind must not be overlooked, as they often precede the more marked signs of general paralysis. The occurrence of such fits or palsies for the first time in men without cardiac or renal disorder, the rapid passing off of the symptoms, and the presence of some such physical signs as pupillary inequality, or sluggishness with defects of expression, suffice to clear up any doubt between epilepsy and general paralysis. The perpetration of criminal acts by men previously moral is often found to be associated with early general paralysis; but in a court of law some marked symptom, such as fits, pupillary changes, or well-marked change in character of a progressive type not attributable to alcoholic excess, must be relied upon. If simple depression or slight hypochondriasis suggest early general paralysis, physical signs of degeneration must be looked for; and I think the most important thing is to observe if there is any inconsistency between the mental complaints and the physical states; thus, the hypochondriacal general paralytic, while making many complaints of his ailments, gains flesh and looks well.

The diagnosis in the acute stage rests chiefly between mania of various kinds and the maniacal onset of general paralysis. I do not think it is always possible to distinguish between *acute delirious mania* which may end in general paralysis and acute delirious mania which may pass off or may end in death.

In all cases of delirious mania not due to alcohol, in which there is a history of syphilis, it is well to be on the look-out for general paralysis. I think in the latter there will probably be found more marked alteration in the articulation, and more marked tremor about the muscles of expression; but in acute delirium also these may be present to some extent. Pupillary inequality may aid diagnosis, but in either case the pupils may be dilated and rather immobile. In ordinary acute mania there may be great difficulty and delay in coming to a definite decision; and the difficulty is the greater when the mania depends on alcohol. As a rule in mania the sleeplessness is better marked, and there is rather more persistence in one line of thought than in the acutely maniacal stage of the general paralytic, in whom there is greater mobility and more rapid change of ideas. In general paralysis, again, the exaltation is more marked and more variable than is common in mania. In general paralysis drugs and alcohol seem to have a greater effect than in maniacal cases. If in mania we meet with great variability, intolerance of drugs, pupillary inequality, and change in speech and expression, general paralysis may be suspected. If the earlier symptoms be melancholic there is nothing characteristic in these alone. The melancholia may be of any form; but if with melancholic symptoms there is "physical inconsistency," that is, if the patient gain flesh, though still complaining that he is ruined or is a miserable sinner, and if it be found that his pupils are unequal and indolent, and his speech defective, then general paralysis is to be feared. The occurrence of convulsive seizures often clears up the diagnosis. In some cases when stupor occurs early in general paralysis great doubt may arise: in these cases, if the patient gain in flesh without losing the apparent melancholy, we should search for inequality of pupils and for tremor about the facial muscles; but, again, it is common for the nature of the disease to remain unsuspected till convulsive seizures occur, or till the patient passes into a state of excitement. It is noteworthy that the majority of melancholic general paralytics have distinctly hypochondriacal ideas—the notion that their bowels are occluded being a common one.

In *progressive dementia* there may be great difficulty, for the mental symptoms are exactly the same in this and in general paralysis; but, as a rule, the progress is more rapid in the latter than in ordinary dementia: the latter, again, is more often associated with age or some definite cause of physical weakness, whilst general paralysis occurs in middle life. If in progressive dementia there are signs of muscular weakness, such as tremors and pupillary inequality and defective articulation, general paralysis must be suspected. The cases of simple general paralysis seen in general hospitals are usually of this type; convulsive seizures frequently reveal their true nature.

Alcoholic Disorders and General Paralysis.—As already pointed out, the symptoms of general paralysis are also the symptoms of alcoholism; hence, unless the history be very clear, it is impossible to form a correct opinion at once. In some cases, indeed, intemperance and increased nervous instability are symptoms of general paralysis. The excitement of delirium tremens resembles the delirious onset of general paralysis; and it is well to remember that delirium occurring in a man as the result of comparatively slight alcoholic excess may depend on this kind of degeneration. The acute alcoholic patient, as a rule, sleeps worse than the general paralytic, and has much more marked visual hallucinations of the terrifying kind. The diagnosis must be made from the history, and the existence of some change in the pupils, speech, or writing; but in spite of the most careful consideration mistakes will occur, and time alone can determine the nature of the disease. I think that in every acute case of mental disorder in which there is any suspicion of alcoholic causation time for observation must be insisted on. Convulsive seizures may result from alcoholic excess, and I do not think anything but time can decide on the cause of these fits. Chronic alcoholism resembles the second stage of general paralysis in many ways; but as a rule there is more evident loss of recent memory in the alcoholic than in the general paralytic; and the drinker "makes believe" more, talking of what he has been doing and seeing, when he has in fact never left his room. There is no pupillary change, the reflexes may be normal, and, though the facial expression is changed, there are not the tremor and the defective articulation which are so common in general paralysis.

Lead-poisoning will produce any of the ordinary symptoms of general paralysis, and may, indeed, be the cause of the disease; we must make sure, therefore, from the history and the physical signs that lead is the cause. The blue line on the gums, the tenderness and the early paresis of the extensor muscles of the arms are the chief points of distinction. I have never seen a case in which morphine or any other vegetable alkaloid has produced symptoms which could be confused with general paralysis; but I have seen several chronic takers of chloral, with loss of mental and muscular power, who were not easily distinguished at first from general paralytics. In these, as in drunkards, removal of the drug and careful observation are our only safeguards. Influenza has in many cases set up degenerative changes which turned out to be general paralysis; it is therefore not to be forgotten that general paralysis may follow rapidly on influenza.

The *convulsions of renal disease* may be mistaken for general paralysis; and, in some cases of alcoholic kidney disease, dementia may have been slowly coming on before the fits, and difficulty may thus arise; but, as I have said, albuminuria is very rarely met with in general paralysis.

Syphilis and General Paralysis.—As I have already said, syphilis is a common cause of general paralysis, and the affinities between some cases of brain syphilis and general paralysis are very well known. The chief point is this, that in syphilitic brain disease apart from general paralysis we expect to do good by specific treatment, whereas in general paralysis this treatment fails; the nocturnal headache and oculomotor palsies are very common in brain syphilis, and very rare in general paralysis. In cases of progressive mental decay, in which there has been syphilis with symptoms pointing to coarse intracranial lesion, the implication of any cranial nerve is against general paralysis; though it must be remembered that in general paralysis such a nerve implication may occur, especially in the prodromal stages. That a widespread syphilitic cerebral arteritis is often mistaken for general paralysis of the insane has been pointed out by various observers. Tabes may be due directly to syphilis or be a part of the general paralysis.

I do not think it necessary to describe the so-called *pseudo-general paralysis* (*vide* p. 341). Suffice it to say that in many cases of syphilis there are brain symptoms which prove not to be progressive, and which are not part of a steady process of decay.

Tumours of the brain, whether cancerous, syphilitic, or gliomatous, may lead to difficulties in diagnosis. A history of malignant growth elsewhere might assist the diagnosis; but the presence of local headache, of vomiting, of optic neuritis are more important distinctions. The better-marked effects left after convulsions due to brain tumour are likewise important. The mental state of the patient with brain tumour is, as a rule, one of apathy rather than of excitement or disorder (*vide* also p. 272).

Tumours about the pons or cerebellum are also at times difficult to distinguish from general paralysis; but, as a rule, the staggering gait is greater than with general paralysis of the same duration; moreover, optic neuritis is pretty sure to be present, with vomiting and general affection of certain groups of neck muscles.

Disseminated sclerosis may in some cases lead to confusion; when this disease was first demonstrated in England, by Moxon, I saw many cases of general paralysis, in general hospitals, which were regarded as insular sclerosis. Insular sclerosis usually occurs in young persons, and its progress is slower; there is a difference in the speech which is more staccato, the movements are rather jerky than tremulous, at least for a time; there is often nystagmus, which is very rare in general paralysis, and muscles are often picked out in various groups; the special degradation does not follow the lines of latest and most special development (*vide* also Vol. VII. p. 844).

Paralysis agitans may be associated with mental weakness, but, generally speaking, the character of the movement is quite unlike that met with in general paralysis; the age of the patient also aids the diagnosis.

Sunstroke may give rise to nearly all the symptoms of general paralysis; and a history of sunstroke occurring in a tropical climate and apart from alcoholic excess must make one hesitate in giving a final judgment on a case.

Epilepsy is hard to distinguish from general paralysis. If the fits alone are considered, there is nothing distinctive about them; they may affect the same centres in the same way; but whereas epileptic fits usually pass in a short time leaving no paralysis, in general paralysis the parts affected are almost always paralysed or enfeebled for some hours or even for days after a fit. The epileptic fit, if the first, does not often leave much mental deterioration, whilst in general paralysis a fit often leaves the patient docile and quiet, however excited and grandiose he was before it. In some cases slight attacks, as of *petit mal*, occur in general paralysis; and in such cases the difficulty is the greater because there is but little mental change after or with them; in such cases we must depend on the pupillary or other general symptoms of general paralysis.

Apoplectic fits occur in general paralysis, and, as with epilepsy, they cannot at first be distinguished from true apoplexy; but with true apoplexy the paralysis is more permanent, leaving the patient for weeks hemiplegic, so that there may be pronounced weakness of one limb long after recovery from the other symptoms, whilst apoplectic fits in general paralysis will leave the patient very little worse in body, though vastly deteriorated in mind. The frequency of fits of this kind may also indicate their nature. I have known a patient have a dozen severe apoplectic fits, yet recover a fair amount of mental and physical power. In some cases apoplexy proper may appear to be the starting-point of the general paralysis. Difficulty may arise in the early stages in cases of locomotor ataxia with psychical symptoms, but in these cases the duration of the disease without mental deterioration clears up the diagnosis.

Probably the greatest difficulty arises in the case of men who have many of the symptoms, both bodily and mental, of general paralysis, but who prove later to be suffering from mental and moral perversion, and not from progressive disease. I can best make this clear by describing a case.

A middle-aged man of very neurotic family, married, with no family, with no history of syphilis, who had led a moral, respectable life, but who had suffered from various gouty symptoms, had a severe bodily illness from which he slowly recovered. After this he was a changed man, his conversation was no longer refined, he spoke openly of sexual matters, and made ribald jests before his wife and family. He then got entangled with a notoriously profligate woman, and was seen with her openly. He even proposed to introduce her to his wife and friends. He could not see the impropriety of his conduct, but suggested that his wife should follow similar ways. He retained his affection for wife and family, though still leading this most immoral life. This patient after years of this conduct once more returned to his home and died in the odour of sanctity, but not of general paralysis. He was moral and weak-minded in the end.

I can only repeat that although such cases are almost always considered to be general paralysis, yet the patients may go on unchanged for many years. I have heard them described as cases of satyriasis. Probably now they will be grouped with the cases of degeneration associated with arteriosclerosis.

It remains to be added that of recent years the examination of the cerebrospinal fluid obtained by lumbar puncture has proved of assistance in diagnosis. In organic diseases with inflammation of the cerebrospinal meninges there is an increase in the amount of albumin contained in this fluid and also lymphocytosis ; the meningitis may be due to various causes including syphilis. In the early stages of a doubtful case excess of albumin and lymphocytosis would strengthen the diagnosis of general paralysis, whilst if these conditions were absent the disease might be excluded. As far as insanity is concerned, lymphocytosis with plasma-cells, especially those with a lattice-like structure of the protoplasm, is regarded by recent writers as a diagnostic of general paralysis. With lymphocytosis alone there is an element of doubt, since persons who have had syphilis long since may develop insanity in one form or another (not general paralysis) and lumbar puncture may in such cases shew lymphocytosis. Much attention is at present paid to the serum reaction in cases of general paralysis. It has been found that in such the cerebrospinal fluid or blood-serum, or both, gives in a very large proportion a positive reaction to the Wassermann test (*vide* Vol. VII. p. 281). It appears probable that the test further elaborated will afford a trustworthy means of diagnosis between general paralysis and non-syphilitic diseases simulating it. The results obtained at the same time strongly support the view that without syphilis there is no general paralysis. The Wassermann reaction shews the existence of lues in persons whose history gives no indication of its presence.

Criminality or General Paralysis.—First, as an early symptom of the disease, the patient may lose control and be guilty of impulsive criminal acts; in a moment he may commit a murder or cause grievous bodily harm; he may destroy property or do other acts which are simply due to his defective power of control; like many persons who have suffered from injury to the head, he may be more easily affected by alcoholic and similar stimulants. Suicide may result from similar want of control.

Wrongful desire of one kind and another may arise, to be gratified without regard to social law; thus, the patient may steal, though in my experience the general paralytic who commits theft does so from a conviction of his great wealth, and his right to all he may desire.

To complete this part of my subject I will now repeat one or two points already referred to.

As to diagnosis, male hysteria may simulate general paralysis, yet general paralysis may begin as hysteria. Exaltation of ideas may occur in mania, especially if due to alcoholic exaltation; it may be the natural result of defective control in youth—an overgrowth of conceit, or it may result from delusions with an apprehension of being noticed or watched; it may be a steady growth of the imagination, the "may be" becoming a reality; but in general paralysis, as a rule, the exaltation is universal and of a benevolent character.

It is difficult to tell whether a patient be suffering from mania of the double form, or from general paralysis of the double form; and time alone can settle the question.

Paraplegia in its various forms may be associated with general paralysis, and set in before, during, or after the mental symptoms; therefore in every such case the mental as well as the bodily weakness must be studied.

Sexual faults or crimes are commonly met with; these acts may arise from exaggerated or inflamed desire, or from loss of control. Indecent exposure may arise from unnatural desire or from simple forgetfulness. Rape and assault on children, indecent and criminal assaults, are apt to occur likewise. In the earlier stages of the disease there is almost always great increase of sexual desire, and some increase of sexual power may be present; and this may last for a considerable time.

The period when criminal acts are most likely to be done is before the full development of the more marked symptoms, or in the first period of their manifestation; hence it is of great importance that careful examination should be made for physical signs of degeneration in suspected individuals.

As far as *mental capacity* is concerned, it is certain that some general paralytics become unfit to dispose of their property long before they can be certified as insane; for in the earlier stages of the disease great alteration of temperament and disposition takes place as well as a tendency to act without due judgment; and later the memory may be defective also. Thus, as the result of progressive brain disease, a patient may be alienated from his relations, attracted by some designing person, probably a woman, and, as the result of these perverted feelings, may make an unjust will. On the other hand, a general paralytic may make a perfectly reasonable will, even in the late stages of the disease, provided the symptoms chiefly affect the muscular side of him. It was even allowed in the case of Crabtree, tried in 1894, that a general paralytic during a period of remission may make a valid will, though he had been previously in a state of apparent dementia, and had had fits.

A general paralytic, indeed, may be guilty of crimes of various kinds as a result of his disease, but may be held capable, nevertheless, of making a will even at an advanced stage of the disease (see Brown *v.* Penn, Nov. 1895).

Prognosis.—This is rather concerned with the probable course and duration of the case than with the result. As I have said, sometimes early threatenings of the disease, occurring in likely subjects, may pass off. In some cases remissions may be so complete for many years that recovery is alleged; but the end of nearly all these cases proves that the respite was but arrest or remission. I will not say that no case of general paralysis ever ends in recovery, but it is excessively rare. The more acute the onset, the greater the prospect of remission; and the stronger the neurotic heredity, the greater, I believe, is the prospect of remission and of the disease running an irregular course. It is very rare for more than one complete remission to occur in any case, save in those of the double form. In a few cases the quiet demented stage may last for years. General paralytics live longer in asylums than in their homes; they live longer in small asylums than in larger ones, and they live longer in the country than in towns. It is not possible to say whether a particular case will run a short course or a long one; yet as a rule the higher the temperature the greater the danger of rapid decline. The presence of fits generally hastens the progress; but in rare cases fits seem to arrest the disease for a time, or even to lead to remissions. Though a patient may have been quiet and placid in the earlier stages it is not certain that he will not have a maniacal outbreak later. The cases of general paralysis of the double form, or of the circular form, generally last longer than others, their duration being extended by the restful periods and prolonged remissions. A clear history of syphilitic infection within five years will give more prospect of amelioration; but, however distinct the history of syphilis, if of many years' standing, I do not think it really affects the prognosis; that is to say, if the diagnosis of general paralysis be confident.

Treatment may be divided into general and special. The first consideration is whether the patient can be properly treated at his own home, or at any rate out of an asylum. Seeing that these cases are almost hopeless, it is well in all cases to decide at once in favour of removal from home to definite asylum care, except in those of simple progressive dementia without excitement, very distinct depression, or marked delusion. The treatment of a very wealthy general paralytic may be undertaken in a private house with large grounds away from a city; but even then, in my belief, it is neither the best nor the kindest treatment. Whether away from home or not, the patient's nearest relations, more particularly his wife, should not live with him. The less emotion there is in the life of the general paralytic the longer will his life last. Indulgence of the sexual passion is injurious, and the prospect of children begotten by such a father is gloomy. The wife in such cases, if installed as nurse, will probably break down in health; and I frequently see neurasthenia as the result of wifely devotion to general paralytic husbands. Sea voyages, though so often recommended,—only, it seems to me, in order to shirk the alternative of an asylum,—are rarely, I think, of any service.

The golden rule of treatment is complete rest. The simplest country life, with as much sleep as the patient can take, is to be desired; only moderate exercise must be allowed, and of course riding and cycling have to be given up. During periods of remission it is difficult to prevent the return of the patient to his own home; but this return is almost always followed by relapse. It is better to send the patient away for the interval; possibly for a short sea voyage, or else to the house of some medical man living inland in the country. The diet of the general paralytic must vary with the stage of the disease; in all I think alcohol is to be avoided. The simpler the food the better; fish, fruit, and plain food are better than much meat and fancy dishes. If any stimulant be found necessary it should be as weak spirit and water or light wine. Tobacco may be of service in the earlier stages of restless excitement; but there is a danger that the patient may smoke too much.

The general paralytic must be warmly clad, and his clothes carefully looked after, as he is very careless of himself.

I do not think that electricity in any form is of service. Specific medical treatment has made but little progress as yet. If antisyphilitic treatment has not been fully tried, I recommend, if the patient be quiet enough, the course of treatment at Aix-la-Chapelle. If this has already been tried, we know of no specific. In some cases counter-irritation along the spinal column has been tried, and said to be of use. Iodine liniment to scalp and spine has been recommended. Surgical treatment has been tried with the idea of relieving pressure, but the results of trephining have not been encouraging. I trust that sooner or later some antitoxin will be found which will aid in defeating this malign disease. Calabar bean was used for some time, but I cannot report favourably on its action; nux vomica is useful, with or without quinine and hydrobromic acid; I cannot say that setons in the neck have produced any good effect. In ordering drugs for general paralytics we have to remember that they are very readily affected by strong remedies; I have known minute doses of alkaloids produce in them very alarming symptoms. If there is much sleeplessness I prefer paraldehyde in dram doses to any other drug; I do not like morphine or hyoscine; veronal, trional, or sulphonal generally fail to produce sleep. During the occurrence of fits we may use chloroform or nitrite of amyl; and nitroglycerin, too, has been found of service. If the *status epilepticus* continue to an alarming extent, I advise an enema containing 20 grains of chloral with 20 grains of a bromide. Other measures consist in the injection of camphor or ether, the infusion (subcutaneous or venous) of physiological salt solution and the promotion of diaphoresis and diuresis. Some writers describe good results obtained by subcutaneous injection of salts of hyoscine. It is essential in these cases to consider details very carefully; thus, it may be necessary to empty the bladder and perhaps to wash it out. Soreness of the skin may be prevented by great attention to keeping it dry, and also by the application, night and morning, of hot water and dry Castile soap. If haematoma occurs it is best treated by immediate and vigorous blistering; this failing, it may be necessary to slit the part open and remove the clot.

G. H. Savage.

REFERENCES

General Bibliography: 1. Bianchi. *Paralisi progressiva e frenosi sensoria*, Naples, 1895.—2. Blandford. "Insanity," *Twentieth Century Practice of Medicine*, London, 1897, xii. 151.—3, Chase. *General Paresis*, Philadelphia, 1902.—4. Christian. "Researches in the Etiology of General Paralysis in Men," *Amer. Journ. Insan.*, Utica, 1888, xliv. 488.—5. Hyslop. "General Paralysis," *Encyclop. Med.*, Edin., 1900, iv. 118.—6. Könfeld. *Über Paralyse bei weiblichen Geschlecht*, 1877.—7. Krafft-Ebing. *Die progressive allgemeine Paralyse*, Wien, 1894.—8. Mairet et Vires. *De la paralysie générale, étiologie, pathogénie, traitement*, Paris, 1898.—9. Mendel. *Die progressive Paralyse der Irren*, Berlin, 1880.—10. Mickle. *General Paralysis of the Insane*, 2nd ed., London, 1886.—11. *Idem.* Tuke's *Dictionary of Psychological Medicine*, London, 1892, i. 519.—12. Selvatico-Estense. "Gli studi recenti sulla demenza paralitica," *Riv. sper. di freniat.*, Reggio-Emilia, 1894, xx. 575.—13. Tuczek. *Beiträge zur pathologische Anatomie und zur Pathologie der Dementia paralytica*, Berlin, 1884.—14. Voisin. *Traité de la paralysie générale des aliénés*, Paris, 1879. **For general references the following Journals**: 15. *Allg. Ztschr. f. Psychiat.*, Berlin, 1844-1910.—16. *Am. Journ. Insanity*, New York, 1844-1910.—17. *Ann. méd.-psychol.*, Paris, 1843-1910.—18. *Arch. f. Psychiat.*, Berlin, 1868-1910.—19. *Arch. de neurol.*, Paris, 1880-1909.—20. *Arch. delle malattie mentali.*—21. *Arch. di psychiatria*, Torino, 1880-1909.—22. *Brain*, London, 1878-1910 (General Index, vols. i.-xxiii. 1878-1900).—23. *Centralbl. f. Nervenh.*, Coblenz, 1878-1910.—24. *Deutsche Ztschr. f. Nervenh.*, Leipzig, 1891-1910.—25. *Encéphale*, Paris, 1881-1887.—26. *Journ. f. Psychol. u. Neurol.*, Leipzig, 1902-1910.—27. *Journ. Ment. Sc.*, London, 1855-1910 (General Index, vols. i.-xxiv., 1855-1879, and i.-xxxix., 1855-1893.—28. *Journ. Nerv. and Ment. Dis.*, New York, 1876-1910.—29. *Monatsschr. f. Psychiat. u. Neurol.*, Berlin, 1897-1910.—30. *Neurol. Centralbl.*, Leipzig, 1882-1910.—31. *Névraxe*, Louvain, 1900-1910.—32. *Rev. neurol.*, Paris, 1893-1910.—33. *Rev.*

Neurol. and Psychiat., Edin., 1903-1910.—34. *Riv. sperimentale di freniat.*, Reggio-Emilia, 1875-1910. **Ophthalmic Observations in General Paralysis**: 35. ALLBUTT, Sir T. C. "State of the Optic Nerves and Retinae in the Insane," *Med.-Chir. Trans.*, London, 1868, xxxiii. 97.—36. COLUCCI. "Sulla cosidetta retinite dei paralitici," *Ann. di nevrol.*, Napoli, 1894, n.s. xii. 317.—37. DAWSON and RAMBAUT. "Analysis of the Ocular Phenomena in Forty Cases of General Paralysis of the Insane," *Brit. Med. Journ.*, 1898, ii. 687.—38. HOLDEN. Early Ocular Signs of Dementia Paralytica," *Journ. Nerv. and Ment. Dis.*, New York, 1905, xxxii. 713.—39. LEWIS. "Remarks on Ocular Symptoms of General Paralysis of Insane," *Brit. Med. Journ.*, 1896, i. 1021, 1079.—40. OLIVER. "An Analysis of the Ocular Symptoms found in the Third Stage of General Paralysis of the Insane," *Trans. Amer. Ophth. Soc.*, Hartford, 1888-90, v. 646. **Pseudo-General Paralysis**: 41. BAILLARGER. "Doit-on dans la classification des maladies mentales assigner une place à part aux pseudo-paralysies générales," *Ann. méd.-psychol.*, Paris, 1889, 7. sér., ix. 196.—42. MOTT, "Some Conditions which simulate General Paralysis of the Insane," *Internat. Clin.*, Phila., 1897, 6th ser., iv. 134.—43. RÉGIS. "Neurasthénie et paralysie générale," *Presse méd.*, Paris, 1897, 153-158. **Relationship of Syphilis to General Paralysis**: 44. BRAMWELL, B. "Etiological Relationship of Syphilis to Tabes and General Paralysis of Insane," *Clin. Studies*, Edin., 1905-6, iv. 97.—45. FOERSTER. "Über das Wassermann-Plautsche Verfahren der Serodiagnostik bei syphilidogenen Erkrankungen des Zentralnervensystems," *Allg. Ztschr. f. Psychiat.*, Berlin, 1908, lxv. 158.—46. FOURNIER. "Quelques propos sur la paralysie générale et la syphilis," *Bull. Acad. de méd.*, Paris, 1905, 3me sér., liii. 155, 187, 206, etc.—47. MASLAKOWETZ und LIEBERMANN. "Theorie und Technik der Reaktion von Wassermann und die diagnostische Bedeutung derselben," *Centralbl. f. Bakteriol. u. Parasitenk.*, Jena, 1908, Abt. i. (*Orig.*), xlvii. 379.—48. MORET-LAVALLÉE et BÉLIÈRES. *Syphilis et paralysie générale*, Paris, 1889.—49. SAVAGE. "Syphilis and its Relation to Insanity," *Amer. Journ. Insan.*, N.Y., 1888, xliv. 323.—50. STERTZ. "Die Serodiagnostik in der Psychiatrie und Neurologie," *Allg. Ztschr. f. Psychiat.*, Berlin, 1908, lxv. 565.—51. VORBERG. *Dementia Paralytica und Syphilis*, Leipzig, 1906. **Various Textbooks on Insanity**: ENGLISH. Blandford, 1892; Clouston, 1904; Lewis, 1899; Maudsley, 1895; Savage and Goodall, 1907.—AMERICAN. Church and Petersen, 1907; Dana, 1908; Kellogg, 1897; Spitzka, 1883.—GERMAN. Schul, 1886; Krafft-Ebing, 1893; Kraepelin, 1899; Kirchhoff, 1892; Arndt, 1897; Leidesdorf, 1889; Wernicke, Weiss, 1889; Ziehen, 1902; Griesinger, 1892.—AUSTRIAN. Meynert, 1890; Benedikt.—FRENCH. Voisin, Marcé, Billod, Régis.—ITALIAN. Agostini-Tamburini, 1900.

G. H. S.

APHASIA AND OTHER SPEECH DEFECTS[1]

By H. CHARLTON BASTIAN, M.D., F.R.S. Revised by JAMES COLLIER, M.D., F.R.C.P.

Introduction.—The word "aphasia" has been commonly used as a general term including the widest variety of speech-defects dependant upon lesions in different parts of the brain. Its use in this respect has led to the employment of the terms "motor aphasia" and "sensory aphasia" for the two chief groups of speech-defects which respectively result from damage to the emissive and to the recipient parts of the cortical region which subserve speech. The names of the great discoverers, Broca and Wernicke, are frequently used to distinguish these two main groups of speech-defects, "Broca's aphasia" corresponding with the former and "Wernicke's aphasia" with the latter, just as the two regions of the brain, lesions of which are responsible for these groups, are called respectively "Broca's area" and "Wernicke's area." This terminology has the great disadvantages that it includes phenomena under the same name which it is all essential to keep clinically distinct and which result from lesions in widely separate parts of the brain, and further that it does not allow of a comparable nomenclature for the phenomena of a similar order which result from lesions of regions of the brain other than those concerned with speech.

---

[1] Throughout this article the use of the first person singular refers to Dr. Bastian.

In this article the word "aphasia" is restricted to the defects caused by damage to Broca's area in the region of the left third frontal gyrus, and it is identical with the terms "Broca's aphasia," "motor aphasia," and "apraxia of the speech musculature."

The defects in the recognition and in the recall of speech that result from lesions of Wernicke's area in the left temporo-occipital region, which have been called the "sensory aphasias," are now termed "verbal agnosia," and the familiar and convenient clinical expressions word-deafness and word-blindness or alexia are retained for the auditory and visual forms of verbal agnosia. Verbal amnesia is a most convenient designation for the inability to recall word-images, and it has been widely used in the same sense that we now employ the term verbal agnosia. Since the speech-centres both on the receptive and on the emissive sides are centres for word-memory, the term amnesia is applicable to the defects produced by lesions of any of them, though the revival of the word-images in the motor speech-centre plays but an insignificant part in the processes of memory and thought. Moreover, this word does not express the inability to recognise, which is so inseparably connected with inability to recall in lesions of the receptive speech-centres.

The left hemisphere of the brain exercises a dominant influence in connexion with language and with the more complex purposive movements of both sides of the body in right-handed persons. Whereas in left-handed people the right hemisphere is commonly, but by no means invariably, the dominant one. The degree to which the less important hemisphere intervenes in the function of language is of great importance, not only physiologically, but also from the point of view of the facility with which compensation and recovery may occur in patients in whom speech-function is lost or disturbed. The possibility of compensation and recovery after destruction of the speech-centres in the dominant hemisphere varies remarkably in different individuals. It is much greater in early childhood, when, presumably, the localisation of higher cerebral function is not fixed beyond alteration, and when the mastership of the dominant hemisphere in the speech-function has not as yet deprived the other hemisphere of its capacity of speech development. Before a child has reached the age of six years, destruction of the speech-centres in the dominant hemisphere, however complete, never produces permanent loss of the speech-function, but recovery always occurs, provided that a fair degree of intelligence remains. From this age onwards the capacity for compensation lessens rapidly, and in adult life it is a rare exception.

Forms of Word-memory.—There are four different kinds of word-memory, each having its seat in a definite region of the cerebral cortex. These are (i.) the auditory word-centre, where the sounds of words are registered; (ii.) the visual word-centre, where the visual images of letters and words are registered; (iii.) a glosso-kinaesthetic centre (eupraxic centre), where the combined impressions which pass to the cortex as the result of the movements of the lips, tongue, larynx, and other parts concerned with articulate speech are registered; and (iv.) a cheiro-kinaesthetic (eupraxic centre), where the sensory impressions resulting from the movements concerned in the act of writing are registered.

The auditory word-centre is a specialised portion of the general auditory cortex, and it is situated in the posterior two-thirds of the left upper temporal convolution and in the posterior part of the second temporal gyrus, possibly extending for some distance around these gyri. The visual word-centre is situated immediately behind the auditory word-centre, extending on to the angular and supramarginal gyri. So close are the anatomical and physiological connexions between these two centres, and so rare have been the cases in which a lesion of the angular gyrus or of the supramarginal gyrus has been associated with word-blindness uncomplicated with any word-deafness, that the existence of a visual word-centre separate from the auditory word-centre has often been questioned. Pierre Marie holds that the recognition and recall of visual language is a function of the whole of the auditory word-centre, and that there is no separate visual word-centre, whilst Dejerine agrees with me in upholding the localisation of this function as a separate centre in the angular gyrus. It is possible, however, that these two centres overlap to some extent. The glosso-kinaesthetic centre is situated in the posterior part of the left third frontal gyrus, probably extending on to the anterior convolution of the insula and on to the contiguous part of the second frontal gyrus. The cheiro-kinaesthetic centre occupies the posterior part of the left second frontal gyrus. These two centres are specialised portions

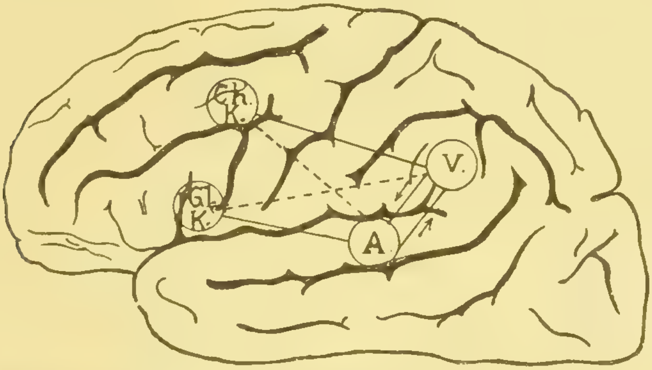

FIG. 20.—Diagram shewing the approximate sites of the four word-centres and their commissures. A, auditory; Ch.K, cheiro-kinaesthetic; Gl.K, glosso-kinaesthetic; V, visual word-centres.

of the general eupraxic centres, which are situated in the posterior parts of the three frontal gyri on the left side and are the centres for the innervation of the specialised purposive movements upon both sides of the body. They are situated immediately in front of, and are in close connexion with, the general kinaesthetic centres (so-called motor cortex), which recent investigation has shewn to be narrowly confined to the ascending frontal gyrus. The outgoing path from these two centres is a mediate one by way of the ascending frontal gyrus and its projection the pyramidal tract; that from the glosso-kinaesthetic centre going to the motor centres in the bulb, and that from the cheiro-kinaesthetic centre going to the motor centres in the spinal cord through which the act of writing is performed.

The afferent connexions of the speech-centres and the inter-connexions between them are of great importance. The auditory word-centre, as part of the general auditory centre, receives the projection of the central acoustic path, and it is in relation with the auditory centre of the opposite hemisphere by means of the fibres of the corpus callosum. The visual word-centre is in direct relation with the primary visual cortex in the cuneate lobes of both hemispheres by means of fibres which converge upon this centre as they come forward. A subcortical lesion in the anterior part of the occipital lobe is likely to sever this path completely, and so isolate the visual word-centre from its afferent connexions, giving rise to "pure" or "subcortical" word-blindness. The connexions of the auditory and visual word-centres with the centres in the frontal gyri are by means of a large tract of fibres which, leaving the white centre of the temporal lobe, traverse the temporo-parietal isthmus and run forwards beneath the cortex of the insula to their destination. A lesion of the subcortex of the insula is likely to isolate these centres, and "trans-cortical aphasia" results. The connexion of the speech-centres in the frontal lobe with the pyramidal system is by means of a subsulcine system of fibres. A subcortical lesion in this region may completely destroy this system of fibres, and, by isolating these centres upon the outgoing side, may produce the condition of "pure" or "subcortical" aphasia.

Order of Development of different Speech-centres.—A clear conception of the order of development of the speech-faculties may be gained from a consideration of the manner in which a child learns language. He learns to understand the meaning of spoken words long before he is able to speak. His auditory word-centre is the first to be called into play, and it develops in advance of the other speech-centres; and from this time onwards throughout life it remains the chief speech-centre, both for the revival of word-memories in the process of thought and as the starting-point for the incitations that result in spontaneous speech. As he learns to speak, the glosso-kinaesthetic centre becomes gradually organised under the influence of incitations from the auditory word-centre, the stimuli passing along certain associational paths which are thus gradually laid down. The glosso-kinaesthetic centre is developed as the subsidiary centre of the auditory word-centre, and such it remains. Later on the child comes to connect certain visual symbols, letters and words, with the sounds of the words he knows, and the visual word-centre becomes organised under the guidance of the auditory word-centre; and simultaneously a double association-path is laid down between the auditory word-centre and the visual word-centre. When the child becomes able to read aloud, there must be activity first in the visual word-centre, then in the auditory word-centre, and immediately afterwards in the glosso-kinaesthetic centre. As the child learns to write, the cheiro-kinaesthetic centre becomes organised under the guiding influence of the visual word-centre, and this is accompanied by the development of association-channels between the two centres. The visual word-centre is in the same relation to the act of writing as is the auditory word-centre to articulate speech. They are the centres of recognition and recall; they are the initiating and driving centres for the centres for writing and speech respectively in the frontal lobes, these latter centres having of themselves little power either of recall or of initiation. In writing from dictation, and probably also in writing spontaneously, the train of activity starts in the auditory word-centre, spreads from this to the visual word-centre, and thence on to the writing-centre, from whence the outgoing stimuli pass over to the executive motor centres. Thus, functional associations of the closest kind become established between the four word-centres, those in the frontal gyri, however, never acting alone, but always in response to incitations coming to them either from the auditory or from the visual word-centre. The regular order of association above given is probably not always observed. There is reason to believe that in some persons, who are strong "auditives" and well accustomed to write, the stimulus for the act of writing may pass at once from the auditory word-centre to the writing centre without passing through the visual word-centre. Similarly, in certain well-educated "visuals" much accustomed to read aloud, the stimulus during the act of reading aloud

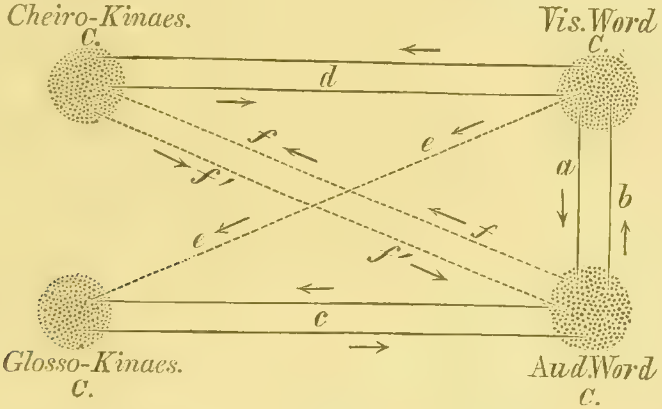

Fig. 21.—A diagram illustrating the relative positions of the different word-centres and the mode in which they are connected by commissures. The connexions indicated by dotted lines indicate possible but less habitual routes for the passage of stimuli.

may pass directly from the visual word-centre to Broca's centre without troubling the auditory word-centre—just as it must do in the case of deaf-mutes who have been taught to speak by lip-reading.

The Part played by the Right Cerebral Hemisphere in Speech.—Several authorities (Lichtheim (53), Wernicke (92), and Dejerine (25)) consider that there are no speech-centres in the right hemisphere of a right-handed person. This view is in our opinion necessarily erroneous. It is clear that from the time of birth the two cerebral hemispheres are equally placed as recipients of the auditory, visual, and kinaesthetic impressions, and it is probable that the development of speech-centres in both hemispheres takes place to a limited, though variable, extent during early life. It is certain that up to the age of six years the less important hemisphere is capable of taking over and completely developing the speech-centres, since a lesion confined to the dominant hemisphere, however extensive it may be, does not produce permanent loss of the speech-functions before the sixth year, provided that a fair degree of general intelligence remains. In right-handed persons the left hemisphere from the time of birth stands hereditarily endowed with a greater receptive capacity than does the right hemisphere, and this major capacity increases with the increasing organisation of the speech-centres in the dominant hemisphere, whilst the organisation of the similar centres in the right hemisphere lessens and soon ceases. It would seem that the capacity of the speech-centres in the right hemisphere to recommence organisation and to attain perfect development, should the left hemisphere fail for any reason, is retained for several years. But in proportion as the perfect functional development and exclusive dominance of the left speech-centres are attained, the potentiality of the right speech-centres is lessened, and finally is lost more or less. It can readily be conceived that the potentiality, which the right speech-centres have, to develop and to take over the functions of the left speech-centres may be retained, and the duration of this capacity may vary within very wide limits in different persons. Such variations may account for the surprising differences in the amount of speech recovery in cases in which similar lesions of the left hemisphere have occurred.

It is probable that the regions of the right hemisphere which correspond with the speech-centres in the left hemisphere act functionally throughout life as adjuvant speech-centres, and that the speech-centres of the two hemispheres are in close association by means of commissural paths forming part of the corpus callosum. It will subsequently be shewn that there is reason to believe that each auditory word-centre is in relation with Broca's centre, since in some cases, in which there has been destruction of the left auditory word-centre, word-deafness has been transitory, and fair speech has soon been possible by reason of the increased action of a highly-developed right auditory word-centre upon Broca's centre, through a diagonally disposed commissure. The question of the relative functions of the right cerebral hemisphere in speech has never received sufficient attention, but it is of great importance when we consider the mode in which restoration of function is brought about in many speech-defects.

The Seat of the Primary Recall of Words.—In silent thought and in ordinary speech the memorial recall of words in the great majority of persons takes place primarily in the auditory centre, though this is immediately followed by more or less activity in Broca's centre. In a very small percentage of persons who are exceptionally strong "visuals" this primary recall may occur in the visual word-centre. All clinical evidence is against the view that initial activity in the recall of words ever takes place in Broca's centre. This, like other kinaesthetic centres, seems only to be called into play at the instigation of stimuli coming from the auditory or the visual word-centres, though the contrary view has been strongly advocated by Stricker, Wyllie, and others.

The Incitation of the Speech-centres.—It is important for the interpretation of speech-defects to bear in mind that the functions of the auditory word-centre are carried on in obedience to stimuli of three degrees of intensity. (1) The weakest stimuli and the first to fail when the centre is damaged are the "volitional" stimuli, under the influence of which spontaneous speech is produced—words being recalled as they are wanted for the expression of thought. (2) Stronger stimuli are those of the "associational" order, such as come to the auditory from the visual word-centre when the latter is actively aroused, as in the process of reading aloud. (3) The strongest stimuli of all, and the last to fail in arousing the activity of the centre, are the "sensory stimuli," which come to the centre directly from without, as when a patient is bidden to repeat a given word. Except in the very rare cases of strong "visuals," in whom words are first revived in the visual word-centre rather than in the auditory, during silent thought, the former centre reacts only to associational and to sensory stimuli, so that amnesia verbalis (a failure of recollection for words in thought) is limited to defects of the auditory word-centre. Broca's centre and the writing-centre react only to associational stimuli reaching them from the auditory and visual word-centres.

Word-images as Integral Components of Percepts and Concepts.—During the organisation of the speech-centres, these centres become intimately linked by associational paths with the cerebral seats of registration of the several sensory impressions. The words heard are in the first place associated in the mind of the child with external objects, and the memories of the visual, tactile, and other sensory attributes which the child derives from the objects become intimately associated with the names of the objects. Thus the name subsequently is revived as part of the perceptive process whenever the object is again presented to the senses. As the development of the speech-centres proceeds, the auditory representative of the name of an object is reinforced by glosso-kinaesthetic impressions as soon as the child learns to speak the word, by visual impressions when he learns to read, and by cheiro-kinaesthetic impressions when he learns to write. The several components of word-percepts are thus brought not only in close relation with one another, but they are also no less intimately associated with the several sensory components of object-percepts. The result is that the hearing or the sight of the name of any object immediately calls up an idea of the object, just as the presentation of the object to sight or to another sense produces a nascent activity in the several speech-centres in which its name is registered, though mainly in the auditory and in the visual word-centres. Words soon become to a very large extent the symbols whereby we carry on our thoughts, and this thinking by means of words becomes all the more thorough as thought becomes more complex. We can think of a particular object as well by recalling the visual image as by recalling the name. But when in thought we have to recall general ideas, such as "animal" or "tree," or still more abstract ideas, such as "virtue" or "vice," it is certain that we use these names as symbols for the more or less complex ideas which they represent. And if we bear in mind that the word-centres in which these words are registered are in structural and functional relation with the seats of registration of the corresponding percepts, concepts, and the like, we can see how the revival of the word as a thought-counter is associated with an almost simultaneous activity in the seats of registration of the corresponding percepts and concepts. In simple acts of perception, and still more in simple thought-processes, there is no limitation of cerebral activity to narrowly localised centres, but widespread processes of activity occur in very various regions of the cerebral cortex and in both hemispheres of the brain. In our considerations of individual cerebral centres and their functions we are apt to forget that the brain acts as a whole, and not in terms of its individual elements or centres, even in comparatively simple operations. We venture to think that many of those who have written on the subject of aphasia have not taken this sufficiently into account, and that the name constitutes an integral element of the percept and of the concept.

Does Conception take place in a Single Centre altogether apart from Perception?—Concerning this most important question much difference of opinion has existed among writers upon defects of speech. Broadbent, Kussmaul, Charcot, Lichtheim, and Wernicke (93) have taken the affirmative position. I was the first to express dissent from this view, and I have since been followed by de Watteville, Ross, Allen Starr, and Wyllie, all of whom have decided against the necessity of postulating a separate centre for ideas or concepts. Recently both Lichtheim and Wernicke have modified their views in that they demand no anatomical separation between the centres for percepts and concepts, and they regard them as two separate functions of the same region. My opposition to this postulation of a single separate centre for concepts or "ideational centre" was based originally upon psychological considerations. It seemed to me wholly unnecessary and at variance with what appeared to be the real nature of the process of perception and conception. Moreover, there is no clear clinical evidence to prove the existence of a separate "centre for concepts," nor are there any existing forms of speech-defect which can only be explained by supposing the existence of a lesion in such a separate centre. I am convinced that the supposed necessity for assuming the existence of a single separate "ideational centre," when seeking to interpret different forms of speech-defect, may be obviated by a proper recognition of the different degrees of functional activity that may obtain in the auditory and visual word-centres in pathological conditions of these centres. Their molecular mobility may be so lowered that they are only capable of responding to powerful stimuli. Thus, whilst volitional recall may be impossible in their province, they may still be capable of acting readily in association with other centres—as when reading may be fluent though voluntary speech is greatly impaired; and they may act still more easily under a direct sensory stimulus—as when a word is repeated which the patient has just heard pronounced.

One cause that has led to the postulation of a "centre for concepts" is an inadequate realisation of the nature of the perceptive process, and of the fact that a name constitutes an integral part in the complex of revived sensory impressions that go to make up a percept. Perceptive processes vary greatly in complexity, and merge by insensible gradations into processes of conception. It is therefore just to suppose that the latter more specialised forms of mental activity, whilst having their roots in perceptive centres, may be completed in outgrowths therefrom—that is, in parts of the brain which are in close structural and functional relation with the several sensory centres. I have commonly spoken of such regions as "annexes" of the perceptive centres. Flechsig has called special attention to four areas of the cortex which differ from the sensory areas, in that they are neither in relation with afferent nor with efferent fibres. He assumes that these regions are concerned with higher mental functions than those occurring in the sensory centres. He terms these regions "association areas." These regions seem to correspond with what I have referred to above as "annexes of the perceptive centres." They occupy a large proportion of the cerebral cortex, and are thus located by Flechsig: (1) in parts of the prefrontal lobe; (2) a large portion of the temporal lobes; (3) a considerable area of the postparietal region; and (4) the island of Reil. These four areas are not directly connected either with afferent or with efferent fibres, and there are other reasons for supposing that they are concerned with higher functions. These regions remain immature and completely devoid of myelin for several months after birth, though at this period the sensory centres have reached comparative maturity. Further, these regions are the parts especially developed in the brain of man as compared with that of the higher animals.

More than twenty years before Flechsig, the late Sir W. Broadbent had pointed out almost exactly these regions as areas of the cortex which were neither in direct relation with peduncular fibres nor with those of the corpus callosum, and he then attributed to these regions just the same functions as those now assigned to them by Flechsig.

It is quite legitimate to suppose that the annexes of the sensory centres correspond with the regions indicated by Broadbent and Flechsig, though the extent of these areas so taken up must remain altogether uncertain. It is also probable that there is no sharp line of separation between these annexes and the several sensory areas, and that the combined sensory areas, together with the annexes, are accustomed to be thrown into functional activity more or less simultaneously. Thus the processes of perception and conception and the revival of words are probably as inseparable in their localisation as they are in their nature and modes of occurrence, and their anatomical substratum must be supposed to occupy a considerable extent of the cortex of both hemispheres.

The cortical centres for the registration and recall of the four different kinds of word-images have already been described tentatively and in a general sense, but something may now be said concerning the sites of the word-centres that could not well have been said at an earlier period. Each of them is probably to be found partly on the confines of its percept-centre and partly on those of its related annex. This supposition is made because some words (especially names of things, persons, and places) are in closest relation with sensory centres; whilst others, such as verbs, adjectives, and other parts of speech constituting the framework of language, are in closer relation with conceptual processes. As has been already pointed out these two forms of functional activity—the percept- and the concept-processes—are absolutely inseparable from one another, and therefore the several word-centres must be in the most intimate relation both with the sensory centres and with their annexes.

If the views above expressed be anything like an approximation to the truth, it may be judged how vain it would be to attempt to base our explanation of any of the different kinds of speech-defect upon the supposed existence of some one separate centre for "ideation," "conception," or "naming" which is connected by means of commissures (long enough and separate enough to permit of isolated damage) with sensory centres on the one side and with motor centres on the other.

The Nature of the Cerebral Lesions producing Speech-defects.—The great majority of the lesions of the cerebral hemispheres which result in defects of speech are focal lesions caused by thrombosis, haemorrhage, embolism, abscess, and new growth. The whole of our knowledge of the speech-functions of the brain must necessarily be based upon deductions from the pathological and clinical phenomena presented by such cases. These deductions are beset with great and varied difficulties, a knowledge of which is all essential to the clear comprehension of the subject. Our conception of the speech-mechanism is built up of certain centres occupying the cerebral cortex, and of afferent, efferent, and associational paths which enter and leave these centres, and which occupy the subcortical white matter. Had we to deal with lesions confined succinctly to the centres alone, or to their inter-connexions and in- and out-going paths alone, in our deductions upon the physiology of the speech-mechanism, our task would be more simple. But the lesions with which we have to deal are never purely cortical. The so-called "cortical" lesions are at the same time both cortical and subcortical, and they are generally much more subcortical than cortical. They are actually lesions of the convolutions as opposed to lesions of the central white matter. Again, the extent of the lesion is often so great as to make impossible any correct correlation between the several clinical phenomena that may present themselves and the parts of the brain destroyed. Further, in connexion with vascular disease, multiple lesions often exist, and some of these may escape recognition, though the most thorough examination may have been made with the unaided eye. In such cases the examination of serial microscopical sections throughout the region of the brain occupied by the speech-centres is the only certain test that will distinguish between the "living" and the "dead," both in tissue and function. This method of examination is now held to be an absolute necessity before any localising deductions can be made in connexion with pathological lesions of the brain. For these several reasons a careful selection must be made of the cases before any conclusion can be arrived at. Those with vast lesions or with multiple lesions must be rejected, as must also be those in which any but the most complete clinical details are available. Moreover, the clinical details must be obtained after the widespread depression of cerebral function, which the occurrence of the lesion often entails, has passed off, and before complicating phenomena that may result from recovery or from compensation have appeared. A very large number of reported cases are valueless from the neglect of these considerations, and there are very few cases in the vast literature upon this subject which are altogether free from the just criticisms that either the extent of the lesion was not delineated with certainty or that clinical details were insufficient.

Diaschisis.—The Effect upon the Dominant Speech-centres of Damage to the Subsidiary Speech-centres.—An absolute distinction must be drawn between those defects of speech which are only seen soon after a lesion has occurred, are slight in degree, and soon pass off, and those defects which are well marked and permanent. The nature of these transitory symptoms is often such as to cause difficulty in the regional diagnosis of the lesion or in forming a judgment of its extent, in that they may suggest damage to centres at a distance from the actual site of the lesion. The speech-mechanism acts as a whole, and not in terms of its individual elements, and these early and transitory symptoms are referable to a lowering of functional activity, in a more or less distant part of the speech-mechanism, due to disturbance of balance between the several parts of this mechanism, which the destruction of one of its integral parts causes. For example, the slight and transient alexia (word-blindness), which is so commonly met with in cases of Broca's aphasia, is explained as the result of damage to the speech-mechanism as a whole, causing lowering of function and temporary loss of the least impressed function, namely, visual speech. To this phenomenon von Monakow has applied the term "diaschisis." Some of the slight and transient speech-disorders which result from lesions of the right cerebral hemisphere in right-handed persons are explicable in this manner as due to destruction of the subsidiary speech-centres in that hemisphere. The transient alexia, agraphia, and amnesia occurring from this cause after lesions of Broca's region led Marie to assume that lesions of Wernicke's zone were invariably present in cases of Broca's aphasia, and it is only just to Marie to admit that many cases have been reported as examples of Broca's aphasia in which such lesions did actually exist.

The Relation of Aphasia and Apraxia.—The posterior parts of the three frontal convolutions of the left hemisphere form an area from which the more specialised subjectively purposive movements of both sides of the body are incited, and destruction of any part of this region is associated with an inability to perform such movements in the corresponding part of the body, while the ability to perform common movements is preserved. To this region the general name "eupraxic centre" has been applied by Liepmann, and for the phenomena which result when any part of this region is damaged the term "apraxia" is used. It is clear, then, that aphasia is apraxia of the tongue, lips, larynx, and palate, and that agraphia is a form of apraxia of the upper extremity, and that these apraxias of speech have the same relation, as consequences, with the agnosias of speech (word-blindness and word-deafness) as have the limb apraxias with the corresponding agnosias of movement. The eupraxic centre will correspond with the higher kinaesthetic region, the annex of the lower kinaesthetic region (the so-called "motor cortex"), which is situated immediately behind it, and which is confined to the ascending frontal gyrus. The regional localisation of function in the eupraxic centre and in the ascending frontal gyrus is the same, and corresponds from below upwards with the higher and with the common movements respectively of the face, upper extremities, trunk, and lower extremities. The outgoing path from the eupraxic centres is by subsulcine fibres to the ascending frontal convolution and thence by the pyramidal tract. Just as aphasia may be a result of transcortical, cortical, and subcortical lesions, so also may apraxia be produced.

The Views of Pierre Marie.—In 1906 Pierre Marie published a hypothesis of the nature of the speech-mechanism which was highly antagonistic to the majority of existing views upon the subject (62). He challenges the hypothesis that speech-memories are registered in the cerebral cortex as "visual word-" memories, "auditory word-" memories, and "kinaesthetic word-" memories. He denies the existence of a visual word-centre, and, indeed, of any centre for visual speech situated in the angular gyrus or supramarginal convolution. He denies the narrow localisation of Wernicke's centre in the posterior two-thirds of the second temporal gyrus. He contends that the left third frontal gyrus has no connexion whatever with the speech-functions. He admits of no distinction between the so-called "motor" and "sensory" cortical aphasias. He argues that there is only one speech-centre diffusely localised in the left temporo-occipital region, and that this centre is a region of intelligence specialised for language and not a centre for the storing of sensory images. The so-called "word-blindness" and "word-deafness" are defects of a special speech-intelligence, and are not due to the loss of function in centres in which sensory images of words are represented. He contends that there is but one form of aphasia from a lesion of the cerebral cortex, and that this corresponds with the aphasia of Wernicke (verbal agnosia or "sensory aphasia"), which results from destruction of the region of intelligence specialised for language and situated in Wernicke's zone; and that according to the degree of the depression of function produced by the lesion, the special functions of the region are lost in the reverse order of their depth of impression. The classical aphasia of Broca is a combination of aphasia with "anarthria," and is the result always of a double lesion, the one situated in the region of Wernicke, determining the aphasia, and a second lesion within a quadrilateral area around the lenticular nucleus determining the anarthria. The limits of the quadrilateral region, a lesion of which produces anarthria, are, anteriorly and posteriorly, vertical frontal planes level with the anterior and posterior limiting sulci of the insula respectively; its outer limit is the surface of the insula, its inner limit is the wall of the lateral ventricle; above, the quadrilateral is prolonged towards the overlying gyri of the convexity, whilst below it is lost in the subthalamic region. The "quadrilateral" is in direct connexion behind with Wernicke's zone by means of the temporo-parietal isthmus. Lesions situated in front of this isthmus produce anarthria; those situated behind the isthmus cause aphasia. Marie's views have given rise to much criticism and controversy, and they have received little or no support from the many and careful investigations which have followed from this controversy. The grounds for Marie's assumptions were briefly these: having examined the specimens (which are still preserved) from which Broca made his great discovery of the functions of the left third frontal gyrus, he proved that the lesions present were not confined to that region, and that in several instances Broca had determined the limits of the lesions from an examination of the surface of the brain only. (That the great discoverer based his deductions upon insecure ground does not justify Marie's assertion that his conclusions were incorrect.) In the second place, very few cases of destruction confined to the left third frontal gyrus have been reported, whereas the vast majority of the cases published as cases of Broca's aphasia have shewn lesions extending beyond this region, and especially into the area named by Marie the "quadrilateral." (From the very nature of the lesions producing aphasia, this same criticism may be applied to all attempts at cerebral localisation from the results of pathological lesions, and the argument therefore loses all weight when directed against the localisation of function in the left third frontal gyrus only.) Thirdly, the large number of reported cases in which destruction of the left third frontal convolution has occurred without aphasia, or in which aphasia has existed without damage to Broca's area. Lastly, the absence of pathological and clinical evidence of the existence of a visual word-centre, apart from the auditory word-centre.

It is interesting that, at the time when Marie was endeavouring to dismiss the left third frontal gyrus from participation in the speech, function, strong evidence was being collected by Liepmann and his followers that the first and second frontal convolutions of the left side were concerned with a similar function as regards limb movements as was Broca's region with movements of the mouth, tongue, and other parts, and that the posterior parts of the three left frontal gyri should be grouped together as the eupraxic centre for the subjectively purposive movements of the whole body. The evidence that has since been brought forward by Dejerine (26, 27, 28) and others is overwhelming for the attribution of its classical function to Broca's centre. Marie has employed the term "anarthria" in a sense that is quite contrary to its common usage, for with him it is equivalent to "subcortical aphasia." In putting forward the "quadrilateral" as part of the essential speech-mechanism, Marie shews us a vast region in no part of which does he attempt to locate any definite function. In this region are contained the path connecting Broca's centre with the auditory word-centre and the greater part, if not the whole, of the projection of the left third frontal gyrus, and lesion of either of these paths is admittedly productive of aphasia by isolation of Broca's region upon the ingoing and upon the outgoing sides respectively, and it is in the region of these very paths that Marie shews that lesion of his "quadrilateral" is most often productive of aphasia, in his attempt to locate an executive speech-centre in the "quadrilateral" to the exclusion of Broca's centre. There is, in fact, no evidence whatever in favour of the existence of any speech-centre in the "quadrilateral."

Marie's conception of a single speech-centre in the temporo-occipital region corresponding to Wernicke's centre is admissible in so far as it is probable that the narrow limitation of the auditory speech-centre hitherto accepted is too restricted, and inasmuch as it is probable that the visual word-centre is actually situated in part within the limits occupied by the auditory word-centre. His contention that this is a centre of intelligence specialised for speech and not a centre for the storing of sensory images, upon close examination resolves itself into a question of terminology. For, whatever he may say, the speech-memories exist and are located in this centre, and so long as we realise that words are integral parts of percepts and of concepts, and are, in fact, the counters by means of which thought is carried on, it is clear that damage to this region must entail impaired intelligence as well as special defects of speech.

CLASSIFICATION OF SPEECH-DEFECTS.—In the study of speech-defects it is necessary to consider the effects of lesions in the following situations: (*a*) in the lower motor centres of the medulla which are concerned with the actual production of speech; (*b*) in the pyramidal fibres connecting these centres with the lower kinaesthetic region (so-called motor cortex); (*c*) in the commissural path connecting the eupraxic centres with the region of origin of the pyramidal tracts; (*d*) in the different kinds of word-centres; and (*e*) in the different commissures by which these centres are connected one with another.

Our subject will therefore be dealt with under the following main divisions: (i.) lesions of the bulbar speech-centres; (ii.) lesions of the pyramidal system supplying these centres; (iii.) subcortical lesions, interrupting the outgoing path from the glosso-kinaesthetic and cheiro-kinaesthetic (eupraxic) centres, to the pyramidal centres; (iv.) lesions of the glosso-kinaesthetic and cheiro-kinaesthetic centres; (v.) lesions of the auditory and of the visual word-centres; (vi.) lesions of the commissures between the word-centres.

I. DEFECTS DUE TO LESIONS OF THE BULBAR SPEECH-MECHANISM. — DYSARTHRIA. — Disorders of articulation are known by the name "dysarthria," and when as a result of articulatory defects speaking becomes impossible or unintelligible, the condition is termed "anarthria." In these conditions there is no defect of the cerebral speech-functions. There is complete comprehension of spoken and of written speech, and the power of communicating the thoughts by writing is unimpaired, provided that the upper limbs are not paralysed, and the power of expressing thought in speech is hindered only in proportion as the utterance is intelligible or not. The causes of dysarthria are numerous. It may be due to local lesions of the buccal cavity which interfere with the correct working of the articulatory mechanism, such as cleft palate, hare-lip, and tumours. It occurs in intrinsic pathological conditions of the muscles of articulation, as in the facial forms of myopathy. Lesions of the trigeminal, facial, glossopharyngeal, and hypoglossal nerves may produce dysarthria, especially if any of these nerves are involved upon both sides. Diseases of the medulla oblongata which implicate the lower motor neurons concerned in articulation are always connected with difficulties of articulation, and the most important of these diseases are the bulbar form of progressive muscular atrophy, the various forms of acute bulbar disease, and myasthenia gravis. In the latter disease the clinical aspect of the dysarthria is peculiar in that it is apt to come and go, and to make its appearance rapidly upon prolonged exertion of the articulatory muscles. Affections of the ponto-cerebellar region often produce dysarthria of a peculiar type, which is in reality an ataxia of articulation due to interference with the co-ordinating mechanism of articulation. This form occurs especially in disseminated sclerosis, Friedreich's disease, and local lesions of the pons. The dysarthria sometimes present in chorea is probably of the nature of a cerebral ataxia, in addition to the embarrassment of utterance caused by the occurrence of the spontaneous involuntary movements in the lips, tongue, and muscles of respiration. General paralysis of the insane causes a dysarthria of a characteristic type, in the production of which both cortical cerebral ataxia and cerebellar ataxia are probable factors.

A spastic condition of the muscles of articulation analogous to the

KING'S COLLEGE HOSPITAL
MEDICAL SCHOOL.

condition of the limb muscles in spastic paraplegia and due to the same cause, which is the destruction of the pyramidal fibres concerned with the innervation of the corresponding lower motor neurons, produces a spastic dysarthria. Since the muscles of articulation are bilaterally associated muscles, and are therefore innervated throughout by fibres of the pyramidal projection of both hemispheres, it follows that the spastic and paretic condition producing the form of dysarthria under consideration does not result, except as a transitory phenomenon, from a lesion, however extensive, of one pyramidal tract alone, but it is brought about when bilateral lesions of the pyramidal systems occur. "Spastic anarthria," "pseudo-bulbar paralysis," or "supra-nuclear bulbar paralysis," as it is indifferently named, is therefore met with as a result (1) of bilateral lesions of the cerebral hemispheres which involve the lower end of the ascending frontal gyrus, or which intercept the pyramidal fibres issuing thence in any part of their course; and (2) single focal lesions in the ventral and mesial regions of the brain-stem where the two pyramidal tracts are in close contiguity and are implicated together (*vide* also p. 106). It follows that the syndrome of bilateral hemiplegia is the usual clinical association of spastic dysarthria.

Other speech-defects of the dysarthric type are due to functional disorders rather than to structural defects of the executive speech-mechanism, and the precise localisation of the disorders is far from certain. They are in all probability cerebral disorders. Of these there are four chief varieties, namely: (*a*) lalling; (*b*) stuttering; (*c*) idioglossia; and (*d*) aphthongia.

(*a*) *Lalling* is the term used to characterise the speech of children before they have learned to pronounce their words so as to be intelligible to all persons. It is a defect due to want of precision in the oral articulatory mechanism. In a healthy child, who is properly taught and who shews an adequate amount of attention, this lack of precision soon disappears, but where the attention and the training are defective it may last long beyond the usual age. In children whose brains and intelligence are defective this imperfect articulation may never disappear. As a temporary defect lalling occurs in different degrees of alcoholic intoxication.

(*b*) *Stuttering* and (*c*) *idioglossia* are described in a separate article (see page 569).

Aphthongia is a very rare affection of the articulatory mechanism which seems to have relation, on the one hand, to the spasmodic craft-neuroses and, on the other hand, to stuttering. Only a few cases have been recorded. According to Fleury, who first named and described the condition, its most striking feature consists of "cramps in the territory of distribution of the hypoglossal nerves, which come on whenever an attempt to speak is made, and render articulate expression impossible." The condition has generally shewn itself after great mental excitement, and on this occasion was due to an operation for excision of the tonsils. The spasm is sometimes limited to the intrinsic muscles of the tongue.

In Panthel's case the hyoid muscles and the extrinsic muscles of the larynx were also involved. In Ball's case, when the patient attempted to speak there was a spasmodic contraction of the muscles of the tongue, which caused that organ to assume a dome-like shape and to be closely applied to the roof of the mouth.

It is unnecessary to dwell upon the majority of the dysarthric defects here enumerated, because a reference to their several characters will be found in the descriptions of the different diseases. The following groups of dysarthrias, however, require further description in this place.

The Dysarthria occurring in certain General Diseases of the Nervous System.—The diseases, disseminated sclerosis, general paralysis of the insane, Friedreich's disease and its sub-variety Marie's ataxia, are often characterised by dysarthria of a peculiar type, and on account of its similarity we may class with this the dysarthria which may be met with in cases of local disease of the pons and of the cerebellum. The peculiarities of the articulation are not always the same in any one of these diseases. In disseminated sclerosis, for example, several types of speech peculiarity occur, each highly characteristic of the disease and upon superficial consideration unlike.

The dysarthrias here classed together are made up of all or several of the following fundamental defects in varying combinations: (1) slurring; (2) syllable elision; (3) syllable stumbling (reduplication of syllables); (4) undue separation of syllables and words (scanning); and (5) festination. The first four of these primary defects are self-descriptive. Festination is the hurrying of articulation towards the end of a sentence or clause or polysyllabic word without syllable elision. The analysis of the dysarthria, into the fundamental defects of which it is made up, is easily accomplished by directing the patient to repeat certain test words or phrases, chosen because their pronunciation brings these defects into especial prominence. A few of these are here cited, which serve every purpose as tests for these varieties of dysarthria: (*a*) "hippopotamus"; (*b*) "mutual eligibility"; (*c*) "West Register Street"; and (*d*) "around the rugged rocks the ragged rascals ran." These test words and phrases do not present the slightest difficulty of articulation to the normal person, but if there be the least tendency to the above-mentioned fundamental defects, their repetition will at once make such defects manifest, even though the patient present no obvious defects in ordinary conversation. For example, a patient presenting dysarthria in disseminated sclerosis will articulate the words "mutual eligibility" as "mewsh-ledg-bil-t-t-ty." Here the slurring and syllable elision, the undue separation of syllables, and the syllable stumbling all become evident in the articulation of this one phrase. The characteristic speech of disseminated sclerosis presents all or several of these fundamental defects in varying degrees of combination. Further than this it cannot be said that there is any one form of speech which is typical of the disease. There are several different forms of speech all highly characteristic of disseminated sclerosis, or, to be more correct, highly characteristic of scattered lesions of any nature in the ponto-cerebellar region. The difference in these forms of speech is determined by the varying combinations of the fundamental defects. Thus, there is the "scanning" form of speech, which may be either "staccato" or "drawling," and in which undue separation of the syllables is the most noticeable defect. Very different from this is a rapid "slithering" speech, in which slurring and syllable elision are of frequent occurrence; different again is the "festinating" speech, in which a hurrying of the syllables occurs towards the end of a phrase. Intention-tremor of the muscles concerned in articulation, or intention-tremor of the trunk, causing a more or less regular interruption in the blast of air and therefore in the voice, may produce peculiarities of speech which are too obvious to need special description. Most cases of disseminated sclerosis with dysarthria present intention-tremor, and it is probable that both of these phenomena are of the same order and are due to a failure of the co-ordinating mechanism. Further, it is clear that these symptoms only appear when patches of sclerosis are present in the ponto-cerebellar region taken in its widest sense (inclusive of the cerebellar peduncles in their whole extent and inclusive of the red nucleus). The occurrence of similar forms of dysarthria in connexion with general disease of the ponto-cerebellar structures or in connexion with scattered lesions of this region other than disseminated sclerosis is thus explained. Upon similar grounds we can account for the slow scanning dysarthria usually met with in Friedreich's disease and allied conditions, which often resembles closely that of disseminated sclerosis. The dysarthria in general paralysis of the insane is described under that heading (p. 370).

II. DEFECTS DUE TO LESIONS OF THE PYRAMIDAL SYSTEM.—SPASTIC DYSARTHRIA OR SUPRA-NUCLEAR BULBAR PARALYSIS.—The name "pseudo-bulbar paralysis," under which this condition was first described and by which it has long been known, is unfortunate. It was so called in contra-distinction to atrophic bulbar paralysis (progressive muscular atrophy of the muscles supplied by the bulb). In the first place, it is a true paralysis of the muscles supplied by the bulb; and secondly, it bears no more resemblance to, nor relation to, atrophic bulbar paralysis than does hemiplegia to anterior poliomyelitis, the only thing common to the two diseases being the paralysis. In fact, the relation between these diseases in each case is anatomically identical, since they are due to destruction of the pyramidal neurons, and of the lower motor neurons respectively.

The pyramidal fibres which subserve articulation pass out from the lower part of the ascending frontal gyrus in each hemisphere. The fibres on the left side are associated with the contiguous eupraxic speech-centre (Broca's centre) by a system of subsulcine fibres which loop under the lower end of the vertical frontal sulcus. Those of the right side are associated with Broca's centre by fibres which cross in the anterior portion of the corpus callosum. From the lower ends of the ascending frontal gyri these fibres descend through the corona radiata to the internal capsule, where they form a bundle occupying the knee of the capsule called the "geniculate fascicle." In the region of the crura cerebri, at the upper part of the mesencephalon, most of these fibres leave the pyramidal tract, and, coursing upwards and backwards, run with the fibres of the fillet to the reticular formation in the dorsal part of the medulla, where they break up in the vicinity of the motor nuclei. Other fibres leave the pyramidal tract in the lower part of the medulla, and proceed upwards and forwards to the reticular formation in a definite round bundle called "Pick's bundle." In the medulla, as in the spinal cord, the pyramidal fibres have never been traced actually to the motor nuclei, and it must be presumed, therefore, that they are connected therewith by a mediate path.

Causation.—It has been already stated that, on account of the innervation of the bilaterally associated muscles of speech from both cerebral hemispheres, a spastic paralysis of these muscles, akin to the spastic paralysis of the limbs from a lesion of the pyramidal tract, can only result from a double lesion in the two hemispheres or from a single lesion damaging both pyramidal tracts where these are contiguous. General and symmetrical cerebral sclerosis and atrophy entail bilateral pyramidal destruction, and therefore spastic dysarthria is commonly met with in cases of cerebral diplegia. Sometimes it occurs in old people as the result of the atrophy occasioned by widespread cerebral arteriosclerosis. Much more commonly it is the result of two separate focal vascular lesions, each involving one of the pyramidal systems at some part of its course. A history of two separate "strokes," affecting first one and then the other side of the body, and a condition of double hemiplegia are therefore the common clinical associates of spastic dysarthria from this cause. Lesions of the brain-stem which interfere with the pyramidal fibres for speech on both sides always produce spastic anarthria, and these are commonly vascular lesions and tumours involving the reticular formation (*vide* also p. 106).

The transient dysarthria commonly seen in left hemiplegia, and in cases of right hemiplegia due to capsular lesions, is certainly in many cases of the nature of spastic dysarthria. The sudden cutting off of one-half of a bilateral innervation may temporarily, until the other half has had time to compensate for the loss, cause spastic paresis even in the most closely associated bilateral couple. Another point to be taken into consideration in this connexion is that the amount of innervation from the two hemispheres for a bilaterally associated act is in some persons very unequal. This will account for the case in which, as a result of a lesion of one hemisphere (usually the left), dysarthria is much more severe and more persistent than is usual.

The extreme infrequence of complete anarthria as the result of lesions of both pyramidal systems requires some explanation. In the first place, the same holds good for complete double hemiplegia. The consecutive lesions of the two cerebral hemispheres, which so commonly account both for spastic dysarthria and for double hemiplegia, are rarely so placed as to interrupt all the pyramidal projection concerned, and the time that elapses between the occurrence of the consecutive lesions probably allows some recovery to occur in the neighbourhood of the initial lesion, and possibly this lapse of time may admit of the opening of a fresh path, however inadequate, by which incitations may reach the bulb. Severe degrees of dysarthria are more often seen in disease of the brain-stem, but here again complete anarthria is uncommon. Lesions in this situation, of such an extent and nature as to sever completely the connexions between the motor tracts and the medulla, do not allow of the survival of the patient for any length of time. In this situation, too, the opening up of an alternative path when the pyramidal tract is destroyed is not only possible, but highly probable, for it is known that the descending systems of fibres from the mid-brain take up the function of the motor tract in all animals below mammals and in some mammals.

Symptoms.—In spastic dysarthria the muscles of articulation are, according to the severity of the affection, stiff and contractured, weak and slow to contract, and slower still to relax when they have contracted. The speech-defect is such as may be deduced from the condition of the muscles, and its severity may vary from a slight slurring and slowness of articulation to the utterance of a succession of hoarse, unintelligible, laryngeal noises. In a well-marked case the tongue is small from contracture, and does not duly fill the lower dental hoop; it is symmetrical and its surface is smooth, and when taken between the fingers it feels unusually firm. When protruded, it is long, narrow, and pointed. The palate is stiff, and acts little on phonation, but the palatal and pharyngeal reflexes are much exaggerated. The face is often stiff and expressionless. The muscles of the jaw are stiff, and it is moved slowly and but little when the patient is speaking. The articulation is slow and slurring, and there is a tendency for the blast to be incompletely cut off at all the stop positions. Consequently the patient seems to use his voice too much and his lips and tongue too little, and there is loss of all the precisions of articulation. From the stiffness and difficulty in moving the larynx as a whole the voice is monotonous. In the more severe cases the moaning monotonous speech, with its slurring articulation and uninterrupted blast, comes to resemble very closely the speech of atrophic bulbar paralysis. From the stiffness and slow action of the pharyngeal muscles some dysphagia is always associated with spastic dysarthria.

Diagnosis.—Spastic dysarthria can hardly be confused with atrophic bulbar paralysis, the stiff small tongue with preserved faradic excitability of the former condition contrasting strongly with the crinkled, wasted, soft tongue with lowered or lost faradic excitability of the latter condition. From aphasia and subcortical or pure aphasia, dysarthria is distinguished by the fact that the patient's utterance (so far as it goes) is consistently and uniformly bad in the latter condition, whereas in the former conditions, if the patient can say any words at all, they are well articulated. Further, when commanded to speak, the dysarthric patient at once makes the attempt, whereas the aphasic patient often does not do so. The local signs of spasticity in the articulatory muscles characteristic of spastic anarthria are never present in uncomplicated aphasia, and will serve to prevent error.

The distinction between complete anarthria and subcortical aphasia may be very difficult, more especially soon after the occurrence of the lesion; for in both conditions there is complete speechlessness, with perfect possession of speech-understanding, and there may be perfect ability to express thought in writing. In such cases a history of two separate attacks suggesting lesions in both hemispheres, or the presence of paralysis of the common movements of the lips, tongue, pharynx, etc., or the presence of any signs of local disease of the brain-stem will be the conclusive evidences of anarthria.

Prognosis.—The duration and the persistence of the dysarthric defects under consideration will depend upon several conditions, and foremost among these conditions are the nature, extent, and position of the lesion or lesions. With unilateral cerebral lesions it is usually rapidly transitory, but it is apt to be more persistent in left-sided lesions than in lesions of the right hemisphere; and this seems to correspond with a usual major innervation of the bulbar speech-mechanism from the left hemisphere. The cases of persistent anarthria, that have been described as resulting from a single lesion of the upper part of the left internal capsule and close to the corpus striatum, are open to the criticism that the extent of the lesion was not determined microscopically, and that they were probably cases of subcortical aphasia from an extension of the lesion into the subcortex of Broca's region. This explanation is the more probable since most of the reported cases have been in elderly people in whom vascular obstruction often causes lesions which are little apparent to the unaided eye.

Much will depend upon whether the pyramidal fibres for speech are actually destroyed or only temporarily disabled. When they are only pressed upon by extravasated blood or interfered with by the partial evascularisation of an area, the circulation of which becomes subsequently re-established, the dysarthria may be transient; whereas no such speedy disappearance of the speech-trouble is possible when these fibres are actually torn across or destroyed.

III. DEFECTS RESULTING FROM SUBCORTICAL LESIONS WHICH INTERRUPT THE CONNEXIONS BETWEEN THE EUPRAXIC CENTRES AND THE CORRESPONDING CENTRES AT THE FOOT OF THE ASCENDING FRONTAL GYRUS: SUBCORTICAL APHASIA, PURE APHASIA OR APHEMIA, AND AGRAPHIA.—The outgoing path from the left third frontal convolution by which the speech incitations reach the motor nuclei in the medulla oblongata is an interrupted or mediate path. The first link of this mediate path is formed by fibres which arise in Broca's convolution and loop through the white matter underlying that gyrus to end in the lower part of the ascending frontal gyrus. They are the subsulcine fibres connecting the higher kinaesthetic centre for the movements of tongue, lips, and so forth, with the lower kinaesthetic centre from which the pyramidal fibres subserving speech have their origin. The second link in the mediate path consists in the pyramidal fibres which arise in the lower part of the ascending frontal gyrus and form the geniculate bundle of the internal capsule. Destruction of the lower link of this path produces the group of speech-defects known as dysarthria, as has already been shewn. Destruction of the upper link of this path, on the other hand, entails defects of speech which are essentially of a different order, and to these defects the name "aphemia" or "subcortical aphasia" is given. There is, however, an alternative outward path from Broca's centre, since a system of fibres arising in this centre, crosses in the anterior part of the corpus callosum to the right third frontal convolution which is connected with the bulbar nuclei by a mediate path similar to that above described. And it is probably the destruction of this first part of this path, together with that of the direct path, that is essential for the production of subcortical aphasia as I formerly suggested. A subcortical lesion confined to the white matter underlying the left third frontal gyrus, and not too far distant from the surface, will, according to its extent, interrupt more or less completely both the paths connecting this centre with the bulbar speech-mechanism, and the centre will thus be more or less completely isolated upon the outgoing side. Since the cortical speech-centres are intact there will not be even a temporary interference with speech appreciation and with speech recall; the processes of thought and the mental capacity are not disturbed, and since the left second frontal gyrus and its connexions are intact, the patient is able to express himself perfectly in writing. Moreover, since the lower part of the ascending frontal gyrus and the pyramidal fibres issuing therefrom are intact, there will be no sign of spastic dysarthria and no interference with the common movements of the tongue, lips, and palate. But if the lesion be complete, the patient will be absolutely dumb; he will be unable to say the simplest word, such as "yes" and "no," and in this respect he differs remarkably from the patient with aphasia in whom such utterances are usually possible. Such is the condition which we term "complete aphemia," and it is distinguished from complete spastic dysarthria or "anarthria" in that the latter is accompanied both by spasticity and by loss of the common movements of the muscles concerned with articulation and phonation. When the lesion is incomplete, the incitations from Broca's centre have to pass outwards along a partially disorganised path, in which they must be interrupted to a greater or less extent, and consequently the pyramidal speech-mechanism is aroused in a disordered fashion, and the patient's utterance (so far as it goes) is consistently and uniformly bad. The words are deformed rather than inarticulate, and there is much less impairment of the modulation of the voice than in spastic dysarthria. When the utterance is intelligible, it is found that the words are always used appropriately, and the use of wrong words and of reiterating expressions does not occur. In some cases the intelligible speech is confined to a few monosyllables; but these are always used with significance, and are never used indiscriminately as replies or for purposes of interrogation, as occurs so often in cases of aphasia. The words which an aphasic can say are well produced and articulated, whilst those of the aphemic patient tend to be ill-produced and deformed.

Aphemia is clearly not a sensory defect—it is not a form of amnesia—because the subjects of it can revive words in all possible modes, and are therefore able to think and to express their thoughts with an unimpaired freedom by writing. If the aphemia be in any way incomplete, such a case can be easily discriminated from a case of aphasia by the fact that the aphemic patient (when bidden) will always at once make the attempt to pronounce some simple word or syllable (however poor the attempt may be), whereas the typical aphasic patient is unable to make any such attempt—he will not try to repeat even the simplest vowel sound. The reason of this important distinction lies entirely in the situation of the lesion in the two cases. In aphasia one of the most important word-centres for the expression of thought is affected, whilst in aphemia all the centres in which the memories of words can be revived are intact, the damage occurs beyond these, and there is consequently nothing to interfere with the flow of thought, and, in incomplete cases, nothing whatever to prevent attempts at articulation being made.

From our knowledge of the nature of subcortical lesions it is obvious that the succinct limitation above postulated is rarely exemplified. Usually the subcortical lesion extends beyond the limits of Broca's area, and it may involve (1) the pyramidal projection subserving speech, which issues from the lower part of the ascending frontal gyrus; (2) the pyramidal projection for the upper limb; and (3) the connexions of the second frontal gyrus which traverse the subcortex underlying this convolution; and in each instance the clinical picture of aphemia will be somewhat modified. In the first place the co-involvement of the pyramidal fibres for speech will add the signs of spastic dysarthria to those of aphemia and spasticity, and defects in the common movements of the tongue, etc., dysphonia and dysphagia will appear. And, since it is well known that the major innervation of the muscles concerned is from the left pyramidal system, these defects may be severe at first and only slowly transient. When the pyramidal tract for the upper limb is involved there will be paralysis and inability to write with that limb, but the relative ability to write with the left hand will be preserved. Lastly, if the subsulcine fibres which connect the second frontal gyrus with its corresponding pyramidal centre in the ascending frontal gyrus be severed, the writing centre will be isolated from its connexion with the right hand, and agraphia without paralysis of the common movement of the limb will result (right upper limb apraxia). And if, in addition, the fibres which connect the left second frontal gyrus with its fellow of the right hemisphere, via the corpus callosum, are cut across, the writing centre will be totally isolated upon the outgoing side, and bilateral agraphia (bilateral limb apraxia) will complicate the clinical picture of aphemia. It is at once apparent that this agraphia from limb apraxia is, in relation to writing, the phenomenon that corresponds to aphemia in relation to articulate speech. It is conceivable that a subcortical lesion situated beneath Broca's region may interrupt the direct outgoing path without damaging the fibres which cross from this centre to the third frontal gyrus of the opposite hemisphere, and that this alternative path would be available for the transmission of the outgoing speech-incitations. Such a condition of things would explain those cases of aphemia which, though severe at first, may shew rapid amelioration.

As regards the compensation, which may occur in or via the right hemisphere in cases of aphemia, certain facts stand out which are difficult to explain. In the cases of typical pure aphemia from lesions confined to the subcortex of Broca's region, which have been recorded by Dejerine (20) (two cases) and by Ladame, there was complete mutism in two of the cases and almost complete mutism in the other, whilst ability to write freely was preserved in all. It seems clear that in these cases both the direct and the alternative outgoing paths were cut across. But in the cases recorded by Pitres (79) and by Oulmont a similarly complete aphemic condition was produced by lesions at some distance from the third frontal convolution in the course of the geniculate fascicle and close to the corpus striatum. Now, since these lesions occurred in elderly people, and since their extent was not microscopically determined, it is just to presume that they extended upwards and destroyed the direct outgoing as well as the first part of the alternative path from the left third frontal gyrus. In all these cases a condition of complete, or almost complete, mutism obtained. On the other hand, in an aphasic patient, in whom the left third frontal gyrus is destroyed, there is often the constant distinct articulation of certain words or phrases, the production of which is commonly attributed to the activity of the right hemisphere. Why, then, it may be said, does the right hemisphere not come into play in many bad cases of aphemia? The only reply that I can make to this question is to suggest that when Broca's area is uninjured and the speech stimulus is able to pass therefrom through a varying length of its own path without much obstruction, the right hemisphere is not called upon, and the speech incitations continue to be sent along the old obstructed path. But when Broca's centre is destroyed any speech that can be produced must result from calls upon the right hemisphere, and for long can only be such familiar and well-worn utterances as "yes" and "no," or common expressions such as "nurse" and "oh dear."

The extent to which recovery may take place in cases of aphemia varies greatly. In severe cases, in which it is probable that a total interruption has occurred of the outgoing paths from the left third frontal gyrus, a permanent condition of complete, or almost complete, mutism results. In less severe cases, when the alternative path remains free for the passage of stimuli, or when the interruption of the outgoing fibres is incomplete, or when this interruption is temporary, such as a transient evascularisation may produce or the pressure of a clot bring about, varying degrees of recovery may occur. In some cases recovery is very rapid, but in only a few cases is this recovery complete, and it is probable that the latter result occurs only when there has been a physiological and not when there has been an anatomical interruption of the fibres. In the majority of cases of aphemia, therefore, some defects of utterance and of articulation persist.

The defects of writing corresponding with aphemia as a speech-defect have been recently brought into prominence by Liepmann and other writers who have investigated the subject of apraxia. The left second frontal gyrus is the eupraxic centre for both the upper limbs, and it has a similar function as regards writing to that of Broca's centre with regard to articulate speech. The anatomical connexions and the outgoing paths are similar, the incitations reaching the spinal centres for the right hand by a mediate path via the corresponding centres in the ascending frontal gyrus and the left pyramidal tract. Those for the left hand reach the spinal centres by fibres which arise in the left second frontal gyrus, and which cross in the anterior part of the corpus callosum to the corresponding gyrus in the right hemisphere. Therefore a subcortical lesion underlying the left second frontal gyrus, if it involve both these paths, will result in complete inability to write with either hand, and also inability to perform other subjectively purposive movements with either hand or bilateral apraxia, and there will be no paralysis of either hand in the ordinary sense of the term. If the direct path be alone involved there will be agraphia and apraxia of the right upper limb only, and the act of writing with the left hand will be unimpaired. If, on the other hand, the path crossing to the right hemisphere from the left second frontal gyrus is interrupted, as it may readily be by disease of the anterior part of the corpus callosum or by a lesion of the white centre on either side of this, agraphia and apraxia of the left hand will result, while ability to write with the right hand is preserved. It is thus clear that aphemia and the form of agraphia here described are examples of apraxia due to the isolation of the eupraxic centres for the organs of articulation and for the upper limbs respectively upon the outgoing side, and this isolation results from lesions of the subcortical white matter.

IV. DEFECTS OF SPEECH AND WRITING DUE TO DISABILITIES IN THE GLOSSO-KINAESTHETIC AND CHEIRO-KINAESTHETIC CENTRES.—**Defects due to Gross Lesions.**—These higher kinaesthetic centres are concerned more with the expressions of thought than with the thinking process. Their activity is in the main aroused when thought is about to be translated into action. Although with lesions limited to these regions the power of thinking may not be greatly impaired, still it is always interfered with to some extent, so that patients with such lesions do not exhibit anything like the mental clearness shewn by aphemic patients.

That speech and writing are so frequently involved together in cases of aphasia is partly due to the proximity of the two centres which are likely to be coincidently damaged by the same lesion, and perhaps no less to the proximity of the two sets of commissural fibres which connect these centres with the auditory and the visual word-centres respectively. Simultaneous damage to these commissures may, in fact, cause typical aphasia with agraphia not distinguishable in its clinical characteristics from the results of combined disease of the centres themselves.

Some of the earlier observers, such as Trousseau, Hughlings Jackson (41), and Gairdner, seemed to consider that inability to write was as much a result of a lesion of Broca's centre as inability to speak, and they contended that there was almost always a parity between these defects. Of late years these views have been adopted by Wernicke (93), Wyllie, Dejerine (26, 27, 28), and Mirallié, who go still farther in attaching an overweening importance to Broca's centre. They maintain that destruction of this region causes, in addition to agraphia, an interference with silent thought by hindering the revival of auditory word-images, and that it also entails alexia. Wernicke, Dejerine, and Mirallié even disbelieve in the existence of any cortical centre having the same relation to writing-movements as Broca's centre has to speech-movements, and Marie and Moutier consider that agraphia is simply a defect resulting from the lowering of intelligence.

These points require some separate consideration. (1) *Does destruction of Broca's centre alone entail verbal amnesia?* To this question we must give a decided negative answer, relying upon the reasons already given to shew that words are primarily revived in the auditory word-centre and not in Broca's centre, and upon the evidence of actual cases, to shew that where the lesion is limited to this region there is no sympathetic disturbance of the auditory centre or "diaschisis" sufficient to cause amnesia verbalis after the general cerebral depression that results at first from the occurrence of the lesion has subsided. The cases recorded by Banti, Dickinson, Wadham, and others (see my work, *Aphasia and other Speech Defects*, chap. vi.), and more recently by Leva, Kostenitsch, and Ballet, fully bear out this view, seeing that the ability of these patients to write freely and clearly proves that they could recall words. It has been already pointed out that destruction of any one of the speech-centres, since in the normal state the speech-centres act together as a whole, must cause some general lowering of the activity of the other centres, and it is to this cause that we must attribute the defects of intelligence that are met with in cases of Broca's aphasia as compared with the perfect preservation of intelligence in cases of aphemia. We admit that it may possibly happen that the severance of Broca's centre from the auditory and visual word-centres may so greatly impair the functional readiness of these latter centres as to hinder in some degree the spontaneous revival of the auditory and visual images of words. This, however, we believe to be the rare exception and not the rule. It has been shewn by Marie that in many of the cases which have been recorded as cases of Broca's aphasia from lesion of the left third frontal gyrus, and in which signs of word-deafness and word-blindness were present, that either (1) lesions of Wernicke's zone were obviously present, or (2) that the absence of any lesion of Wernicke's zone was not proved by any certain test. Further, it is important that in the cases of lesion confined to the third frontal convolution recently published by Dejerine (28) (two cases), Laignel-Lavastine and Salamon, in which the limits of the lesions were determined by serial microscopical sections throughout the brains, there was no sign of word-deafness, whilst signs of slight alexia were present in one case and absent in the others

(2) *Does destruction of Broca's region entail alexia?* Visual speech is one of the last of the speech-functions to be acquired, and therefore it tends to be the least impressed of these functions, and the depth of its impression in people who read but little and with difficulty must be but slight. It is presumable, therefore, that if in such people a general depression of the speech-function occurs from destruction of some part of the speech-centres not concerned with visual speech, this function would be the first to fail, the first to be lost, and the last to be regained.

In reading, a proper comprehension of the meaning of the text requires a conjoint revival of the words in the visual and in the auditory word-centres, but for this mere comprehension it is not necessary for the stimulus to pass on to Broca's centre as it must do in reading aloud. If, however, the way is open and this latter centre is in a healthy condition, it does commonly receive, in reading to oneself, a slight stimulus from the auditory word-centre, a fact which is often enough shewn by the occurrence of involuntary half-whispered mutterings when reading. Again the rousing of all three centres does give assistance to the comprehension of anything difficult, as is shewn by the common practice of reading aloud any passage the meaning of which is at all obscure. By this proceeding aid is obtained not alone by the full rousing of the glosso-kinaesthetic centre, but there is also the fullest activity of the auditory word-centre awakened by the stimulus of the spoken words. This kind of aid is indeed commonly needed, as Wernicke pointed out, by an uneducated person for the full comprehension of almost all that he reads (93). For the above reasons, therefore, it may easily happen that when such a person becomes aphasic from destruction of Broca's region, some degree of alexia may accompany the aphasia as a result of diaschisis. It would seem, however, that in some persons, and exceptionally, the comprehension of writing does not even require the associated activity of the auditory word-centre, so long as the visual word-centre is in a healthy condition and its other associational fibres are intact. This is proved by the fact that in a case recorded by Wernicke and Friedländer, in which there was aphasia associated with word-deafness (due to a lesion in Broca's area and also in the upper temporal gyrus), the patient was able to read well and even to write. There is also a valuable case of word-deafness (with lesions in both upper temporal convolutions) recorded by Mills, in which the patient was able to understand what she read; and a still more remarkable case recorded by Pick, with very similar lesions, in which, although the patient was word-deaf, he could write, and comprehended perfectly what he read. In many of the cases, in which there has been alexia coexisting with aphasia, there have, doubtless, been rather wide lesions, extending far beyond the third frontal gyrus and involving also the visual word-centre. Some of them may be cases in which there was originally word-deafness as well as word-blindness, but in which the former has disappeared early—which, as Lichtheim first pointed out, is the rule. Further, I have shewn that in some of the cases hemianaesthesia has been present, and this would tend to prove that in such cases we have to do either with a wide lesion extending from the third frontal convolution back to the posterior part of the internal capsule or else with a separate lesion in this situation; that is, in a region contiguous to the visual word-centre, which might, therefore, itself have been involved.

(3) *The question of the existence of a special centre for the registration and regulation of writing movements.* The case in respect to writing seems to be almost exactly comparable with that of speaking, and, indeed, with all other habitual voluntary movements whether complex or simple. Each set of movements must be associated with a set of ingoing kinaesthetic impressions which are registered in different parts of the cerebral cortex. This must be as inevitably true for writing movements as for speech movements; and just as re-excitation of Broca's centre under stimulation from the auditory word-centre is necessary for speech, so a re-excitation of a centre in which the impressions generated by the act of writing are registered, is, under stimulation from the visual word-centre, needful for the production of writing movements. The truth of this hypothesis does not in our opinion admit of any discussion, yet both Wernicke and Dejerine hold that the intervention of a special centre for writing movements is not necessary on the ground that writing consists of a simple copying of the visual images stored up in the visual centres.

It is, however, altogether another question whether the cortical centre in which the sensory impressions produced by writing movements are registered exists altogether apart from, or whether its structural elements are inextricably mixed with, other centres pertaining to less special movements of the hand and arm. We shall see later on that evidence is rapidly accumulating, and at the present time is almost conclusive, that there is a completely separate centre for the movements of writing and for other subjectively purposive movements of the upper limbs apart from the kinaesthetic centres for common movements or pyramidal centres in the ascending frontal gyrus.

One of the first to point out the inequalities that may be met with between the aphasia and the agraphia which result from lesions involving the left frontal convolutions was W. Ogle, to whom we owe the introduction of the term "agraphia." Since then many cases have been recorded which, though not conclusive, strongly support the argument of the existence of a separate centre for writing. These cases are of three classes: (1) cases in which lesions were confined to the left third frontal convolution, and in which severe aphasia was present while ability to express thought freely in writing was preserved; (2) cases in which lesions of the left frontal convolutions were present, and in which agraphia with some minor degree of aphasia existed; and (3) cases of aphemia without agraphia, from subcortical lesions. There was, until recently, no uncomplicated case of agraphia recorded, but Gordinier in 1899 reported a case of cerebral tumour involving the base of the left second frontal gyrus in which pure agraphia was present. This case seems to support Exner's localisation of the higher cheiro-kinaesthetic centre in the posterior part of the left second frontal gyrus. The recent researches of Liepmann and others upon the subject of apraxia have added much weight to the argument that the writing centre exists entirely apart from Broca's centre, since they have gone far to prove that the more highly specialised purposive movements of both sides of the body are registered in, and are incited from, centres situated in the posterior ends of the left frontal convolutions. Further, these researches, in conjunction with the recently determined fact that the pyramidal system takes its origin exclusively from the ascending frontal gyrus, seem to prove that the centres for more highly specialised movements (higher kinaesthetic or eupraxic centres) have a separate existence and situation from the centres for common movements (lower kinaesthetic centres), and that these higher kinaesthetic centres are situated immediately in front of the lower kinaesthetic centres of the left side. Dejerine's arguments (26) against the existence of a special centre for writing movements do not carry much weight. It cannot possibly be alleged as a valid argument against the existence of a cheiro-kinaesthetic centre in a man accustomed to write with his right hand, that this same man is able to write rudely on the sand with his foot, or that he can, after a fashion, write with his elbow or with a pencil placed between his teeth. Clearly, in these crude methods of writing, he would be dependent upon the activity of other parts of the general kinaesthetic centre. Further, the two well-known cases brought forward by Pitres (81, 82) in support of Exner's localisation of the writing centre in the second frontal, upon which Dejerine partly based his arguments, were not examined pathologically, and they admit of a different interpretation from that given by Pitres. These were cases in which there was inability to write spontaneously or from dictation, whilst ability to copy writing was preserved. "If the writing centre is destroyed," says Dejerine, "the possibility of executing any kind of writing should be abolished." This at first sight seems perfectly true, and it may be a valid criticism against Pitres for quoting these cases as instances of agraphia. But it is highly probable that these cases were not due to a destruction of the writing centre, since such a combination of symptoms without agnosia or amnesia may be easily explained by the cutting across of the path which transmits impressions from the auditory to the visual word-centre.

The Clinical Phenomena of Aphasia.—In the majority of cases there is some general mental impairment, as shewn by defective attention and initiative and by the readiness with which mental exertion produces fatigue, and in this respect aphasia contrasts strongly with aphemia in which no such psychical defects appear. The mental impairment is always slight, and it usually lessens as time elapses. There are, however, many instances on record in which the aphasia has been complete and associated with agraphia, and in which the mental powers have been fairly well preserved. Many of such patients have been able to read intelligently to themselves, and to play games perhaps better than their neighbours. Soon after the occurrence of the lesion, and when the general cerebral depression consequent upon the latter has passed off, the patient is for a time quite dumb. He comprehends everything that is said to him, and he is able to express himself correctly in gestures. There is usually some difficulty in the comprehension of written speech, and this slight alexia varies in degree and in duration. It is probably slighter and more rapidly transient the more accustomed the patient has been to read. The patient is able to recognise letters of the alphabet and single words, and can generally comprehend simple written sentences even from the first, but the sense of written phrases tends to escape him in proportion as they are long and complicated. The initial mutism is sometimes absolute, and it is then impossible to startle or provoke the patient into the utterance of any sound, cry, or interjection. In other cases the patient makes indistinct noises, or the utterance of a cry or of an interjection of displeasure is possible. Before long the patient regains the power of producing some simple articulate sounds and words, and even short phrases or oaths may appear under the influence of strong emotion. Such simple words are often used inappropriately, and the same word may be used in answer to every question and upon every attempt to speak. These recurring utterances may consist of a word or words, or of an unmeaning combination of syllables, such as "tan tan," which was the constant expression of one of Broca's patients, "cousisi," "pou tou ta ti," "macasa," "taratata." It has been suggested by Dr. Hughlings Jackson that some of the recurring utterances are the words that the patient was about to speak when the lesion occurred, or that they are the last words uttered immediately before the "stroke." Thus, a patient who told a cabman to drive her to "Mrs. Waters" became speechless a few moments afterwards, and had the recurring utterance, "Missis." The constant expression of a girl who was seized when riding a donkey was, "Gee gee"; that of a signalman, taken ill at his post, was, "Come on to me"; and that of a librarian was, "List complete." The articulation of the simple words and recurring utterances seems to be brought about by the intervention of the comparatively uneducated right third frontal convolution. As Dr. Hughlings Jackson originally pointed out, a patient is quite unable to repeat one of the words that he is continually bringing out when he is asked to do so, that is to say, he cannot speak the word in a purely volitional manner in response to a command.

When recovery is in progress the voluntary power to speak words comes back little by little, and there is more difficulty in the production of words used as substantives than in the articulation of the other parts of speech. A patient will often attempt to get over this difficulty by using a periphrase to express his meaning, and when shewn an object such as a pen, for example, he will say, after a few vain endeavours to find the word, "It is for writing with." Similarly, a patient who was to say "no," after one or two ineffectual attempts, said, "I can't say no, sir." Under the influence of powerful emotion or when moved by the elements of rhythm and of music, aphasic patients, and especially those who are recovering, can sometimes produce many words though they are unable to say one of them voluntarily. For instance, an aphasic patient summoned aid to a burning ward in which she was alone by shouting "Fire," and again such a patient may join in a familiar song, and sing every word of it correctly though unable to utter a single word voluntarily. During the process of recovery the words that are produced often shew apraxic defects, such as deformity, and wrong articulation and repetition (perseveration). As examples, a patient may say, "spastitié" for "pâtissier," "clab" for "cab," "glag" for "glad," "ventif" for "vendredi." Those who suffer from aphasia are always conscious of their errors in speech-production, and they thus contrast strongly with those whose defects of utterance result from amnesia and agnosia. Paraphasia and jargonaphasia do not occur in connexion with lesions confined to Broca's region, they are indicative of lesions of Wernicke's zone.

An interesting peculiarity is usually seen when resident foreigners become aphasic. During recovery it is found that they are at first only able to express themselves in that language in which they are most thoroughly versed, namely, in their native tongue. This is because a partial damage to a cortical centre will deprive that centre of its several functions in the inverse order of their depth of impression, and recovery of these functions will take place in direct order as they are deeply impressed. We have seen this in several patients. Two were Germans who had been long resident in England; yet, after an attack of right hemiplegia and aphasia, each of them was for a long time unable to say a word of English. When they began to speak they used German words only, and after they had further recovered, if in want of a word while speaking English, it was always a German equivalent which presented itself. An Italian, who had resided in France for a long time, subsequently for a few years in Germany and then in England, recovered his native tongue and his use of French rapidly and completely, while his German remained defective and his English still more so, as long as he was under observation.

From the close proximity of Broca's centre to the writing centre it follows that aphasia is almost invariably associated with agraphia, the case reported by Gordinier being the only recorded instance of uncomplicated agraphia from a lesion of the left second frontal gyrus. The relative degrees of the two conditions, however, are very unequal in some cases, as was first pointed out by Dr. W. Ogle. On the one hand, severe aphasia may coexist with slight agraphia, and, on the other hand, severe agraphia may be present with but slight aphasic defects. These conditions will be determined by the relative extent to which these two centres are involved by the lesion.

Since Broca's centre is situated close to the pyramidal centres and to the pyramidal projection, and since it is not far removed from the other eupraxic centres, it follows from the nature of the common cerebral lesions that aphasia is usually accompanied by right hemiplegia and not unfrequently by apraxia of the limbs.

Agraphia.—The phenomena which result from a lesion of the higher cheiro-kinaesthetic centre are in every respect comparable with the phenomena of aphasia, but they are less in evidence both to the patient and to the observer, since the right hand is generally paralysed. In such cases the condition of the writing faculty can only be judged by the medium of the unaccustomed left hand. In the majority of cases of aphasia and agraphia the latter is more severe and more persistent than the former, and there are two reasons why this should be so. In the first place the writing centre is the least important, the last to be organised, and usually by far the least educated of the speech-centres, and consequently the lowering of the functional activity of the speech-centres collectively, which results from the interference with one of its parts, will be most marked in connexion with the least impressed function—that of writing. Secondly, some degree of alexia is often present in aphasic patients who have been little accustomed to read, and this alexia usually entails agraphia. For these reasons, therefore, if a lesion partially destroys both Broca's centre and the writing centre to a similar extent, the resulting agraphia will be more severe and more persistent than will the aphasia.

The agraphic patient may be unable to form a single letter, or letters may be formed but combined wrongly, or there may be merely mistakes of letters in words that are otherwise correct. The defects in shape and in the regularity of the individual letters are sometimes remarkable. The patient can often sign his own name—which is almost an automatic performance—when he cannot express any proposition in writing. Perseveration or the re-energising of processes recently in activity is one of the commonest defects; the patient having written a letter or a word will write it over and over again. Writing to dictation is defective in the same degree as is spontaneous writing. When the agraphia is severe only servile copying is possible, and this is done slowly and clumsily. In partial cases the patient will usually copy print into cursive characters, and this ability may be much less defective than voluntary writing, since the most powerful arousal of the partly disabled centre occurs from the sensory stimuli of the writing which is copied. It must be borne in mind that agraphia is a particular variety of limb apraxia, and that it is likely to be associated with apraxia of movements other than those concerned in writing.

The distinction of the forms of agraphia already described from the agraphia occurring under other conditions is well shewn in the following table, in which are embodied conclusions the legitimacy of which will be subsequently shewn :—

AGRAPHIA

Complete	Uncomplicated.	1. Destruction of outgoing path from cheiro-kinaesthetic centre. (Subcortical agraphia.) 2. Destruction of cheiro-kinaesthetic centre. 3. Destruction of the visuo-kinaesthetic commissure.
	Complicated with word-blindness.	4. Destruction of the visual word-centre.
Partial	No spontaneous writing, nor from dictation, but can copy ; no word-deafness.	5. Destruction of the audito-visual commissure.
	No spontaneous writing, nor from dictation, but can copy ; word-deafness present.	6. Destruction of the auditory word-centre.
	Can write spontaneously or from dictation, but cannot copy ; word-blindness present.	7. Destruction of the visual word-centre in some educated auditives. 8. Isolation of the visual word-centre from all afferent connexions.

Prognosis.—The possibilities of recovery from aphasia and agraphia depend upon a number of circumstances : the nature and extent of the lesion ; whether the damage involves the whole of the centre or a part of it ; whether the damage is permanent or temporary only, as when a temporary ischaemia is followed by a re-establishment of the circulation, or when pressure is exerted upon the centres by some removable cause, such as a gumma or a blood-clot, are conditions which have an important bearing on the result. Again the capacity of the speech-centres in the right frontal lobes to compensate, and in this connexion the age of the patient also are of great importance. Such compensation seems to occur readily in early childhood and to vary greatly in different individuals in adult life, but in the degenerative period of life it occurs but little. The general condition of the cerebral blood-vessels and of the general circulation affects the prognosis, for, if there be universal disease of the cerebral arteries, or if the heart be failing, the probability of the re-establishment of any compensatory circulation upon the borders of a thrombosed area and a consequent reduction in the extent of a lesion is remote. It is probable that complete recovery takes place in two circumstances only : (*a*) in childhood when compensation occurs readily, and (*b*) when the lesion of the centre is of a temporary nature. There is no possibility of recovery after bilateral destruction of the frontal speech-centres. It is not rare in cases of aphasia for symptoms to be present which indicate the existence of lesions in the regions of the auditory and visual word-centres in addition to the frontal lesion, and such indications influence the prognosis adversely. Severity of the initial symptoms, and especially prolonged loss of consciousness, are unfavourable signs, since they indicate that the lesion is extensive. The shorter the interval between the ictus and the regaining of some power of speech, however little this may be, the better the outlook for recovery becomes, and the more rapidly the recovery progresses in the early days of the illness, the more perfect will it ultimately be. In very few cases, however, does speech become quite normal after a permanent lesion of the left frontal speech-centres.

Defects due to Functional Disabilities and Transitory Disturbances.—In association with many different conditions, aphasia is met with not infrequently as a temporary phenomenon, and sometimes with a marked tendency to recurrence, where the brief duration and the complete recovery make it improbable that it can have been due to any gross lesion. It is impossible to say definitely what are the proximate causal conditions in these cases; and in order to account for these defects we can only appeal to ischaemia from irritative congestion, temporary thromboses, minute embolisms, or spasm of vessels in the region of the speech-centres; or to "inhibition" due to various factors, or to some other little understood cause leading to a degradation of function in the same regions. These conditions persist for variable periods (hours, days, months, and even two or more years), and yet permit ultimately of a perfect restoration of function, more or less rapidly brought about.

The principal associated conditions may now be referred to in succession.

(*a*) *Irritative congestion or thrombosis* following upon excessive mental strain or as a result of exposure to cold. Trousseau (91) has recorded several interesting cases of this type, and some of them presented amnesia or paraphasia, as well as aphasia and agraphia, so that the disability must have affected the speech-centres generally.

(*b*) *Minute Embolisms.*—Nothnagel has called attention to cases of valvular disease of the heart in which aphasia suddenly occurred unattended by other symptoms, the loss of speech disappearing in the course of one or two days.

(*c*) *Spasm of Vessels: Intermittent Claudication.*—E. O. Daly has recorded a remarkable case of an elderly gentleman in whom recurring attacks of right hemiplegia and aphasia occurred, the duration of which varied from three minutes to three hours. A somewhat similar case has been recorded by Dr. W. Russell. Brissaud refers to a case of recurring aphasia with right hemiplegia in connexion with grave heart failure. At each relapse of the cardiac asystole there was a return of the paralysis and aphasia. Similar cases have occurred in association with migraine, Raynaud's disease, Stokes-Adams' disease, and in connexion with cardiac failure from arteriosclerosis.

(*d*) *Narcotic and other Poisons.*—Poisoning by stramonium, belladonna, cannabis indica, tobacco, opium, and snake-bite have not infrequently given rise to temporary aphasia and sometimes to verbal amnesia, as has also chronic lead poisoning. References to cases of this kind will be found in Bateman's work.

(*e*) *Auto-intoxication.*—Under this heading may be grouped the cases in which aphasia occurs as a temporary symptom in the course of certain specific fevers (especially enteric fever, but more rarely in typhus, small-pox, measles, yellow fever, and acute tuberculosis), in the puerperal state, diabetes, Bright's disease, gout, and general paralysis of the insane. The precise mode of production of the toxic aphasias included in the last two groups may possibly be, either the occurrence of small and perhaps temporary thromboses in the vessels of the centres at fault, or the transient ischaemia of the same centres which local arterial spasm may cause, or the direct action of the various poisons upon the nerve elements of the cortical centres affected.

In the following classes of temporary aphasia the pathogenesis is no less obscure; in some of them it seems to be due to "exhaustion" of nerve elements following upon epileptiform discharges; in others, to an even more mysterious process known as inhibition, to which we are obliged to appeal where explanation is difficult.

(*f*) *Before or after Epileptiform Convulsions.*—It is well known that in cases of local epileptiform convulsions, when the convulsion begins with sensations or twitchings in the right side of the mouth or in the tongue, aphasia may precede or follow for a brief period the occurrence of each of such fits. The so-called "congestive attacks" of general paralysis of the insane are often associated with convulsions, and the temporary aphasia that follows the attack and which has been above referred to as possibly of toxic origin may be in part also the result of post-convulsive exhaustion.

(*g*) *In Association with Insanity, Catalepsy, and Ecstasy.*—A condition of mutism is not at all uncommon in cases of chronic dementia or melancholia. It is often a lack of will to speak (sometimes under the influence of hallucinations or delusions) which leads to mutism, lasting, it may be, over a long series of years. In catalepsy and ecstasy the speechlessness is probably due to a mere suspension of volition. In all these cases, therefore, we have to do with what may best be called "pseudo-mutism."

(*h*) *From Fright and other powerful Emotions.*—Many cases both of aphasia and of mutism have been recorded as resulting from these causes. In the case of accident the importance of the mental shock far transcends that of the physical injury.

(*i*) *Reflex Inhibition.*—Bateman refers to three cases in which temporary loss of speech occurred in association with severe paroxysms of pain in trigeminal neuralgia.

(*j*) *By Hypnotic Suggestion.*—Charcot and others have been able to produce typical hysterical mutism in hysterical patients who proved capable of being hypnotised. The mutism thus produced in the hypnotic state was found to persist in the waking state unless proper measures were taken to terminate it.

(*k*) *Hysteria.*—The cases of temporary speechlessness that occur in hysteria belong almost solely to the type known as "hysterical mutism." This term was first used by Revilliod of Geneva for this condition, which up to that time had been included under the general designation of "functional aphasia." Its clinical features were subsequently made widely known by Charcot, and valuable series of cases have since been recorded by Cartez, Bock, and Nattier. Liouville and Debove's view that hysterical mutism is only a rarer and more severe form of a condition which is familiar enough as hysterical aphonia has been enforced by Prof. Wyllie, and is undoubtedly correct. This doctrine was also adopted by Charcot, who shewed that there are many features common to the two conditions. Thus, they occur in the same type of patients; they are produced by similar exciting causes, and are not infrequently found to alternate with one another in the same patient. Mutism is clearly the more severe condition of the two, as in it there is an absence of the power of whispering as well as of speaking aloud. The leading peculiarities of hysterical mutism are these. The onset is generally sudden, and often follows a fright or some other emotional disturbance. Sometimes it accompanies or follows an hysterical seizure with or without paralysis of the limbs. At other times it occurs without assignable cause, or it may be induced under hypnosis by suggestion. The subjects of this disability are completely mute, thus contrasting notably with ordinary aphasics since they do not use any recurring utterances or articulate sounds of any kind. The intellect seems unimpaired, and they are able freely to express their thoughts in writing. Though the common movements of the lips, tongue, and palate are preserved, these organs are unable to act in the particular combinations needful for speech movements, and in association with the other combinations of muscular action pertaining to the vocal mechanism. Some of the muscles of the larynx are often found to be paretic, though the particular muscles involved have varied in different cases. There is frequently more or less complete anaesthesia of the pharynx and of the larynx. Hysterical mutism may coexist with many other hysterical stigmata, but, on the other hand, it may be the sole manifestation of this neurosis. Recovery may occur suddenly, perhaps after a fit or after a recurrence of some strong emotion, or it may be gradually brought about by treatment; and in some of the latter cases there may be a stammering articulation for days or even weeks before speech is completely restored. Several cases have been rapidly cured by putting the patients under the influence of ether—during recovery from which they have begun to speak volubly—and this occurred in a case recorded by Jacob, after the dumbness had existed for five years. It is not uncommon for this mutism to recur on two or three occasions in the same patient, and in rare cases the attacks may occur repeatedly as in a patient who came under my care in the National Hospital, in whom frequent attacks occurred over a period of eighteen months. Delasiauve has recorded the case of a lady who for three years, at each menstrual period, was affected with mutism and partial paraplegia. A still more remarkable case has been recorded by Hun, in which frequently recurring attacks of mutism were generally associated with deafness or blindness—one or both of these disabilities often ceasing after an attack of hysterical convulsions.

Much discussion has taken place as to the pathogenesis of hysterical mutism. Charcot adopted the view of Marey that whispered speech is the product of the oral mechanism alone and that the larynx takes no part in its production. He thought, therefore, that aphonia was the result of a paresis of the adductor muscles of the larynx, and that hysterical mutism was due to the lack of ability to execute the proper specialised movements necessary for the articulation of words. Prof. Wyllie, following Sir Michael Foster, maintains that the larynx does take part in whispering, and that there can be no speech without the co-operation of both the oral and the laryngeal mechanisms. He believes that disabilities of either of these mechanisms may produce mutism, and he refers to several cases in Nattier's list in support of his opinion. The question as to what part of the nervous system we can presume to be at fault in cases of hysterical mutism and aphonia now remains to be considered. I formerly thought that these cases of mutism were examples of complete aphemia dependent upon a functional defect in the outgoing path from the left third frontal gyrus (10) since cases of subcortical defect, in which there was no damage to either of the word-centres, at that time seemed to me alone capable of producing the particular combination of symptoms met with in hysterical mutism. I always recognised that it would have been more satisfactory if one could have supposed that in hysterical mutism a region of the cortex itself was at fault. It is true that in some cases of complete aphemia due to organic lesions a mutism has been produced almost identical with that met with in hysterical cases. On the other hand, a very important case recorded by Guido Banti has convinced me that in some individuals a lesion narrowly confined to Broca's centre may neither affect the ability to write nor interfere with the general intelligence any more than does a subcortical lesion. And if such may be the case when an organic lesion is present, we may the more readily admit that a similar absence of agraphia and a similar preservation of intelligence will result from a functional disability affecting this region. This, in fact, was Charcot's explanation of the condition. He considered hysterical mutism to be an instance of pure motor aphasia from a functional disability of Broca's convolution. This, however, is by no means an adequate explanation.

The experiments of Sir F. Semon and Sir V. Horsley and those of Dr. Risien Russell have shewn that there is complete bilateral representation of the laryngeal movements in the cerebral cortex, so that destruction of the centre on one side will not produce paralysis of the muscles of the opposite side of the larynx, since these can still be called into activity by the intact centre in the opposite hemisphere (*vide* also Vol. IV. Part II.

p. 259). The position of the centre for the representation of the laryngeal movements was determined by Semon and Horsley to be the anterior part of the foot of the ascending frontal gyrus. If a similar bilateral representation of the laryngeal movements concerned with speech exists in man, it would follow that to account for hysterical aphonia, characterised by paresis of the laryngeal adductors, we must suppose that the corresponding centres in both hemispheres have been simultaneously affected as regards their functional activity. A bilateral representation of the laryngeal speech-movements in man cannot, however, be considered to follow from the fact that experiments upon the lower animals shew that the common movements of the larynx are bilaterally represented. We have shewn, moreover, that there is every reason for thinking that the highly specialised subjective movements of the larynx, such as occur in speech, aré represented in a centre apart from that in which the common movements are represented, the higher movements being represented in the eupraxic centre of the left hemisphere (Broca's centre), and the common movements in the lower part of the ascending frontal gyrus of both hemispheres. Charcot's explanation of hysterical mutism does not account in any way for the fundamental difference that exists between it and simple aphasia. The subject of the former affection is, as we have seen, absolutely mute, and has no recurring utterances of any kind. It is commonly supposed that in aphasia, due to a lesion of Broca's gyrus, the recurring utterances are brought about through the activity of the third frontal gyrus of the right hemisphere. The same thing would, therefore, doubtless occur in the functional affection if the left third frontal gyrus were alone involved, but as instead there is absolute dumbness, we are, I think, bound to assume that in this disorder there must be a simultaneous affection of the third frontal convolution and of the lower part of the ascending frontal gyrus in both hemispheres.

In this discussion we have not referred to the hypothesis of Prof. Wyllie as to the existence of certain subdivisions of what he calls Broca's region. Instead of limiting this region, as is customary, to the cap and foot of the third frontal gyrus, Prof. Wyllie seeks to include in it also the foot of the ascending frontal and the foot of the ascending parietal gyri. He suggests that in these two latter sites the "executive cortical motor mechanisms" concerned with speech are to be found—comprising a "centre for phonation" and a "centre for the oral articulative mechanism"; whilst in the foot of the third frontal gyrus are stored up "the guiding psycho-motor images for spoken speech," or, in other words, what we have termed "higher glosso-kinaesthetic impressions." These views have been accepted and further elaborated by Elder.

Now, as regards the impressions registered in the foot of the left third frontal gyrus, it seems clear that Prof. Wyllie's "guiding psycho-motor images" correspond with what we term "higher kinaesthetic impressions," and that these impressions which result from speech-movements must correspond partly with impressions from the oral and partly with impressions from the vocal subdivisions of the speech-mechanism. These, according to our view, are the guiding sense-impressions which (in conjunction with the auditory word-impressions) act upon and are capable of evoking the activity of the executive bulbar centres. We do not, therefore, believe in the existence of the "cortical executive motor mechanisms" which Wyllie and Elder assume to exist in the bases of the ascending frontal and ascending parietal gyri. These writers seem to have been guided too much by the results obtained from experiments upon monkeys and dogs, and not to have kept in mind that the movements of the larynx produced by stimulation of the cortex in these dumb animals could not have been such movements as are concerned with speech, though they might have been the reproductions of much less specialised movements of the vocal cords; and, again, that the movements of the tongue and lips similarly induced in these dumb animals could not afford any evidence of the site of the "oral articulative mechanism," although they would serve to determine the seat of registration in such animals of the impressions resulting from the common movements of these parts.

The very fact that such common movements are registered in one of these sites, namely, the ascending frontal gyrus (recent researches having beyond all doubt excluded the ascending parietal convolution from the so-called "motor cortex"), would of itself lead us to look elsewhere for the registration in man of the highly specialised movements concerned in speech; they might, for instance, be found on the contiguous foot of the third frontal gyrus. And, as Prof. Wyllie himself admits, the clinical evidence points to the importance of the foot of the third frontal gyrus, and not to the lower end of the ascending frontal gyrus, as the region most concerned with the production of speech. It ought to be almost superfluous to enforce the point that it is upon clinical evidence that we must alone rely, and not upon the conclusions of experimental physiologists drawn from experiments upon dumb animals, for the localisation of speech-centres. This view, which I put forward long ago, is confirmed in the light of recent researches. The limitation of the origin of the pyramidal tract to the ascending frontal gyrus and the discovery of the eupraxic centres, or centres for specialised subjectively purposive movements other than those concerned in spoken and written speech, in the posterior parts of the second and first frontal gyri of the left hemisphere, have answered the question which I raised in the first edition of this work as to the likelihood of the more specialised movements being represented in separate regions of the cortex from those in which the common movements of the same parts are registered. My former conception of the kinaesthetic centre must be modified by separation of portions of the common centre into those concerned with the more highly specialised movements, and those concerned with the common movements of the same part of the body. These higher kinaesthetic centres are contiguous to, and are situated immediately in front of, the corresponding lower kinaesthetic centres. The former are situated in the posterior parts of the three frontal gyri of the left side, and correspond with what have recently been called the eupraxic centres. The latter are situated in the ascending frontal gyrus, and correspond with the region of origin or of emission of the pyramidal tracts—the so-called "motor cortex." The recently ascertained fact that the pyramidal tract has its origin in the ascending frontal gyrus exclusively, and that the geniculate fascicle does not proceed directly from any part of Broca's convolution, as was formerly held, has necessitated an alteration in my former conception of the direct outgoing path from Broca's gyrus to the bulbar centres.

No real evidence is therefore forthcoming in favour of the hypothesis of Prof. Wyllie; and that the distinction I have dwelt upon between the common movements of the lips, tongue, and palate, and the common movements of the vocal cords, as contrasted with the highly specialised combinations of movements of these parts concerned in speech, is a perfectly valid one, is shewn by the fact that in hysterical mutism, though the latter movements are impossible, the common movements of the lips, tongue, and palate are unaffected—just as in hysterical aphonia the vocal cords can be perfectly approximated during the act of coughing, though they cannot be brought together as a component of the movements necessary for speech.

V. Defects of Speech and Writing due to Disabilities in the Auditory and Visual Word-centres.—Since the publication in the year 1874 of Wernicke's important memoir, the defects of speech due to lesions in the visual and auditory word-centres have been widely known as "sensory aphasia," in contradistinction to those defects produced by damage to Broca's centre, which, in accordance with then prevalent notions were, and still are, commonly spoken of as "motor aphasia." Since we hold that the latter is as much a sensory region of the brain as the former, this terminology is not in accordance with our views. The term "aphasia" is here restricted to the defects of speech produced by lesions in Broca's region, and the term "aphemia" to those dependent upon subcortical lesions, which isolate this region upon the outgoing side, leaving the speech-defects produced by lesions of Wernicke's zone to be grouped as so many varieties of agnosia and amnesia. Under these generic names many forms of speech-defects are included, due to defective recognition of visual and auditory word-stimuli or to defective recall of the visual and auditory images of words, and produced either by lesions in the visual and auditory word-centres themselves, or by lesions of the paths by which they are united to one another and to corresponding subsidiary centres in the opposite hemisphere. Whatever name we may apply to these cases, in each instance alike, the precise degree and nature of the defects must be arrived at by a systematic examination which will determine whether there is defective recall of words with loss of their auditory and visual images, or whether other combinations of symptoms are present which point either to partial isolation of these centres from one another or to their isolation from the general auditory and visual centres.

The nomenclature we have adopted involves a slight inconsistency, since aphasia and agraphia are, according to our views, forms of amnesia due to the non-revival of the higher kinaesthetic images. But we maintain that these kinaesthetic images play but a small part in the processes of thought, and that they are not subject to independent conscious recall as are the auditory and visual images of words which constitute our habitual thought-counters.

The nature of word-deafness and word-blindness was clearly recognised by me in 1869; and Wernicke, in 1874, determined the region of the brain at fault in word-deafness as the posterior half of the left upper temporal gyrus, and possibly part of the hinder end of the left middle temporal convolution. He concluded that the symptom-group resulting from a lesion in this situation was word-deafness, paraphasia, alexia, and agraphia. This conclusion was not founded upon a sufficiently wide clinical and pathological basis, and a wider experience and knowledge have necessitated its complete revision. The symptom-group which Wernicke named "sensory aphasia," and which he considered to be the unvarying result of a lesion in the region which bears his name, was shortly afterwards divided into two symptom-groups by Kussmaul, who contended that word-deafness was the primary and essential result of destruction of the hinder end of the left upper temporal gyrus, whereas word-blindness was due to destruction of the angular gyrus, and possibly also of parts of the supramarginal convolution. Kussmaul was, therefore, the first to suggest the localisation of a separate visual word-centre in the region of the angular gyrus. This doctrine of Kussmaul is now generally accepted, and both word-blindness and word-deafness are spoken of as varieties of "sensory aphasia." Wernicke's original opinion (92) as to the very complex results of a lesion of the auditory word-centre is still held by Dejerine and by Mirallié, who continue to speak of word-deafness, paraphasia, alexia, and agraphia as the results of such a lesion.

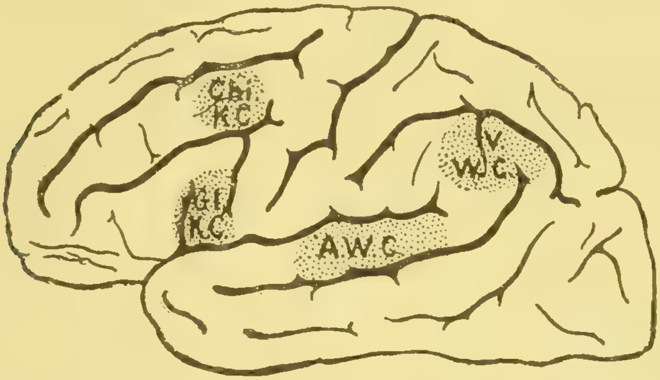

FIG. 22.—Diagram shewing the approximate sites of the four word-centres in the left cerebral hemisphere.

Now, while we hold strongly to the view that a lesion of the auditory word-centre is productive of one essential symptom-group only, namely, word-deafness and amnesia, and that a lesion of the visual word-centre similarly entails one essential symptom, namely, word-blindness, yet it must be freely admitted that word-deafness and word-blindness are the usual concomitant results of a lesion in the convolutions surrounding the posterior end of the Sylvian fissure of the left hemisphere, although this association of the two symptom-groups is by no means so universal as the statements of Wernicke and of Dejerine (25) would imply. There are many reasons for the common association of word-deafness with word-blindness, and these also explain the very well-marked clinical variations that are met with as results of lesions in Wernicke's region. The auditory and visual word-centres are situated close to one another, and are supplied by the terminal branches of the same artery, and therefore they are often coincidently involved in one lesion, though the relative amount of destruction in the two centres may vary greatly. Again, since a rapidly occurring lesion of the cerebral cortex always occasions a temporary depression in the functional activity of the surrounding regions of the cortex, a lesion in one of the two centres under consideration can hardly fail to produce a temporary loss of function in the other. We have shewn that the primary revival of words, both for speech and writing, takes place in the auditory centre in the majority of persons, and that the activity of the visual word-centre which immediately precedes the act of writing depends upon incitations which reach it from the auditory word-centre. Therefore, when the latter centre is damaged, we may readily understand such a depression of functional activity in the former as may cause agraphia and even word-blindness. The activity of the visual word-centre in connexion with the speech-functions, however, varies within the widest limits in different individuals, from the person who can neither read nor write and in whom this centre may be said not to possess any functional activity, through varying degrees of education, up to the highly-educated strong "visuals," in whom the visual word-centre has an importance in connexion with speech second to none, not even to the auditory word-centre. Thus, a lesion confined to the auditory word-centre in a person little educated as regards reading and writing may give rise to considerable alexia and agraphia as a result of diaschisis. In a highly educated person the same lesion would not be likely to cause more than slight and transitory agraphia, and in a strong "visual" with an identical lesion it is probable that these symptoms would be absent from the first. It is clear, then, that the results of lesions of either of these centres will vary not only according to the different degrees of education of the persons attacked, but also according to the individual endowments in regard to the relative activity of their different word-centres, or, in other words, according as they are strong "auditives" or "visuals" respectively.

The arguments for the existence of a visual word-centre as apart from the auditory word-centre are much the same as those we have already put forward for the existence of the writing centre as apart from Broca's centre. The frequency of the association of symptoms pointing to damage of both these centres is explained by their contiguity and by their close functional relationship. The wide variations in the relative degrees of word-deafness and of word-blindness which are met with in cases of lesion of Wernicke's zone is important evidence that each of these symptom-groups is a result of damage to a separate centre. Upon theoretical grounds, moreover, there must be some region of the cerebral cortex set apart for the registration of impressions connected with so special a function as that of visual speech. It is clear that the visual word-centre is situated upon the confines of the auditory word-centre and at a distance from the general visual cortex, which is limited to the region of the calcarine fissures upon the mesial aspects of the hemispheres. This situation seems to correspond with the manner in which the visual speech-function is acquired, for the child learns to read by coupling up the visual word-symbols with the sounds of the words he knows, and the visual word-centre is organised directly from the already organised auditory word-centre. It is probable, therefore, that the former centre is developed as an annex of the latter centre.

Just as symptoms pointing to an isolated lesion of Broca's gyrus or of the left second frontal gyrus are of great rarity, so are symptoms indicative of lesions confined to the auditory and to the visual word-centres, and affecting the functions that we have attributed to either of these two centres alone; and the same reasons that have led Dejerine to the denial of a separate writing-centre have drawn him to the conclusion that alexia and agraphia are essential results of a lesion in the auditory word-centre.

Paraphasia is certainly not an essential symptom of destruction of the auditory word-centre as Wernicke, Dejerine, and Mirallié have held, for it is present in little more than one-third of the cases of lesions of Wernicke's region.

In the following pages the most common combinations of symptoms that are met with in lesions of this region are dealt with in the following order: (1) defects resulting from abnormal conditions of the left auditory word-centre; (2) defects resulting from the destruction of the auditory word-centres in each hemisphere; (3) defects resulting from destruction of the visual and auditory word-centres in both hemispheres; (4) defects resulting from the isolation of the left auditory word-centre upon the afferent side; (5) defects resulting from abnormal conditions of the left visual word-centre; (6) defects resulting from isolation of the left visual word-centre upon the afferent side; (7) defects resulting from combined lesions of the left auditory and visual word-centres, together with some remarks upon the conditions which have been named "mind-blindness" and "object-blindness."

(1) **Defects resulting from Abnormal Conditions of the Left Auditory Word-centre.**—Of all the word-centres the integrity of the auditory word-centre is the most important, and the defects due to different degrees of functional disability therein are the most varied, because this is the centre in which, in a great majority of persons, words

are first revived during thought, whether this be silent thought, or whether it be the first stage in the processes of thinking and writing.

(*a*) *Effects produced by Functional Degradation or Partial Damage in the Left Auditory Word-centre.*—In the slighter degrees of damage and lowering of functional activity of this centre we have to do with the typical forms of verbal amnesia, in which various words fail to be recalled as they are needed in ordinary speech. Except in rare cases it is in this centre alone that words are spontaneously revived as thought-counters. The kinaesthetic impressions of words are, as we maintain, not spontaneously revivable, and a primary recall of visual word-images is confined to the rarely occurring strong "visuals," and therefore the term "amnesia verbalis" is especially applicable, and is practically confined, to this functional disability of the auditory word-centre. It is generally agreed that in amnesia words are lost in a fairly definite order. The failure is always most marked in the recall of proper names, and then of nouns, these being the most specialised units of speech, and much more rarely is there any difficulty in the recall of verbs, adjectives, and pronouns. As Lichtheim points out, the evidences of amnesia are "more easy to demonstrate when a patient is made to name objects than when he is engaged in ordinary conversation; names that are recalled without effort in fluent speaking arrest him when he has to find them for objects or persons shewn to him." In the slighter forms of amnesia the efforts of recollection of a person who is "at a loss for a word" tend to call the visual word-centre into incipient activity; for many persons, when they cannot get out a word, are often able to recollect the initial letter, and may even seem to know something about the length of the word, since they may be able to say that it consists of so many letters, thus seeming to shew an abortive revival in the visual word-centre. But the fact that this partial revival is not associated with full consciousness of the word, and does not enable it to be written, is one of considerable significance, since it seems to shew how all-important is the primary revival in the auditory centre, not only for the accomplishment of speech, but also for that in writing, the visual word-centre being probably called into action in writing spontaneously, as well as in writing from dictation, through the intermediation of the auditory word-centre. It is reasonably certain that in reading aloud there is first an excitation of the visual word-centre, then the passage of stimuli to related portions of the auditory word-centre, before the incitation passes on to Broca's centre. This affords the explanation of many recorded cases in which speech has been so disordered that the patients could scarcely say more than three or four consecutive words, and could perhaps utter no nouns; and yet, when a book was placed before them, they were able to read aloud almost correctly and with ordinary facility. I have seen three such cases, and several others are recorded in my work on *Aphasia and other Speech Defects.* These cases are of great importance because of the above simple explanation that may be given to them in opposition to the view of Lichtheim, who explained such cases as the result of a lesion of the commissure between his hypothetical "concept centre" and Broca's centre. Such cases as these are almost the only ones that he brought forward as evidence of the existence of his "ideational centre," seeing that he could produce no cases illustrating disease of this "concept centre" itself. He was only able to adduce one case as demonstrating the effects supposed to be due to damage of the other commissure in relation with the "concept centre" (namely, that by which it is brought into relation with the auditory word-centre). This case, however, was of a complicated nature, in which the symptoms varied on successive days, and in our opinion cannot be considered to give any real support to Lichtheim's interpretation and to the existence of an ideational centre.

We have seen, then, that in the cases of slight amnesia the auditory word-centre may fail to respond to volitional incitations while it still responds to the stronger associational stimuli coming to it from the visual word-centre. A still further reduction of the activity of the auditory word-centre may result in the failure of this response to associational stimuli while the centre still responds to direct sensory impressions. When this is the case the speech of the patient is entirely limited to an imitative repetition of words spoken in his hearing, and he is powerless to volunteer any statement. In such cases, which are usually included under the term *echolalia*, a marked impairment of general intelligence almost invariably coexists.

The conditions of amnesia above described may be met with as the result of defective cerebral nutrition, in advancing age, in connexion with general sclerosis of the cerebral arteries, and during convalescence from prostrating diseases, and in these latter conditions the slighter forms of amnesia are met with, such as a halting speech from inability to recall certain words. Slight lesions in the region of the auditory word-centre may cause identical symptoms. The more severe forms of amnesia are due to a partial lesion of this centre of greater extent than the preceding, and they are especially evident in cases of lesion of Wernicke's zone as the lasting symptoms when some recovery has occurred. In patients who have had a knowledge of languages other than their native tongue the amnesia is more marked in connexion with the foreign language, and it is the more obvious the less the facility which the patient has possessed in the use of the foreign tongue, that is, the languages are lost inversely as they are deeply impressed, and when recovery occurs they are regained in the direct order of their depth of impression. The same remarks that have been made upon this subject in connexion with aphasia (*vide* p. 415) are equally applicable to amnesia and to the forms of agnosia that are described in the following pages, namely, word-deafness and word-blindness.

(*b*) *Effects produced by Destruction of the Left Auditory Word-centre.*—When the left auditory word-centre is more or less completely destroyed, a correspondingly complete verbal agnosia is produced which we name word-deafness. Spoken words become to such a patient mere meaningless sounds. It often happens that the patient who is word-deaf can recognise his own name when he hears it, just as the word-blind patient may be able to recognise his name when he sees it and to write his name, though he cannot recognise or write anything else. This most familiar word of all is the one the recognition of which persists the longest, and after the meaning of all other words may have been blotted out. In cases in which the word-deafness is not complete, there may be still the power of comprehending many familiar words, and by this means patients occasionally may guess correctly what is being said to them. Such patients may, therefore, for the moment appear to understand more than they really do, as the questioner may readily ascertain by using some of the same words, but in different combinations and so as to require different answers. For reasons of this kind some of the slighter forms of word-deafness may escape observation unless especially looked for. Again, when a word-deaf person has been acquainted with two or more languages, that which is most used continues to be understood the longest, and when recovery occurs it is the first to be comprehended again.

Word-blindness is also not an infrequent consequence of such a lesion, though for the reasons we have already stated it is not to be regarded as a necessary accompaniment, whatever Wernicke, Dejerine, and others may say to the contrary.

Since the activity of Broca's centre is aroused by incitations coming from the left auditory word-centre it follows that destruction of the latter centre will entail aphasia, but the nature and degree of the speech alteration so resulting may vary surprisingly. If we bear in mind that in certain persons, and especially in strong "visuals," the visual word-centre is capable of acting directly upon Broca's centre, and further, that the right auditory word-centre may in certain persons possess the capacity of arousing the left Broca's centre, the clinical differences that may result from a similar lesion in different individuals become intelligible.

In rather more than one-third of the reported cases of destruction of the left auditory word-centre more or less complete aphasia has been present; in some cases aphasia and paraphasia have existed, whilst in others voluntary speech has been less affected. In a few cases fairly correct speech has been preserved, and we believe that these were examples of strong "visuals" in which the visual word-centre was capable of directly and fully arousing the activity of Broca's centre. This same explanation is applicable to a remarkable case recorded by Pick, in which fairly correct speech was preserved although the auditory word-centre in each hemisphere had been destroyed. It must be presumed, therefore, that the cases of destruction of the left auditory word-centre in which severe degrees of aphasia occur are cases in which for some reason or another compensatory action cannot be effected either by the left visual word-centre or by the right auditory word-centre. Evidence in favour of both of these methods of compensation will subsequently be given, which also shews that, when unaided by the auditory or the visual word-centres, Broca's centre acting alone cannot lead to the production of intelligible speech.

We are of opinion that the direct action of the visual word-centre upon Broca's centre is a possible cause of the paraphasia which sometimes results from destruction of the left auditory word-centre.

(2) **Defects resulting from Lesions of the Auditory Word-centres in each Hemisphere.**—Four cases of this kind have been recorded, and abstracts of them will be found in my work on *Aphasia*. In the cases recorded respectively by Kahler and Pick, by Mills, and by Wernicke and Friedländer, speech was either absent or unintelligible though Broca's centre was intact in each hemisphere. In Pick's patient, already referred to, speech was scarcely at all impaired, and its preservation drives us to the conclusion that this patient must have been a strong "visual" in whom words could be primarily revived in the visual word-centre from which Broca's centre was directly incited.

(3) **Defects resulting from the Destruction of the Auditory and the Visual Word-centres in each Hemisphere.**—In a case of this nature, recorded by J. C. Shaw, the patient was completely word-blind and word-deaf, and could only utter unintelligible sounds, though the third frontal gyrus on each side was intact. Of great interest in this connexion is the well-known case of Laura Bridgman, in whom complete blindness and deafness had been produced in early infancy by peripheral lesions with the result that in later life she was only capable of uttering inarticulate sounds.

(4) **Defects resulting from Isolation of the Left Auditory Word-centre.**—The symptoms that would presumably result if the left auditory word-centre were completely severed from the central acoustic path would be uncomplicated word-deafness. A patient suffering with such a lesion would hear ordinary sounds though more or less badly, since part of his general auditory cortex would be isolated from incoming impressions; he would be able to talk correctly, to write correctly, to read aloud, and to understand what he read, since the speech-centres and their inter-connexions are intact. His three disabilities would be that he would be unable to comprehend spoken words, to repeat words, and to write from dictation. Lichtheim was the first to describe a case presenting these striking clinical peculiarities, and he gave the name of "isolated speech-deafness" to this condition, which he attributed to a subcortical lesion severing "the irradiations of both acoustic nerves in the left temporal region." There was no necropsy in this case (54). Lichtheim's explanation of this case necessitated that there should be a semi-decussation of the central acoustic path, so that each auditory nerve should be in relation with both temporal regions of the cortex. This assumption, which gave rise to some controversy at the time, has during recent years been definitely placed beyond dispute, for it has been shewn that a semi-decussation of the central acoustic path takes place in the corpus trapezoides and for some distance above this, and that the direct and crossed acoustic paths come together at the uppermost level of the mesencephalon. This condition was referred to by Wernicke as "subcortical word-deafness," and Dejerine (25), who published another case, applied to it the term "pure word-deafness" as comparable to the conditions "pure aphasia"

and "pure word-blindness" which are also produced by subcortical lesions. Both Lichtheim and Dejerine seem to think that the right auditory word-centre is either non-existent or not possessed of any functional activity, and, consequently, that it is not brought into relation with the left auditory word-centre. They seem also to hold similar views as to the absence of the visual word-centre in the right hemisphere.

Now, since the condition under consideration never results from destruction of the left central acoustic path in the region of the upper mesencephalon and internal capsule, it is clear that the explanations of Lichtheim and of Dejerine are inadequate, and that for the production of subcortical word-deafness the lesion must not only sever the left central acoustic path proceeding to the left auditory word-centre, but it must also sever the connexions of this centre with the right auditory word-centre. A subcortical lesion situated beneath and not far distant from the left auditory word-centre would presumably sever the left central acoustic path and also the fibres which cross from this region by the corpus callosum to the opposite temporal lobe.

Strangely enough it has been found that a similar clinical condition may be produced in quite a different way, as may be seen by reference to the very remarkable case recorded by Pick, and previously referred to, in which there was a destruction of the auditory word-centre in each hemisphere. Here the clinical condition was almost identically the same. This case seems absolutely inexplicable unless we suppose that the patient was a visual who could speak, write, read aloud, and understand what he read through the activity of the visual and kinaesthetic word-centres—at all events without the co-operation of the auditory word-centres, since these were destroyed.

It will thus be seen that this rare group of symptoms can be induced in two very different modes, just as we shall find subsequently that the condition which Dejerine (25) has called "pure word-blindness" may also be produced in two different modes and that the modes are analogous in the two cases.

(5) **Defects resulting from Abnormal Conditions of the Left Visual Word-centre.**—There is not the same variation in the defects of speech caused by disease of the visual word-centre as is to be met with as a result of disabilities in the auditory word-centre. This is due to the fact that in a majority of persons voluntary revival of words occurs primarily in the auditory word-centre, whilst the visual word-centre is aroused mainly by the stronger stimuli coming to it through associational paths or reaching it directly from without. In rare cases only does the visual word-centre take the place occupied in the great majority of individuals by the auditory word-centre, as the seat of primary revival of words in silent or in spoken thought. Consequently amnesia verbalis is met with almost solely as the result of lowered functional activity in the auditory word-centre, and only in the very rare cases of strong "visuals," as the result of lowered activity of the visual word-centre. Since a volitional stimulus is weaker than that which comes through an associational

channel from a centre more strongly aroused, and weaker still than that which comes to the centre directly from without, and since a lowered activity of a centre from any cause might lead only to a failure in the centre to respond to the weakest stimuli, it is to be expected that such results in relation to language would shew themselves only in connexion with the auditory word-centre which is accustomed to respond to such stimuli, and they would be practically absent in connexion with the visual word-centre.

Consequently, all that can be said is that when functional degradation of the visual word-centre is present it would (*a*) in the rare event of the patient being an extremely strong "visual," of itself tend to produce verbal amnesia, as in a patient of Charcot's whose case is referred to by Ballet; or (*b*) even in an "auditive" tend greatly to augment any disability caused by a coexisting defect of the auditory word-centre, and also to hinder recovery therefrom; and further (*c*) where the functional defect involves both of these word-centres, the amnesia ought to be more than usually bad, and probably associated with more or less of paraphasia.

We have now to consider the *effects produced by destruction of the left visual word-centre*, which is generally supposed to be situated in the region of the angular and supramarginal gyri. A striking difference between the effect of lesions in this situation and of lesions of the left auditory word-centre is that speech is but little if at all interfered with. Word-blindness is present, but there is no well-marked aphasia or paraphasia. At times there may be slight disturbance of speech, and even slight paraphasia, and this is not to be wondered at if we bear in mind the close functional relations of the visual and auditory word-centres, and how easily the functions of the latter may be disturbed by a lesion of the former. The word-blindness is often associated with right-sided hemianopsia due to the co-involvement of the inferior longitudinal fasciculus in the lesion; but where the lesion is limited to the convolutions and to the immediate subcortex, these structures will not be involved, and therefore hemianopsia is not a necessary accompaniment of word-blindness.

Agraphia is a symptom which is sometimes present and sometimes absent in cases of word-blindness, and the presence or absence of this symptom is of such importance that it is convenient to group cases of word-blindness into two categories, namely (*a*) those in which the word-blindness is associated with agraphia, and (*b*) those in which there is no agraphia. In the latter group it is found that the patients can write as well with the eyes closed as when they are open, and further that they are, even after a brief interval, unable to read what they have written.

(*a*) When agraphia is present it is commonly found that the patients can write their names correctly though they can write nothing else. The signature is an emblem which may, by reason of its familiarity, be executed by the cheiro-kinaesthetic centre with the smallest amount of prompting; and possibly this prompting may come from the common visual centre when the left visual word-centre is destroyed. Similarly a patient who is word-blind may be able to recognise his own name when he sees it, although apart from this there may be complete alexia. As Dejerine (25) says, "He recognises it by its general form, by its physiognomy, and not by the assemblage of letters of which it is composed"—just, in fact, as he would recognise a geometrical figure or any other drawing. Differences of degree, however, occur in word-blindness. In some cases, though the patients are unable to recognise words, they can recognise individual letters; whilst in others not even letters can be recognised (letter-blindness). These two forms represent differences in degree rather than in kind, and all intermediate varieties are met with. Sometimes certain letters are recognised and not others, and in one case recorded by Batterham vowels alone were recognised.

Dejerine and Sérieux have each published cases of this type in which word-blindness was associated with agraphia, and in which the lesion was strictly limited to the visual word-centre.

(*b*) In the second group of cases word-blindness is not associated with agraphia, and the patients are able to write, and as well with the eyes closed as open. This group of symptoms may be produced in two totally distinct manners. In the first place, it may result from destruction of the left visual word-centre, which is a rare event; and secondly, it is the constant result of a subcortical lesion which isolates the left visual word-centre from all connexions, both direct and indirect, with the common visual centres in the cuneate lobes. The latter group of cases will be fully considered in the next section.

The effects of destruction of the left visual word-centre are liable to vary in different individuals according to their different sensorial aptitudes and different degrees of education, just as we have found the results of destruction of the left auditory word-centre to vary markedly in different cases. The results already described under group (*a*) are those which are usual when the left visual word-centre is destroyed in ordinary individuals. But suppose a similar lesion to occur in a person who is a strong "auditive" though a weak "visual," and at the same time an educated person who has been in the habit of writing much. It may happen in such a person, after the stage of learning to write has long passed, that the activity of the visual word-centre might be reduced to a minimum during the act of writing. We have seen that the words about to be written become nascent first in the auditory word-centre, and it seems probable that in persons with the endowments above mentioned this centre, rather than the visual word-centre, may be the one that acts upon and arouses the activity of the cheiro-kinaesthetic centre, just as we have seen that in strong "visuals" the visual word-centre may be capable of directly arousing the activity of Broca's centre for the production of articulate speech. We contend, therefore, that the preservation of ability to write, where there has been word-blindness and where the left visual word-centre has subsequently been found to be destroyed, is to be explained upon the ground that the auditory word-centre, instead of acting as it usually does by rousing the visual word-centre to conjoint action, in these cases acts directly upon the cheiro-kinaesthetic centre (by way of the commissure *f f* in Fig. 21). The writing thus produced may be good and without mistakes, but some patients may shew defects of a paragraphic type. The handwriting, too, is usually larger than that which was previously customary to the patient. There is also the peculiarity that these patients can write as well with the eyes closed as open, and the further peculiarity that they cannot subsequently read what they have written except by a manœuvre which causes a stimulus to pass in the reverse way, that is, from the cheiro-kinaesthetic centre back to the auditory word-centre. This is brought about by passing the tip of the finger over the outlines of the letters that have been written, and so reading off the result. The possibility of this mode of reading by the tip of the finger was first noticed in a word-blind patient by Westphal.

(6) **Defects resulting from Isolation of the Left Visual Word-centre. Pure Word-blindness.**—A clinical group of symptoms identical with that described in the last section is produced when the left visual word-centre is cut off from all the afferent fibres which reach it from the common visual centres in the cuneate lobes, both those that reach it directly from the cuneus of each hemisphere and those that reach it indirectly by means of the right visual word-centre and fibres crossing thence in the corpus callosum.

The recorded cases of pure word-blindness in which the visual word-centre remains intact are comparatively few. Three complete cases with necropsies have been published (by Dejerine, Wyllie, and Redlich), whilst in several others the clinical details only have been published. Dejerine's case is remarkable for its completeness and for the great care with which it has been recorded (24). Its most noticeable features were the absence of any hemiplegia, the combination of complete right lateral hemiachromatopsia with partial right hemianopsia, the absence of word-deafness or of speech-defect of any kind, the absolute word-blindness coupled with ability to write freely both spontaneously and from dictation, whilst ability to copy writing was very defective, together with the length of time that these symptoms lasted without change. The subsequent sudden supervention of complete agraphia and paraphasic speech, followed by the discovery of a recent lesion in the angular gyrus and adjacent parts were also very significant features in this patient. In the cases recorded by Prof. Wyllie and by Redlich there was also no motor paralysis, but a slight degree of right hemianaesthesia was present. There was right hemianopsia in both cases, but no mention is made of hemiachromatopsia nor of the ability to read words by means of kinaesthetic impressions, though both these phenomena were present in Dejerine's case. It is highly probable that these two features would have been found had they been specially looked for in the cases of Wyllie and Redlich, and that hemiachromatopsia (when the hemianopsia is partial) and ability to read words by means of kinaesthetic impressions are characteristic accompaniments of the form of pure word-blindness under consideration, since, in the several reported cases without necropsy, whenever these two symptoms have been mentioned in the records they have been referred to as present and never as absent.

The lesions found in each of the three cases with necropsy have shewn a striking similarity. There has been softening and atrophy of the white substance of the occipital lobe with damage to some of the convolutions upon the under and inner surface of the occipital lobe, either to the lingual and fusiform lobes, the hippocampal gyrus, or to the cuneus. Dejerine attaches most importance in the production of the symptoms to the destruction of the white matter in the region where this includes (*a*) the inferior longitudinal fascicle, (*b*) the projection from the left cuneus to the left visual word-centre, and (*c*) the projection of the right cuneus to the left visual word-centre, which crosses in the splenium of the corpus callosum. In this connexion it must be mentioned that the "optic radiations" of Gratiolet, which were formerly supposed to constitute the path by which visual impressions passed to the half-vision-centres in the cuneate lobe, have been proved to consist solely of corticifugal fibres, and that the afferent visual path is the inferior longitudinal fasciculus which is situated in the external sagittal layer of the occipital white matter.

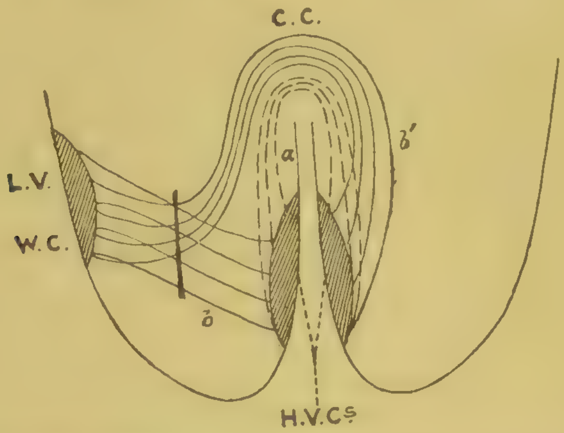

Fig. 23.—A simplified diagram representing Dejerine's view as to the mode of production of pure word blindness. H. V. C., Half-vision-centres; L. V. W. C., left visual word-centre; C. C., posterior extremity of corpus callosum, containing commissural fibres (*a*) connecting the half-vision-centres and also fibres (*b′*) from the right half-vision-centre to the left visual word-centre. (The "optic radiations" have been omitted.) The dark line indicates the site of a lesion which would cut off the left visual word-centre from the half-vision-centre of each side.

Dejerine's explanation is obviously an adequate one only if one holds, as he does, that auditory and visual word-centres do not exist in the right hemisphere in right-handed people. We, however, believe in the existence of a visual word-centre in each hemisphere (though unequally developed and of widely different functional capacity), and that the two are brought into functional relations with one another by means of commissural fibres crossing in the corpus callosum, and further that each of the visual word-centres is in relation by associational fibres with the half-vision-centre of its own side, while the two half-vision-centres are also commissurally connected. In our opinion, therefore, Dejerine's form of pure word-blindness is produced by severance of the associational fibres between the left visual word-centre and its corresponding half-vision-centre, together with severance of the commissural fibres between the two visual word-centres in some part of their course (see Fig. 24). The isolation of the left visual word-centre upon the incoming side would thus be complete, and brought about moreover in the same way that we have postulated for the isolation of the auditory word-centre in the condition "pure word-deafness." The commissural fibres between the two word-centres may be damaged by the lesion in the white centre of the occipital lobe extending far enough forward to involve them just as Dejerine supposes those from the right half-vision-centre to have been destroyed; or else by a separate lesion in the splenium of the corpus callosum, such as was actually found in Dejerine's case, or in the part anterior to this known as the tapetum, for in Redlich's case the fibres of the tapetum were in part degenerated. When we consider how one eye suffices for perfect vision, although under these conditions only one-half of the proper amount of visual fibres goes to each half-vision-centre, we see all the more fully how complete and intimate must be the co-activity of these centres in vision, even when one eye only exists. Therefore, it would be vain with our present imperfect knowledge to attempt to define the actual modes of association of the half-vision-centres with the visual word-centres. Such an arrangement as we have above indicated seems, however, to be more in accord with all known probabilities than that of Dejerine, who assumes that the right angular gyrus has no visual functions, just as he assumes that the right temporal region has no speech-functions. What has been already said as to the partial co-education of the centres in each hemisphere, as well as what we know as to the modes in which recovery takes place in various forms of speech-

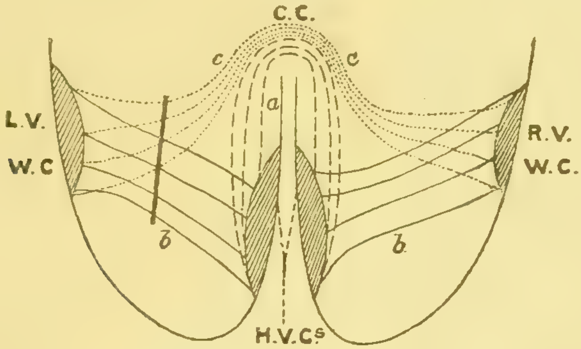

FIG. 24.—A diagram representing my view as to the mode of production of pure word-blindness. c. c., Posterior extremity of corpus callosum; c, c, commissural fibres connecting the two visual word-centres; b, b, fibres connecting each half-vision-centre with the visual word-centre of the same side.

defect, may be considered to lend support to this view. As has already been indicated, the two modes of production of pure word-blindness are strictly comparable with the two modes of production of pure word-deafness. One of these forms, which may be called the cortical type, is brought about by a destruction of the left visual word-centre in persons by whom writing can be executed under the instigation and guidance of the left auditory word-centre; whilst Dejerine's form, which may be called the subcortical type, is due to isolation of the left visual word-centre from the half-vision-centre of its own side as well as from the opposite visual word-centre.

At present there seems to be no definite means of clinically distinguishing these two types of pure word-blindness, but it may be borne in mind that the presence of slight paraphasia is suggestive of a cortical word-blindness, and that in the subcortical type hemianopsia is invariably present, and where this is incomplete there is probably hemiachromatopsia.

7. **Defects resulting from Combined Lesions in the Left Auditory and Visual Word-centres.**—It will have been seen from the cases already recorded that destruction of the left auditory word-centre or of the left visual word-centre alone, and especially in the last case, may be quite compatible with the preservation of a fair degree of intelligence. The result, however, is altogether different when both these centres are simultaneously and severely involved. The unfortunate patients thus affected are reduced to a most deplorable condition, seeing that they can neither speak nor write intelligibly, and that they are unable to understand the speech which they hear or the language that they may see either in writing or in print. They can communicate with others, and be communicated with, only by means of signs and gestures; and at the same time they must necessarily suffer a considerable mental impairment, owing to the blotting out of the principal symbols by means of which all but their most elementary thinking processes are carried on. This is the condition that has been met with in the majority of cases, yet a careful examination of all the published records shews that, as in cases in which there is destruction of the left auditory word-centre, so here with combined lesions of the auditory and visual word-centres a considerable variation in the resulting symptom-group may be met with in different cases. These variations depend partly upon differences in the relative completeness of the damage in one or other of these two centres; and where one of these centres is only partly damaged, probably also to a considerable extent upon individual variations in original endowment—that is, upon the question whether the patients are "auditives" or "visuals," as well as in part upon their degree of education. Moreover, where the destruction of the left word-centres has been complete, variations in symptoms may depend upon the degree of the development of the corresponding centres in the opposite hemisphere.

A consideration of 15 of the most typical cases that can be found on record shews that in 3 of them the lesion was complete in the visual and partial in the auditory word-centre; in 2 it was complete in the auditory and partial in the visual word-centre; in 4 it was incomplete in both centres; whilst in 6 it was fairly complete and equal in both the word-centres. An examination of the clinical aspects presented by these 15 cases tells us that speech was fairly good in 2 cases; that there was marked paraphasia or actual jargon-speech in 7, and more or less complete speechlessness in 6. These different results may seem astonishing at first, but the more one considers cases presenting this double lesion, in which speech was fairly good or only slightly paraphasic, the more it becomes necessary to suppose that the auditory word-centre of the right hemisphere must have been able to act upon and with the left glosso-kinaesthetic centre. Such cases cannot be explained by supposing that speech was produced by the right auditory word-centre acting with the right Broca's centre, because all present knowledge goes to shew that this could only be brought about after a long interval during which these centres were educated to act together. But in the cases in question there was evidently no such interval, seeing that the speech-alteration was present in each case just after the lesion occurred. It is important to note also that in the 2 cases in which speech was best preserved the visual word-centre was only partially damaged, so that some help may also have come from its co-operation.

A case recorded by myself (6) belonging to this group is one of a very extraordinary nature in many respects, the patient having lived eighteen years after his seizure, and his speech-defects having remained constant throughout almost the whole of this period. His spontaneous speech was limited to a few words, and though he was neither word-deaf nor word-blind, both the visual and the auditory regions of the left hemisphere were completely destroyed.

Object-blindness or Visual Agnosia.—In connexion with cases in which lesions of the auditory and visual word-centres have occurred, and with other cases in which lesions have involved the white substance of the occipital lobes, a defect is sometimes met with which has been commonly known as "mind-blindness." For this condition Ballet and Wyllie have introduced the more appropriate term "object-blindness," which is not only more descriptive of the nature of the defect, but it is also a better companion term for word-blindness, since these two conditions are not infrequently associated. When object-blindness is present, although the patient sees familiar persons and things perfectly he seems unable to recognise them, and he is therefore unaware of the uses of common objects. There is conservation of sensation but failure of recognition owing to complete or partial isolation of the common visual centre. It is clear that a similar lack of recognition, due to defect in perceptive processes, may exist in connexion with each of the senses, and, therefore, Freud has introduced the term "agnosia" as a generic term for all these conditions. Thus, we may have auditory agnosia, in which sounds are heard but their import is not recognised; visual agnosia or object-blindness; tactual agnosia, in which there is no loss of sensibility, but the patient is unable to appreciate the nature and uses of familiar objects by the feel of them; olfactory agnosia; and gustatory agnosia. It will be at once seen that word-blindness and word-deafness are special varieties of visual and auditory agnosia respectively.

As above indicated, object-blindness is probably due to the partial isolation of the common visual centres upon the outgoing side—to a severance of the paths by which these centres are brought into association with other sensory centres. A failure in the irradiation of the sensory impressions which pass from the visual to other sensory centres upon the presentation of an object to the visual sense would, of course, absolutely check the neural processes essential to perception, and thus lead to a non-recognition of the object and its uses.

Optic aphasia is a term which has been given, somewhat needlessly, by Freud to a condition in which, though the patient recognises persons and objects, he is unable to name them at sight, though he may be able to do so at once when the visual impression is reinforced by some other sensory impression—either by smelling, tasting, and especially touching the object. This defect is one of slight visual agnosia, for, since the name forms an integral part of the complete percept of an object, it is obvious that perception in this case is not complete. This inability to name at sight may be brought about in various ways: (*a*) by severance of the associational fibres between the common visual centre and the visual word-centre; (*b*) where the visual word-centre is weak, it may then respond to two incitations, though not to the visual alone; (*c*) when the visuo-auditory commissure is damaged in a person who is not a strong "visual."

Inability to Read and Write Music or to Read and Write Numerals.—It is often found that inability to read or write numerals or music is not proportionate to the inability to read or write words. In reference to music, a case was long ago reported by Lasègue in which a musician, completely aphasic and unable to read or write in the ordinary way, could, after hearing a passage of music, write such a passage on paper with facility. Many other instances of the same kind have been recorded, the inference from which would seem to be that the seat of registration of the two kinds of impressions may be more or less separate though probably contiguous.

In other cases there may be complete word-blindness and musical-blindness, but with preservation of ability to read and write figures and to calculate, as in the celebrated case of pure word-blindness recorded by Dejerine. It is, indeed, frequently found that there is ability to read and write figures with varying degrees of facility when the reading or writing of letters or words is impossible. This curious fact has given rise to much discussion and to many attempts to explain it. It is probable that various factors are operative, though in different combinations in different cases. Some of the most important causative conditions may be these: (1) the greater simplicity in the recall of numerals as compared with letters—there being only nine of the former as against twenty-six of the latter; (2) there is the fact that our attention is much more habitually called to individual figures, whilst letters tend to be merged into multitudinous words; (3) there is the possibility that numerals may much more often than words be primarily revived in the common visual centre rather than in the auditory word-centre; and (4) the further possibility that numerals may be registered in some part of the general visual cortex apart from the visual word-centre.

Paraphasia and paragraphia are defects resulting from inco-ordination or apraxia in the speech- or writing-mechanism. They may each be described as occurring in three forms: (1) in the minor form of the defect, the patient uses some wrong words instead of those which he wishes to employ; (2) in a more severe form, he collocates words in such a disorderly manner as to convey no meaning; (3) in the most severe form, he does not make use of words at all, but utters a mere gibberish or jargon in which actual words are not to be detected; or, in writing, makes random collections of letters or perhaps unmeaning strokes not even representing letters.

The first form is the most common, and is usually associated with a certain amount of amnesia, when the patient will often substitute some noun of general import (such as the word "thing") in place of that which he is unable to recall, or else he substitutes a periphrase in order to express his meaning. An allied defect may also be met with in the form of a transposition of syllables of words in the same sentence, or the interposition of syllables that do not belong to the word. Defects in writing of a similar kind are also met with. All transitions are encountered between the first and the second forms of paraphasia, and the two forms are probably similar in nature and mode of production. Some of the slighter defects are apt to shew themselves in weak and exhausted states of the system and in cases in which there is impaired nutrition of the brain, either from previous disease or from old age. They may occur also when the attention of the person speaking is distracted by counter currents of thought being carried on at the same time. In all such cases wrong words are apt to slip out.

Apart from these causes of a more general order it is highly probable that paraphasia or paragraphia, or both of these phenomena, are produced by lesions which cause speech or writing to be performed by some unaccustomed cerebral process. For example, paraphasia not infrequently occurs in a case of word-deafness, when the visual has to take the place of the auditory word-centre in the incitation of Broca's centre; or in cases of combined word-deafness and word-blindness, when the right auditory word-centre has to take on the functions of the left. In each of such cases there is apt to be, at all events for a time, either a lacking of proper association between the new centre in which words have to be revived and the several sensory centres and their annexes, or else an inco-ordinate action between the driving centre and the centre driven.

Under similar conditions paragraphia may also be present, since here, too, there is a change in the locus of the initiatory recall of words and a less perfect system of associational fibres radiating therefrom, as well as a less evolved system of associational channels between this newly evolved dominant site and the left cheiro-kinaesthetic centre. Paragraphia may also be caused in cases in which the visual word-centre is destroyed in a person much accustomed to write, when this act begins to be brought about, by the auditory word-centre acting directly upon the cheiro-kinaesthetic centre. Further, some partial damage to the auditory word-centre may cause paraphasia; and similarly partial damage to the visual word-centre may cause paragraphia.

Recent investigators upon the subject of apraxia (Liepmann, Heilbronner, Maas, Wilson, and others) have adopted an explanation very similar to that above given for certain defects in the performance of subjective purposive movements other than those concerned in speech and writing, which are phenomena of the same order as are paraphasia and paragraphia. For such conditions the term "parapraxia" has been used.

Prof. Wyllie seems to suppose that in cases of word-deafness the paraphasia which often occurs may result from an unguided action on the part of Broca's centre (96). Thus, he says, "In cases of word-deafness—which implies the obliteration of the auditory store of images—the motor centre may emit the wrong words when the patient tries to speak." As we have shewn, however, in previous sections, Broca's centre alone appears to have no power of initiating intelligible speech—undirected it seems only to lead to the production of mere gibberish. Lichtheim's view, that paraphasia is dependent upon a severance of the associational fibres between the left auditory word-centre and Broca's centre, is founded upon insufficient evidence as Dejerine also has pointed out. We have shewn that destruction of the auditory word-centre gives rise to paraphasia in a certain number of the cases, and in about an equal number it gives rise to aphasia. Precisely the same thing must hold good, therefore, with regard to severance of the associational path between the auditory word-centre and Broca's centre. Thus, paraphasia might be produced when either the left visual word-centre or the right auditory word-centre are capable of partially taking on the functions of the left auditory word-centre when the latter centre has been cut off from its connexions with Broca's centre, whilst aphasia would result in cases in which from one cause or another neither of the centres mentioned could develop compensatory functions so as to incite and direct the action of Broca's centre.

In the third form of paraphasia and paragraphia speech is reduced to a mere jabber of meaningless sounds, and writing to a mere scrawl in which words and sometimes individual letters are indistinguishable. It is often found that these two disabilities coexist, and that the patient is also word-deaf and word-blind, and, consequently, is not aware that what he says or writes is mere gibberish. This condition results in certain cases of destruction of the left auditory and visual word-centres when the corresponding centres in the right hemisphere remain comparatively undeveloped, and still more certainly when both hemispheres are the seat of this double lesion. Again, much the same condition may exist (save for the possible absence of word-blindness) in cases in which both auditory word-centres are destroyed. These defects must therefore be the results of unguided or relatively unguided activity in Broca's convolution, and they correspond with those apraxic defects which result from agnosia and which have been called parapraxia. An extreme defect in speech of the jargon type or a similar defect in writing may exist alone —that is, without the association of word-deafness, word-blindness, or mental failure. Such cases belong to an entirely different category from those last referred to. A well-known instance of such a defect in speech without defect in writing was recorded long ago by Osborne. It occurred in a scholar of Trinity College, Dublin, who was able to talk only in a meaningless jargon and who read aloud in much the same way, though he perfectly comprehended what he read and all that was said to him and expressed his ideas in writing with considerable fluency. His power of repeating words after another person was, however, confined to certain monosyllables. This case seems only explicable on the supposition of some perverted activity of the glosso-kinaesthetic centre.

In other cases, examples of which have been recorded by Dr. Hughlings Jackson (42), P. J. Cremen, and by myself (11), a similar defect has occurred in connexion with writing. Though these cases were not free from complications, there is reason to believe that the agraphic defects must have been especially due to a perverted condition of activity of the cheiro-kinaesthetic centre. The case recorded by myself was of a somewhat different type. There was a constant repetition in this patient's writing of certain groups of letters which varied from time to time; and this case also afforded an admirable example of what Gairdner long ago termed "intoxication of the brain" with words or letters, and to which Neisser in 1894 gave the name "perseveration," a term which has since come into general use. This phenomenon of perseveration is not unfrequently seen in cases of aphasia and in those who are partially agraphic, and recently Pick (78) has emphasised its importance in connexion with apraxia, and he has attributed its occurrence to feebleness in the association of ideas. In all such cases we may expect to meet with evidence of some general mental defect over and above that which may be due to the mere lesion causing aphasia and agraphia.

VI. DEFECTS DUE TO DAMAGE OF THE COMMISSURES BETWEEN THE DIFFERENT WORD-CENTRES.—It seems better to reserve, as I have for some years done, the word "commissure" as an appellation for the fibres which connect centres of a like kind, that is, either motor centres or sensory centres, and to name "internuncial" the fibres which connect sensory with motor centres. These two orders of fibres will thus be clearly distinguished from one another as well as from the sensory and motor nerve-fibres which connect the periphery with sensory centres and which connect the motor centres in the bulb and cord with muscles respectively.

The three chief commissures in the cerebral speech-mechanism are (i.) that between the visual and auditory word-centres; (ii.) that between the auditory word-centre and Broca's centre; and (iii.) that between the visual word-centre and the cheiro-kinaesthetic centre. The effects of structural defects in the course of these paths will be referred to in this order.

(i.) *Defects due to Damage in the two Commissures between the Auditory and the Visual Word-centre.*—One of these commissures conducts impressions from the auditory to the visual word-centre (the audito-visual commissure), and is called into play in writing from dictation and probably also in any spontaneous writing. The other conducts impressions from the visual to the auditory word-centre (the visuo-auditory commissure), and is habitually called into play when we read aloud or name an object at sight.

Where both of these commissures are destroyed without notable damage to other parts of the speech-mechanism, a very interesting group of symptoms is produced. The patient understands perfectly what is said to him and what he reads. He cannot read aloud a single word or letter, though immediately he hears the word or letter pronounced he can at once repeat it. He cannot write a single word or letter from dictation, but he can copy any word or letter that he has before him. Spontaneous writing is similarly impossible, though if the auditory word-centre be intact spontaneous speech may not be much interfered with. An interesting case of this condition, which was for many years under my observation, will be found recorded in the *Medico-chirurgical Transactions*, 1897, lxxx. 61.

Cases which I interpret as illustrating defects of the audito-visual commissure alone have been published by Pitres (82), who, however, brings them forward as instances of the destruction of the cheiro-kinaesthetic centre. Another such case has been recorded by Dingley.

(ii.) *Defects due to Damage to the Commissure between the Auditory Word-centre and Broca's Centre.*—To the result of such a lesion (the "commissural aphasia" of Wernicke) we have recently referred when speaking of paraphasia, so that nothing more need be said here.

(iii.) *Defects due to Damage of the Commissures between the Visual Word-centre and the Cheiro-kinaesthetic Centre.*—Such a lesion should in most persons produce complete agraphia—nothing more than slow and tedious copying being possible under the guidance of the general visual centre, just as a drawing would be copied. But in persons who have been much accustomed to write, it is possible that writing may be executed owing to the direct action of the auditory word-centre upon the cheiro-kinaesthetic centre, though at first the writing would shew paragraphic defects.

The other commissures which have been mentioned seem to be only for occasional or supplemental use, and the effect due to their severance which would tend to prevent compensation in different ways may be easily imagined by reference to the centres which they connect, together with the direction in which the stimulus passes.

Thus, referring to Fig. 21, it will be seen that there are the following additional commissures, the functions of which have been referred to at different times in the course of this article.

(iv.) From the visual word-centre to the glosso-kinaesthetic centre.

(v.) From the auditory word-centre to the cheiro-kinaesthetic centre.

(vi.) From the cheiro-kinaesthetic centre to the auditory word-centre.

(vii.) From the cheiro-kinaesthetic centre to the visual word-centre.

(viii.) There is another important commissure not represented in this diagram, by means of which the right auditory word-centre is enabled to exercise some influence upon Broca's centre in most persons, and a much greater influence in some persons when the left auditory and visual word-centres are destroyed, as seems to be shewn by the fact that some of these patients preserve a fair power of speech, though mostly of a paraphasic type.

H. Charlton Bastian, 1899.
James Collier, 1910.

REFERENCES

(A very full bibliography of this subject up to the year 1907, in chronological order, will be found in Moutier's work *L'Aphasie de Broca*. This work also contains short details of 387 cases from the literature. The publications in 1908 will be found in reference No. 18, and the literature connecting this subject with apraxia in reference No. 95.

In Dr. Bastian's work, *A Treatise on Aphasia and other Speech Defects*, 1898, the whole subject is treated in a more complete manner than is possible in the present article, and is illustrated by details of over one hundred cases.)

1. Amidon. *New York Med. Journ.*, 1885, xli. 113, 181.—2. Ballet, G. *Le Langage intérieur*, Paris, 1886, 101.—3. *Idem.* *Rev. neurol.*, Paris, 1900, viii. 364.—4. Banti, Guido. *Sperimentale*, Firenze, 1886, lvii. 261.—5. *Idem.* *Clin. mod.*, Firenze, 1907, xiii. 49.—6. Bastian. *A Treatise on Aphasia and other Speech Defects*, London, 1898.—7. *Idem.* *Lancet*, 1897, i. 933, 1005, 1131, 1187.—8. *Idem.* *Ibid.*, 1897, i. 1016,—9. *Idem.* *Ibid.*, 1897, i. 1190.—10. *Idem.* *Brit. Med. Journ.*, 1887, ii. 734.—11. *Idem.* *Brit. and For. Med.-Chir. Rev.*, Lond., 1869, xliii. 482.—12. Bateman. *Aphasia*, Lond., 2nd edit., 1890, 273.—13. Batterham. *Brain*, Lond., 1888, xi. 488.—14. Beevor, C. E. "On the Distribution of the different Arteries supplying the Human Brain," *Phil. Trans. Roy. Soc.*, London, 1908, B. cc. 1.—15. Broadbent, W. H. *Med.-Chir. Trans.*, Lond., 1872, lv. 178.—16. Bernheim, F. *Thèse*, Paris, 1900.—17. Charcot. *Diseases of the Nervous System* (New Syd. Soc.), Lond., 1889, t. iii. 372.—18. Collier, J. "Recent Work on Aphasia," *Brain*, Lond., 1908, xxxi. 523.—19. Daly. *Brain*, Lond., 1889, x. 233.—20. Dejerine. *Compt. rend. Soc. biol.*, Paris, 1891, 9me sér., iii. 155.—21. *Idem.* *Bull. Soc. anat. de Par.*, 1879, 4me sér., iv. 16, 13.—22. *Idem.* *Rev. de méd.*, Paris, 1885, v. 174.—23. *Idem.* *Compt. rend. et mém. Soc. biol.*, Paris, 9me sér., iii. 197.—24. *Idem.* *Ibid.*, 1892, 9me sér., iv. 87.—25. *Idem.* "L'Aphasie sensorielle," *Presse méd.*, Paris, 1906, xiv. 437.—26. *Idem.* "L'Aphasie motrice," *ibid.*, 1906, xiv. 453.—27. *Idem.* *Ibid.*, 1906, xiv. 742.—28. *Idem.* *Encéphale*, Paris, 1907, ii. 471.—29. Dingley. *Brain*, Lond., 1886, ix. 492.—30. Elder. *Aphasia and the Cerebral Speech-Mechanism*, Lond., 1897, 31.—31. Flechsig. *Neurol. Centralbl.*, Leipz., 1894, xiii. 674; and *Gehirn und Seele*, Leipzig, 1895.—32. Fleury. *Gaz. hebdom.*, Paris, 1865, 2me sér., ii. 225, 244.—33. Friedländer. *Fortschr. der Med.*, Berl., 1883, i. 176.—34. Freund. *Arch. f. Psychiat. u. Nervenkr.*, Berl., 1889, xx. 276.—35. Gairdner. *The Functions of Articulate Speech*, Glasgow, 1866, xx.—36. Gordinier. *Amer. Journ. Med. Sc.*,

Phila., 1899, cxvii. 526.—37. *Idem.* *Ibid.*, 1903, cxxvi. 490.—38. *Idem.* *Ibid.*, 1907, cxxxiii. 717.—39. HEILBRONNER. *Arch. f. Psychiat. u. Nervenkr.*, Berl., 1907, liii. 234, 698.—40. HUN. *Amer. Journ. Insan.*, Utica, 1851, vii. 358.—41. JACKSON, HUGHLINGS. *Brain*, Lond., 1877, i. 320.—42. *Idem.* *Lond. Hosp. Rep.*, 1864, i. 432.—43. JACKSON, SCORESBY. *Amer. Journ. Med. Sc.*, Phila., 1828, iii. 272.—44. JACOB. *Brit. Med. Journ.*, 1890, ii. 622.—45. KOSTENITSCH. *Deutsche Ztschr. f. Nervenh.*, Leipz., 1893, iv. 369.—46. KUSSMAUL. Ziemssen's *Cyclopaedia Pract. Med.*, Lond. 1878, xiv. 844.—47. *Idem.* *Ibid.*, 1878, xiv. 676.—48. *Idem.* *Ibid.*, 1878, xiv., 777.—49. LADAME. *Rapp. à la Sect. de neurol., 13^e Congr. internat. de méd.*, Paris, 1900, 29.—50. LADAME et von MONAKOW. "Observation d'aphémie pure," *Encéphale*, Paris, 1908, iii. 195.—51. LAIGNEL-LAVASTINE et SALAMON. *Bull. et mém. Soc. méd. hôp. de Paris*, 1906, 3me sér., xxiii. 1298.—52. LEVA. "Zur Localisation der Aphasien," *Virchows Arch.*, 1893, cxxxii., 333.—53. LICHTHEIM. *Brain*, Lond., 1885, viii. 477.—54. *Idem.* *Ibid.*, 1885, viii. 460.—55. *Idem.* *Ibid.*, 1885, viii. 480.—56. *Idem.* *Ibid.*, 1885, viii. 482.—57. LIEPMANN. "Das Krankheitsbild der Apraxie," *Monatsschr. f. Psychiat. u. Neurol.*, Berl., 1900, viii. 15, 102, 182.—58. *Idem.* *Berlin. Gesellsch. f. Psychiat. u. Nervenkr.*, 1906.—59. *Idem.* *Journ. f. Psychol. u. Neurol.*, Leipz., 1907, ix. 279.—60. *Idem.* *Neurol. Centralbl.*, Leipz., 1907, xxvi. 473.—61. LIEPMANN et MAAS. *Presse méd.*, Paris, 1907, xv. 421.—62. MARIE, PIERRE. "Revision de la question de l'aphasie," *Semaine méd.*, Paris, 1906, xxvi. 493.—63. *Idem.* *Bull. et mém. Soc. méd. des hôp. de Paris*, 1907, 3me sér., xxiv. 1291.—64. *Idem.* "Sur la fonction de langage," *Rev. phil.*, Paris, 1907, xlviii. 23.—65. MIRALLIÉ. "De l'aphasie sensorielle," *Thèse de Paris*, 1896.—66. *Idem.* *Ibid.*, 171.—67. *Idem.* *Ibid.*, 191.—68. MONAKOW, von. "Aphasie und Diaschisis," *Neurol. Centralbl.*, Leipz., 1906, xxv. 1026.—69. *Idem.* *Ergebnisse der Physiologie*, Wiesb., 1907, 23.—70. MOUTIER. *L'Aphasie de Broca*, Paris, 1908, 2 vols.—71. NEISSER. *Allg. Ztschr. f. Psychiat.*, Berl., 1894, 1015.—72. NOTHNAGEL. v. Ziemssen's *Cyclopaedia of Pract. Med.*, Lond., 1877, xii. 673.—73. OGLE, W. *St. George's Hosp. Rep.*, Lond., 1867, ii. 83.—74. OSBORNE. *Dublin Journ. Med. Sc.*, 1834, iv. 157.—75. OULMONT. *Bull. Soc. anat. de Paris*, 1877, 4me sér., ii. 327.—76. PICK. *Arch. f. Psychiat. u. Nervenkr.*, Berl., 1892, xxiii. 909.—77. *Idem.* "Zur Psychologie der motorischen Apraxie," *Neurol. Centralbl.*, Leipz., 1902, xxi. 994.—78. *Idem.* *Studien über motorischen Apraxie*, Leipz., 1905.—79. PITRES. *Sur les lésions du centre ovale*, Paris, 1877; cases 21, 25.—80. *Idem.* *Ibid.*, case 39.—81. *Idem.* *Rev. de méd.*, Paris, 1884, iv. 864.—82. *Idem.* *Ibid.*, 1884, iv. 855.—83. ROSS. "On Aphasia," *Med. Chron.*, Manchester, 1887, v. 1, 97.—84. RUSSELL, RISIEN. *Proc. Roy. Soc.*, Lond., 1895, lviii. 237.—85. SÉGLAS. *Sur les troubles de langage chez les aliénés*, Paris, 1892.—86. SEMON and HORSLEY. *Phil. Trans. Roy. Soc.*, Lond., 1890, B. clxxxi. 187.—87. SÉRIEUX. *Rev. de méd.*, Paris, 1893, xiii. 733.—88. *Idem.* *Compt. rend. et mém. Soc. de biol.*, Paris, 1892, 9me sér., iv. 13.—89. SHAW, J. C. *Arch. of Med.*, New York, 1882, vii. 77.—90. TROUSSEAU. *Lectures on Clinical Medicine*, transl. by Bazire (New Syd. Soc.), Lond., 1868, i. 261.—91. *Idem.* *Ibid.*, 219.—92. WERNICKE. *Der aphasische Symptomencomplex*, Breslau, 1874.—93. *Idem.* *Fortschr. der Med.*, Berl., 1883, i. 176.—94. WESTPHAL. *Ztschr. f. Ethnol.*, Berl., 1874, *suppl. Verhandl. Gesellsch.*, 94.—95. WILSON, S. A. K. "A Contribution to the Study of Apraxia," *Brain*, Lond., 1908, xxxi. 164.—96. WYLLIE. *The Disorders of Speech*, Edinburgh, 1895, 487.—97. *Idem.* *Ibid.*, 238.—98. *Idem.* *Ibid.*, 300-302 and 309.

H. C. B.
J. C.

APRAXIA AND AGNOSIA

By James Collier, M.D., F.R.C.P.

Apraxia (*Motor Apraxia, Dyspraxia*). — **Definition.** — A disorder of cerebral function characterised by inability to perform certain familiar purposive movements, in the absence of motor paralysis, sensory paralysis, and ataxia. The condition does not depend upon defective perception (agnosia), nor upon lowering of general intelligence.

The clinical features of apraxia are most frequently seen in certain cases of hemiplegia in which, notwithstanding the complete recovery of the motor and sensory paralysis, the performance of familiar acts, from the most highly skilled movements, such as the fingering of the pianoforte or of the violin, or the use of his tools by the craftsman, to the simplest act may be no longer possible. Motor aphasia and motor agraphia are examples of apraxia.

Historical.—Attention was first drawn to the phenomena of apraxia by the greatest pioneer of cerebral function, Dr. Hughlings Jackson, in 1866, and towards the end of the last century many observations were recorded by Pick, Wernicke, von Monakow, Westphal, and others; but it is only within the last few years and as the result of the labours of the German School that the profound significance of apraxia in our conceptions of cerebral function and localisation has been realised. From the confusion of symptoms that were at first known collectively under the term apraxia a separation has been arrived at, strictly comparable to Wernicke's separation of aphasia into motor and sensory aphasia, apraxia corresponding with the former and agnosia with the latter.

Dr. Hughlings Jackson originally used the term "imperception" to denote the failure of object-recognition presented by his patient. Freud in 1891 suggested the word agnosia as more descriptive of this condition. The term "apraxia" was first used by Kussmaul, Starr, and Pick in 1892; but it is plain from their descriptions that they included the phenomena of agnosia and of astereognosis in their conception of apraxia. Finkelnburg in 1870 used the word "asymboly" to denote inability to understand certain conventional signs, and Wernicke adopted this terminology and elaborated it to mean the loss of the memory pictures of an object. Meynert in 1890 separated asymboly into "motor asymboly" and "sensory asymboly." He argued that in various cortical centres there is for one and the same object a symbol, a sign by which the object can be recognised. The loss of the sign of the object is asymboly. On the motor side the use of the object is associated with this sign of recognition so that asymboly reveals itself in the patient's inability to make use of the object. A lesion of the central convolutions which hinders the awakening of kinaesthetic memories of the upper extremity, for example, will produce motor asymboly of that limb. Meynert's sensory asymboly is identical with agnosia and his "motor asymboly" is akin to, but not identical with, the present conception of apraxia. He was the first, however, to suggest a localisation for apraxia and to suggest that the condition might be confined to one limb. The great advance in our knowledge of this subject has occurred since 1900 owing to the researches of Liepmann, who has put upon a sound clinical and pathological basis his conception of a true motor apraxia apart from any condition of agnosia, that is, a defect in the use of objects though they are perfectly recognised. To this "motor apraxia" the term apraxia is now confined.

Pathology.—The investigations of Liepmann, Maas, van Vleuten, Hartmann, and Abraham have resulted in the attribution of the phenomena of apraxia to lesions of the posterior parts of the three frontal convolutions on the left side and to disease of the outgoing tracts from these gyri, and in an important degree to disease of the anterior part of the corpus callosum. The hypothesis has therefore arisen that, just as the posterior part of the left third frontal gyrus (Broca's area) is the centre for the subjectively purposive movements of both sides of the tongue, lips, etc., which are concerned in articulate speech, so also are the posterior ends of the second and first frontal gyri of the left side the centres for the subjectively purposive movements of the limbs upon both sides of the body. Disease of the anterior part of the corpus callosum severs the connecting path between these higher motor centres in the left frontal lobe and the right motor area, and left hemiapraxia is the result.

Apraxia may occur as the result either of general or of local disease of the brain. Amongst general cerebral diseases, general paralysis of the insane, cerebral sclerosis, and the several forms of dementia are important. In these cases the apraxia is not of the pure type and usually depends upon or is associated with agnosia and accompanied by general mental deterioration. Dr. S. A. K. Wilson has recently pointed out the presence of apraxia in cases of paralytic chorea. It is as a result of local lesions of the brain that uncomplicated motor apraxia is met with, and from such lesions only is it found to be confined to one region of the body. The lesions are situated in the anterior part of the hemisphere involving the posterior ends of the frontal gyri and they are much more frequently in the right than in the left hemisphere in right-handed people. In the posterior part of the left third frontal gyrus (Broca's area) there is a centre in which the movement-complexes of the tongue, throat, etc., requisite for speech are combined prior to the actual incitation of the cortico-muscular apparatus which takes place in the senso-motorium proper. Destruction of this centre or of its ingoing or outgoing connexions produces apraxia of the speech musculature or motor aphasia. There is evidence to prove that in the posterior parts of the left first and second frontal gyri analogous centres exist for the co-ordination of certain movement-complexes of the limbs of both sides, and that the movements of all four limbs are here combined ideationally to be subsequently innervated in the senso-motorium. Destruction of these centres entails bilateral apraxia. Destruction of the connexion of these centres with the right hemisphere (especially a lesion of the anterior part of the corpus callosum) results in left hemiapraxia, whilst a lesion separating these centres from the left motor cortex produces right hemiapraxia. The production of motor aphasia by cortical, transcortical, and subcortical lesions is here paralleled by the production of apraxia of the limbs by cortical, transcortical, and subcortical lesions. It is important to realise in our conception of apraxia that just as word-blindness and word-deafness (loss of recognition of visual and auditory word-symbols) cause aphasia, so does the loss of recognition of an object and of its uses or the loss of comprehension of an act entail apraxia. The terms sensory apraxia, sensory asymboly, and agnostic apraxia have been suggested for these phenomena but they have been discarded in favour of "agnosia."

Clinical Features.—Apraxia may be summarised as an inability to convert a correct idea of an action into the corresponding movement. A case of left-sided hemiapraxia is well suited for the demonstration of its features. There is neither loss of power nor loss of sensibility nor inco-ordination in the left upper extremity. When such a patient is commanded to perform some familiar act with the right upper limb he at once performs the act correctly, but when ordered to perform the same action with the left upper limb he is unable to do so. Either he makes aimless wandering movements with the left hand or he may succeed in making a movement somewhat resembling that which is required of him with much slowness and clumsiness. Sometimes he may perform some act which is entirely different to that required of him; this phenomenon has been called parapraxia. When the degree of the apraxia is partial the patient may be able to perform some acts and unable to perform others, his inability usually but not always increasing with the complicity of the act required. Or he may sometimes be able to perform an act in which he more commonly fails. Not unfrequently such a patient, wearied with the unsuccessful attempts of his left hand, will abruptly perform the act correctly with his non-apraxic hand to get rid of it. Sometimes at the end of an unsuccessful endeavour he will succinctly define his defect by saying, "I know quite well what you want me to do but I cannot do it." The performance of the required act by the observer standing in front of him does not increase his ability. Spontaneous volitional movement is similarly affected, and according to the degree of the apraxia the most highly-skilled movements are the first to be lost and the last to be regained if recovery takes place. For example, a violin-player affected with slight left hemiapraxia may be entirely unable to finger his instrument, though all ordinary actions of the left hand may be performed correctly. With this defect in the performance of volitional movements is associated invariably a remarkable loss of initiative as regards the use of the limb—the patient will not try to use it. The apraxic is often, to an astonishing extent, unaware of his disability and frequently becomes conscious of it for the first time when it is pointed out to him by another person. For instance, a patient who had been skilled in boxing was affected with apraxia of the left arm. When asked by his physician to stand up and spar with him, he at once arose with evidence of confidence and pleasure, and placing his feet, body, and right arm in the correct attitude, commenced to box with his left upper extremity swinging helplessly from the shoulder. He then for the first time became aware of his incapacity.

Apraxia in the region of the face is commonly seen in cases of right hemiplegia with aphasia. Such patients may be unable voluntarily to close the eyes, or to protrude the tongue, or to sniff, or to cough, though these actions are constantly performed subconsciously or as associated acts. Motor aphasia is apraxia of the speech-musculature.

Diagnosis.—Apraxia may be confused with astereognosis, with agnosia, or with cortical ataxia. A correct conception of the nature of the two former conditions will exclude the possibility of error. In cortical ataxia the patient obeys the word of command at once and succeeds more or less with the required act, the defect being clumsiness of execution. The clinical examination of patients for apraxia must include (1) the general psychical condition as regards attention, memory, capacity for retaining impressions, reasoning, association of ideas, delusions, etc.: (2) an inspection of the psycho-sensory phenomena; the presence of any defects of simple perception in the regions of sight, smell, hearing, taste, cutaneous sensibility, and muscular sense; defects of recognition of sensory impressions in these regions (agnosia); defects of memory of, and of ability to recall former sensory impressions in the same regions: (3) an examination of the psycho-motor phenomena; any defects in the movements determined by visual, auditory, tactile, and kinaesthetic stimuli. What response does the patient make to objects held in front of him or to gestures made to him? Can he imitate movements? Can he when requested make simple movements, movements of expression, and purposive movements with and without the objects in his hands? When given an object, how does he hold it and how does he use it?

Agnosia.—In certain conditions of cerebral disease it is found that each or all of the sensory organs when called into play may fail to arouse an intelligent perception of the object exciting them. For this inability to recognise the import of a sensory stimulus the term agnosia is employed. The varieties of agnosia will correspond therefore with the several senses, auditory, visual, olfactory, gustatory, and tactile agnosia. Visual agnosia is subdivided into verbal agnosia (word-blindness) and object agnosia (inability to appreciate the nature and use of objects though the objects are seen). Similarly auditory agnosia comprises verbal agnosia (word-deafness) and inability to appreciate the nature of sounds though the sounds are heard. Munk has shewn that agnosia is produced by lesions involving the cortex of the posterior halves of the hemispheres upon the external surface and more especially by lesions of the outer surfaces of the occipital lobes. A further localisation is only possible as yet in the case of verbal agnosia (word-blindness and word-deafness) which is dependent upon a lesion of the chief speech-centre situated in the left temporo-occipital cortex (Wernicke's zone). It is all-important to recognise when considering the phenomena of agnosia and of apraxia that the innervation of the higher cortical motor centres is incited largely from the sensory perceptive centres, destruction of which entails agnosia, and that agnosia necessarily causes phenomena of an apraxic order since the usual incitations which arouse the "eupraxic" centres in the left frontal convolutions are lacking. For example, if a patient suffering from tactile agnosia is blindfolded and a familiar object is placed in his hands with the command that he is to make use of it, he is unable to do so because he cannot recognise what the object is. If the patient in addition to his tactile agnosia has also visual agnosia he will be unable to put the object to its proper use even though his eyes be open. Similarly patients who have verbal agnosia (word-deafness and cortical word-blindness) necessarily present verbal apraxia (motor aphasia and agraphia respectively) since the centres in the left third and second frontal gyri no longer receive their customary incitations from the sensory perceptive speech-centre in Wernicke's zone.

In those who present the clinical manifestions of apraxia and agnosia certain other phenomena are often met with and are therefore important; these are (1) inattention, (2) defective capacity for retaining recent impressions, (3) lack of initiative, and (4) perseveration. The last of these phenomena consists in the repetition of an already executed movement when, and only when, the patient desires to make a fresh movement. A patient of Pick's, for instance, blew out a candle and then blew upon all objects presented to him during the next few minutes.

Perseveration in speech and writing are well-known phenomena in connexion with aphasia (*vide* p. 443).

The relation of apraxia and of agnosia among the disorders of cerebral function that cause impairment of voluntary movement cannot be better shewn than by a scheme which, passing along the cerebral sensory-psycho-motor path from sensory to motor, sets down in order these clinical conditions. The following scheme is from Dr. S. A. K. Wilson's monograph:—

1. *Cortical Blindness, Cortical Deafness, Cortical Sensory Paralysis.*—Loss of visual, auditory, and cutaneous ingoing impulses.
2. *Cerebral Ataxia.*—Loss of afferent kinaesthetic impressions, resulting in erroneous estimation of range of movement, power, etc.
3. *Mind Palsy.*—Incapacity of movement from loss of kinaesthetic images and memories for complex movements.
4. *Agnosia.*—Conservation of sensation but failure of recognition; loss of sensory memory images.
5. *Ideational Agnosia.*—Loss of the spatial associations and interconnexions which make up the idea of an object from its component ideas.

6. *Ideational Apraxia.*—Defective synthesis of the ideational components of a movement-complex; defective psychical sketch of movements or acts.

7. *Motor Apraxia.*—Intactness of the cortico-muscular apparatus, but inability to translate a normally produced idea of a movement into the corresponding movement-form.

8. *Cortical Motor Paralysis.*

JAMES COLLIER.

REFERENCES

1. ABRAHAM. *Allg. Ztschr. f. Psychiat.*, Berl., 1904, lxi. 502.—2. BERNHEIM. "Contribution à l'étude de l'aphasie : de la cécité psychique des choses," *Rev. de méd.*, Paris, 1885, v. 625.—3. FINKELNBURG. *Berlin. klin. Wchnschr.*, 1870, vii. 449.—4. HARTMANN. "Beiträge zur Apraxielehre," *Monatsschr. f. Psychiat. u. Neurol.*, Berl., 1907, xxi. 97.—5. HEILBRONNER. "Ein Fall von Asymbolie," *Allg. Ztschr. f. Psychiat.*, Berl., 1894, li. 1014.—6. *Idem.* "Über Asymbolie," Breslau, 1897.—7. *Idem. Ztschr. f. Psychol. u. Physiol. d. Sinnesorg.*, Hamb. u. Leipz., 1905, xxxix. 161.—8. *Idem.* "Über isolierte apraktische Agraphie," *München. med. Wchnschr.*, 1906, liii. 1897.—9. JACKSON, HUGHLINGS. *Med. Times and Gaz.*, 1866, i. 659.—10. *Idem. Roy. Lond. Ophth. Hosp. Rep.*, 1876, viii.—11. *Idem. Lancet*, 1878, i. 716.—12. KUSSMAUL. "Die Störungen der Sprache," Ziemssen's *Handbuch*, 1885.—13. LÉPINE, "Apraxie sans aphasie," *Rev. de méd.*, Paris, 1897, xvii. 452.—14. LIEPMANN. "Das Krankheitsbild der Apraxie," *Monatsschr. f. Psychiat. u. Neurol.*, Berl., 1900, viii.—15. *Idem.* "Über Apraxie mit Demonstration," *Neurol. Centralbl.*, Leipz., 1902, xxv. 614.—16. *Idem.* "Über Störungen des Handeln bei Gehirnkranken," Berl., 1905.—17. *Idem. Monatssch. f. Psychiat. u. Neurol.*, Berl., 1905, xviii., and 1906, xix.—18. *Idem.* "Uber die Funktion des Balkens beim Handeln und die Beziehungen von Aphasie und Apraxie zur Intelligenz," *Med. Klin.*, 1907, 725.—19. *Idem. Allg. Ztschr. f. Psychiat.*, 1907, lxiv. 450.—20. MAAS. "Ein Fall von linkseitiger Apraxie und Agraphie," *Neurol. Centralbl.*, Leipz., 1907, xxx. 789.—21. MEYNERT. "Klinische Vorlesungen über Psychiatrie," Wien, 1890.—22. PICK. "Über Störungen der Identification (Asymbole, Apraxie, Agnosie). Beit. z. Path. u. path. Anat. d. Centralnervensystems," Berl., 1898, i.—23. *Idem.* "Studien über motorisches Apraxie," Leipz., 1905.—24. VAN VLEUTEN. "Linkseitige motorische Apraxie : eine Beitrag zur Physiologie des Balkens," *Allg. Ztschr. f. Psychiat.*, Berl., 1907, lxiv. 203.—25. WESTPHAL. "Über ein Fall von motorischer Apraxie," *Allg. Ztschr. f. Psychiat.*, Berl., 1907, lxiv. 452.—26. WILSON, S. A. K. "A Contribution to the Study of Apraxia," *Brain*, Lond., 1908, xxxi. 164 (contains an exhaustive bibliography of the subject).

J. C.

THE CEREBRAL PALSIES OF CHILDREN

By JAMES TAYLOR, M.D., F.R.C.P.

THE cerebral palsies of children are divided into two great classes: (i.) those in which there is evidence of the presence of paralysis when the child is born, and (ii.) those in which after a period, more or less prolonged, of apparently normal health and unimpaired activity some form of paralysis appears. In the latter class the paralysis is, in the majority of cases, hemiplegic in form. In the former, both sides usually suffer; sometimes the legs only are affected, at any rate in a marked degree; sometimes both arms and both legs suffer; and in other cases one side only appears to be weak. Cases in which paralysis is present at birth are further subdivided into those in which the paralysis is the result of some difficulty or abnormality in the labour, and those in which it results from some condition arising during intra-uterine life; either primary disease or mal-development in the fetus itself, or some morbid condition secondary to disease in the mother, or to injuries which she may have sustained during her pregnancy. By far the largest number are the result of abnormalities in the labour, the abnormality consisting usually in the length and difficulty of the labour, due to deformity in the mother or to malposition of the child. Nevertheless an unusually quick or precipitate labour may likewise give rise to birth palsy.

BIRTH PALSY.—**Causes.**—In the great majority of the cases of birth palsy, as has been said, the lesion is determined by some fault in parturition. As a rule there is a history of a long and difficult labour, in many cases the result of an abnormal presentation of the fetus; and nearly always of a labour necessitating the use of instruments, or of turning. Often also the child is born cyanosed, and not breathing, so that artificial methods of promoting respiration have to be resorted to. Not uncommonly also the child is born before full term. But, as already pointed out in reference to these cases, any injury to the child occurring in a labour in which instruments have been used, or in which operative interference has been resorted to, is not necessarily to be ascribed to the instruments used or to the operation performed, but rather to those abnormal conditions which rendered interference necessary. Besides the long and difficult labour, the quick, or, in technical language, the precipitate labour also seems occasionally to have an effect in the causation of the condition under consideration.

As abnormal labours are much more common in primiparae than in multiparae, it is to be expected that birth palsy will occur much more frequently in first-born children; this is actually the case in so large a majority of instances as to place the fact outside the region of mere coincidence. It is also to be noted that in some cases in which the patient is not a first-born, it is a child born long after the preceding child. Two other points come out in a series of cases of this character seen by the writer; namely, that of children affected with birth palsy there are twice as many males as females; and that the child of an elderly primipara is more liable to be afflicted than is the first child of a younger woman. These two facts support the doctrine which ascribes the great majority of cases of birth palsy to mechanical difficulties in the process of parturition; for in the former case the larger size of the child, in the latter the greater rigidity in the maternal parts will tend to increase the difficulties of the labour.

There is some evidence that inherited syphilis is occasionally a factor in the production of this condition. It is unusual to see a child the subject of birth palsy who has any of the recognised signs of congenital syphilis; but it is quite possible, of course, that a syphilitic child may suffer from birth palsy if it has been exposed also to the causes of this condition: and it is likewise probable that inherited syphilis, through affection of blood-vessels or membranes, may occasionally give rise to a morbid process which causes in the child symptoms similar to those which we have under consideration.

Morbid Anatomy.—There is little doubt, from the cases which have been examined after death, that the condition upon which the paralysis depends is, in most cases, haemorrhage occurring under the dura mater, and so affecting the cortical structures as to abolish or interfere with their function. From what has been said as to the causation, it will be seen that such a condition is what we should expect; and there seems no doubt that its occurrence is due to rupture of vessels in consequence of the mechanical difficulties in the act of parturition. The site of this haemorrhage gives the clue to the distribution of the paralysis. As a rule the legs are more affected than the arms, sometimes they are affected alone. In such a case the haemorrhage will be found on each side of the longitudinal fissure, spreading in each cortex over the area in which the leg movements are chiefly represented. If we suppose the haemorrhage on one side to spread further, so as to destroy or impair the area in which the arm movements are represented, a condition would be produced in which both legs and one arm would be affected; if the haemorrhage spread still further, one side of the face would be affected; and if it spread on each side, so as to affect both arm areas as well as the leg areas, a condition would be produced giving rise to paralysis of both arms and both legs. In cases in which the speech function is in abeyance, either completely or partially, the lesion is of the third frontal convolution on both sides or only on one; and it will readily be understood that the ocular movements may be interfered with if the lesion spread forward so as to involve the area related to these movements; thus a variable squint may be produced. A varying degree of psychical defect is found to occur in children the subjects of birth palsy; and it may reasonably be assumed that this depends upon the degree of injury to that part of the brain which is probably most intimately associated with psychical processes—the prefrontal area.

This morbid condition is only distinctly recognised in cases in which death occurs in the early stage. Such, however, are not common although they are occasionally met with. There is, however, evidence of the frequency of intracranial haemorrhage in cases of stillbirth, and it is to be presumed that in many cases subjected to conditions identical with those present in cases of stillbirth, but not of such severe degree, similar haemorrhage is present, sufficiently severe to interfere materially with cerebral function but not of such severity as to cause death. Dr. Herbert Spencer describes the result of his examination of 130 bodies of stillborn children. In one there was haemorrhage into the substance of the brain. But haemorrhages into and beneath the membranes were found in a much larger number, namely fifty-three. Thus, bilateral haemorrhage over the convexity of the brain of this character was found in 29 cases, haemorrhage on the right side 10 times, on the left side 10 times. Haemorrhage at the base of the brain was found in 35 cases. In 6 cases the haemorrhage was limited to the base of the brain, there being no intracranial haemorrhage elsewhere. Haemorrhage into the ventricles was found 7 times. Harvey Cushing also examined a number of infants stillborn, or who had died during the early hours or days of life, and found that a considerable number had died with intracranial haemorrhage. And his surgical procedures in 4 cases furnish stronger and more direct evidence of the morbid anatomy of this condition. In the first case the twin child of a primipara of thirty-five suffering from convulsions and paralysis of one third nerve, an operation was carried out on the left parietal region and a considerable amount of blood-clot was removed. The child unfortunately died and no examination was permitted. The probability is that in this case, besides the haemorrhage over the convexity, there was also haemorrhage at the base. In the second case, also a first child born tediously by the breech, convulsions set in on the sixth day. An operation was performed and a submeningeal clot turned out from the parietal region. The child recovered, and at the age of a year was apparently normal. The third case was that of a large female child born asphyxiated after a long labour completed by forceps. In this case both sides were operated upon and clots washed away. The child recovered, and at the time at which the account was written was apparently developing normally. In the fourth case, that of a child born precipitately, signs of intracranial pressure developed after a few days; an operation was performed, and a large subdural clot was found, but the child died during the operation.

The evidence furnished by these cases seems to shew conclusively that many cases of birth palsy are certainly the result of subdural haemorrhage, and the probability is that most are the result of some kind of intracranial haemorrhage.

When death takes place after the lapse of some years, the condition discovered varies considerably. Sometimes it is what is known as porencephalus, a condition in which there is much cystic formation surrounded by cerebral substance more or less altered: sometimes there seems to be a sclerotic condition of the brain itself, and in other cases a condition resembling pachymeningitis, due, no doubt, to the effused blood having become organised or altered. In short, from the condition found in such cases it would be impossible to discover the nature of the original lesion were it not for the information gained from the examination of recent cases. It is also held by some writers that in certain cases, those especially in which the child is a premature one, the resulting condition is due to incomplete development of the pyramidal tracts. In such cases it is said that gradual improvement usually follows, and may go on to allow of an almost normal power of locomotion.

Symptoms.—In this form of paralysis there is a variable distribution of weakness. Sometimes the lower limbs are affected alone, equally, or unequally, producing the condition sometimes spoken of as "cerebral spastic paraplegia" or "congenital spastic paraplegia" ("Little's disease"). Sometimes the paralysis is of one side, affecting both arm and leg; sometimes both arms and both legs are affected; sometimes both legs and one arm only, or at least only one in a very obvious degree. In the limbs the weakness is usually associated with much stiffness. The gait, if the patient is able to walk, is characteristically spastic, each leg being dragged forward as a whole, and with considerable difficulty—"cross-legged progression"; if the upper limbs are affected there is, besides the weakness of the arms and hands, a great awkwardness in using them, probably occasioned by the spasticity. The knee-jerks are much exaggerated, but ankle-clonus is not so frequently present as might be expected; and in the case of patients suffering from this condition it is usually present only in those who are or have been able to walk. In patients unable to walk, in spite of the fact that a high degree of spasticity may be present, it is usually impossible to evoke it. The response evoked on stroking the sole is of the extensor type (Babinski's sign). In some patients the peculiar mobile spasm known as athetosis is present; and in patients in whom all four limbs are affected, athetosis affecting both sides—double athetosis—sometimes occurs. In this form of spasm there is usually a cycle of movements, one position slowly and gradually passing into another. Sometimes the hand only is affected, and in the great majority of cases it is most affected; but the spasm may affect all the parts of the upper limb, and may also affect the leg. It involves the head and the face in some cases, and the platysma muscle may markedly participate in the affection.

Besides athetosis, or mobile spasm, another form of involuntary movement is sometimes present, a form very closely resembling the tremor present in cases of insular sclerosis, in that it is only induced during voluntary movement. This tremor is well marked when an attempt is made to carry out a definite movement with the affected limb. In carrying the affected hand to the face, for example, at the beginning of the act the tremor is very slight; but as the hand approaches the face it becomes more and more distinct and even violent, so that it is impossible for the patient to keep the hand there in a position of rest. This condition has been unfortunately named *chorea spastica*, just as athetosis has been as unfortunately named *post-hemiplegic chorea*. In neither form of tremor is there, as a rule, anything more than a distant resemblance to the movements of true chorea, although certain cases of so-called hereditary chorea have undoubtedly been cases of birth palsy with double athetosis. But both athetosis and "chorea spastica" are probably more common in cases of infantile hemiplegia than in cases of birth palsy. In some cases spontaneous tremor, not uniform or rhythmical, is present, although this also is usually increased by movement. Sometimes it is so severe as to throw the patient to the ground when an attempt is made to walk without support.

Paralysis or paresis of some ocular movements is also not infrequently met with; and this occasions a squint of varying character and intensity, apparently not the result of an affection of one muscle in one eye, but of a slight affection of the different movements in each eye. The sphincters of the rectum and bladder are only affected to the extent of being less easily controlled than usual. In patients in whom there is much psychical change, the calls are unattended to, and the habits are consequently dirty. Even in these cases, however, this is only the rule in the early years of life. There is rarely any affection of swallowing, although slobbering is not uncommon.

The *mental condition* varies much in different patients. In those in whom all four limbs are affected there is, as a rule, much mental change; if the lower limbs only are paralysed the mental condition is frequently good; and in some instances the children are precociously sharp. Yet occasionally even in these cases there is much mental change. When the paralysis is hemiplegic in form the psychical condition varies, being very defective in some, scarcely abnormal in others. In some cases in which the paralysis is only slight, much change is present; a result probably due not to the nature but to the position of the lesion. In nearly all the cases mental development is retarded, and, as a rule, does not attain a high level.

Speech also is interfered with, and sometimes seems to remain entirely in abeyance; in nearly all its development is retarded, and generally it remains long imperfect. Anything like true aphasia is, of course, scarcely to be expected, for this can only arise from interference with the functions of a speech mechanism already developed, and in cases of birth palsy we have to do with centres the development of which has been interfered with or entirely arrested.

Recurring convulsions are not so common in cases of birth palsy as might be expected, possibly because of the completeness and limitation of the destructive process which has been present. But in many cases a history of a severe convulsion is to be obtained, or more often of a series of severe fits, occurring a few days after birth. Occasionally such convulsions recur at intervals during the first few years of life, and in a small number of cases this tendency persists during the whole of life.

Deformities are apt to occur in consequence of the interference with development and the malpositions encouraged by the form of paralysis. The most common deformity is some form of talipes; and, in patients in whom one side is weaker than the other, lateral curvature is almost inevitable, unless extreme care be taken. The reason of this is that the affected side does not develop equally with the other, the bones as well as the muscles sharing in this mal-development to some degree.

Diagnosis.—The diagnosis of the condition will not, as a rule, present many difficulties. The onset at birth although it may not be noticed until a few days after it, the not infrequent occurrence of convulsions at this time, and usually the history of some difficulty or abnormality at birth, are the points on which a diagnosis will depend.

Infantile hemiplegia may be similar in the physical condition, or in the psychical symptoms; but in this disease the onset is later, it comes on in a previously healthy child, it is ushered in with a convulsion or convulsions, and it is strictly hemiplegic. The spastic paralysis due to caries may closely resemble cerebral spastic paralysis; but in the former condition there has been a period during which the limbs were used normally. Of infantile paralysis also—acute anterior poliomyelitis—the same may be said; but the muscular wasting, flaccidity, and loss of reflex action and faradic irritability in the muscles in this condition will sufficiently distinguish it.

Prognosis.—Such a condition as that described entirely precludes any hope of complete spontaneous recovery. The degree of improvement which takes place naturally depends on the extent and situation of the lesion, and will be greater when this is limited to the leg areas on each side of the longitudinal fissure. A patient with this condition will be late in acquiring the ability to walk, and even at the best will always walk stiffly and imperfectly: but the arms will be useful limbs, and the mental and intellectual powers will not necessarily be impaired at all, may even be rather above the average. In cases in which the upper limbs are affected there is, as will be evident from what has already been stated, a much greater likelihood of mental impairment accompanying the bodily ailment; and where such mental defect is present the improvement will not be great, at any rate in reference to that particular symptom.

But physical improvement does take place in all these cases. Often, although the children attain the age of five or six before they can walk, many of them, in whom the prospect of walking seemed out of the question in an early stage, do ultimately walk, imperfectly it is true. In short, physical improvement takes place in all, but the degree of this can only be estimated by ascertaining actually how much impairment is present; mental improvement also takes place even in children apparently quite imbecile at first. But in cases in which mental impairment is present, a normal mental condition must not be hoped for, any more than a perfect physical condition is to be expected in any case.

Treatment.—A child affected with birth palsy naturally requires very great care. Good and intelligent nursing is essential: suitable food and warm clothing are even of more importance than in the case of ordinary babies. No effort should be spared in attempting to develop the physical and mental capacity; for much may be done in this way to increase these powers. But such training is apt to be discouraging because of the slow, almost invisible progress which takes place, and it is well, therefore, that the nurse should not expect very rapid progress. Gymnastic exercises, such as will assist in increasing muscular power, rubbing of the limbs and passive exercises are of much importance. Cod-liver oil, cream, and fat in an easily digested form are to be regarded as articles of diet rather than as medicines.

Attention also will be given to measures calculated to prevent deformities such as talipes or lateral curvatures; or, if they have already occurred, to the operative or mechanical treatment which will tend to correct them, or at least to minimise their effect.

The surgical procedures already alluded to, which have thrown fresh light on the morbid anatomy of this condition, also inspire a more hopeful view as to the future of some of these cases. Cushing has shewn that such interference is clearly indicated in certain cases, and with more frequent resort to operation it is not improbable that fresh light will be thrown on their nature and also their symptomatology in the early stages. For it must be acknowledged that such cases frequently come under observation when it is too late to hope for complete success from operation. And in the early stages, unless there are convulsions or very obvious paralysis of some kind, there may be nothing to raise a suspicion that anything is seriously amiss. On the accoucheur will devolve the responsibility of carefully examining every child whose entrance into the world has been attended with any unusual difficulty, in order to see whether any paralysis be present or anything indicating the probability of submeningeal haemorrhage. The next few years will probably witness considerable advance in our knowledge of this condition in its varied phases, and also, it is to be hoped, in the means of dealing successfully with them.

CEREBRAL PARALYSIS OCCURRING AFTER A PERIOD OF NORMAL HEALTH.—**Diplegic conditions.**—The cases of cerebral paralysis in children in which the paralysis occurs after a period of normal health and development form an extremely complex group, in which, however, there is evidence of a division into two large classes: (i.) those in which the onset of the weakness is slow and unaccompanied with much constitutional disturbance, and (ii.) those in which symptoms suddenly appear and are ushered in with much constitutional disturbance and usually with convulsions. In the former class, as a rule, the weakness is paraplegic; in the latter, hemiplegic—the ordinary infantile hemiplegia; yet in many of the cases in which the symptoms indicate bilateral weakness there is present before the onset of symptoms much constitutional disturbance, and also convulsions, antecedents and accompaniments similar to those present in cases of infantile hemiplegia. In some also the symptoms follow an acute disease such as scarlet fever, measles, mumps, pneumonia, enteric fever, diphtheria, and influenza. In the cases in which the symptoms come on gradually there may not be any antecedent disturbance: in some there is a history of head injury trifling in degree and probably without any definite relation to the onset of the symptoms. The cases in which *amaurosis* is associated with paraplegia form a definite group and are described separately (p. 468).

Various classifications of these cases of diplegia have been attempted. So far it has been impossible to adopt any really scientific classification based upon morbid anatomy, and the clinical appearances and symptoms have therefore dominated all such attempts. Yet these clinical types so constantly overlap that it is impossible to accept them as the basis for a really useful classification. Thus, the distinction between general rigidity and paraplegic rigidity is probably an artificial one, as it must be assumed that both clinical conditions depend upon the same anatomical changes differing in their distribution; and so also with the attempted distinction of a group of cases of "bilateral athetosis," which never exists without paralysis of some degree and not infrequently this is considerable. The same is true with regard to psychical changes: in many cases of diplegia the mental condition is extremely good; in many it is profoundly deteriorated; this is also seen in cases of infantile hemiplegia of acute onset, and this difference is no doubt determined by the degree to which changes are present in or absent from the prefrontal areas—changes of the same character as those in other parts, the affection of which gives rise to paralytic symptoms.

We have thus, in the class of cases under consideration, a period of apparently good health before any signs of definite weakness are obvious. In some of them there is a history of some peculiarity in the labour and of long-continued ill-health in the mother during the pregnancy. When such is the case it is not uncommon to find that more than one member of the family is affected with the rigidity, sometimes paraplegic sometimes diplegic. Occasionally it is impossible to exclude the influence of congenital syphilis: in a few cases definite stigmata of this disorder, disseminated choroidal change or interstitial keratitis, are present and no doubt stand in causal relationship to the basis of the paralytic symptoms.

The weakness then in one of the cases of this class may come on slowly and insidiously without any constitutional disturbance, first of all causing weakness in the lower limbs, gradually spreading so as to implicate the upper limbs. Even the movements of the face and head may be affected, and there may be superadded some involuntary athetoid movements chiefly in the upper limbs and the head and face. The presence of these athetoid movements combined with the rigidity may give rise to the assumption of various attitudes depending upon the degree to which the different groups of muscles are affected; for example, the cramped attitude, the attitude of adoration, the extension-supination position, etc. The gait is stiff and difficult, *pes cavus* is present, and the patient in walking assumes a gait like that of a tardigrade animal. The progression is slow, the feet are moved with difficulty from the ground, and each is moved in a segment of a circle so as to get in front of the other. In this way a characteristic cross-legged mode of progression is assumed. There may be a similar affection of the upper limbs; the stiffness may then not be so obvious, but it usually results in a certain degree of difficulty in carrying out fine movements, such as buttoning and writing. Squinting also may be present, and has been ascribed to an affection of ocular nuclei. It is much more probable, however, that it is the result of the morbid process spreading so as to affect the centres controlling ocular movements. The knee-jerks are exaggerated, ankle-clonus is usually present, and the plantar reflex is of the extensor type.

In many cases, however, the onset of the condition is ushered in by some acute illness with malaise, high temperature, and perhaps delirium. Convulsions also may be present, and, as will be seen later, the whole clinical aspect closely resembles that in the early stage of infantile hemiplegia. The development of the disability is much more rapid in such cases, the patient being immediately paralysed, although there may be a slight tendency towards subsequent improvement. The distribution of the paralysis may be similar to that in the cases of gradual development, similar ocular difficulties may be present, and the reflexes are similarly exaggerated and altered. Perverse involuntary movements may also occur; indeed, they are more likely to be present in such a case than in one of slow development. The mental symptoms may be present in varying degrees from merely a slight mental change inducing abnormal precocity, to the most profound dementia. The degree of this no doubt depends upon the extent to which the prefrontal lobes are affected.

The **etiology** of these conditions is exceedingly obscure. The two different classes described, the one in which the onset is gradual, the other in which it is sudden, are probably determined by different conditions. In the former we are probably dealing with a class of cases in which there is a congenital endowment of defective vitality, and after a certain period of normal development a break-down occurs and then we have the slow, gradual development of motor defect resulting from impaired nervous structure. That this is an inherent defect, although not obvious at the time of birth, is shewn by the occasional occurrence of the condition in more than one member of the same family, and by the frequency of a history of illness in the mother during pregnancy. In one case known to me two children born of a mother with disseminated sclerosis became paraplegic in the early years of life. In the other cases, in which the diplegic symptoms appeared after an acute illness, it is possible that in some of these also the acute illness was only the determining condition of the diplegia—the last straw in an already overloaded nervous system. In many, however, it is probable that some toxic condition determined the changes in the nervous system.

The **morbid anatomy** of these conditions varies. In all there is destruction of certain elements, usually cortical, of the nervous system. In some there is a condition of what has been called atrophic sclerosis—decay and death of nerve cells and fibres, and subsequent overgrowth of neuroglia. The distribution of the sclerosis varies with the symptoms; in cases of paraplegic rigidity it is limited to the paracentral lobes and neighbouring convolutions; in cases with mental symptoms the change extends forward, and when the upper limbs are affected the sclerosis is still more widespread in the Rolandic areas. This sclerosis leads to a gradual shrinking of the brain-tissue and a consequent widening of the sulci, resulting in a condition resembling a peeled walnut—the so-called "walnut brain." The underlying change, as has already been stated, is a decay and disappearance of nerve cells and fibres, and a subsequent overgrowth of neuroglial tissue. The decay of nervous structures is not consequent upon overgrowth of connective tissue but precedes it, the overgrowth of neuroglia being compensatory. This change renders it probable that the condition is primarily determined by some poison which affects nerve cells and fibres.

But in some cases porencephalic defects are found to exist, and the initial condition is then probably a vascular one—either thrombosis, which may readily occur, especially in marantic cases, or haemorrhage, single or multiple. The condition determining these haemorrhages are obscure. It is possible that some infective conditions may give rise to several inflammatory foci, and this may result in porencephalic defects. The morbid condition and process would thus be similar to that probably present in infantile hemiplegia, except that in the latter there is a single focus of inflammation, whereas when there are several porencephalic defects there have probably been several foci of inflammation. In other cases such defects, or even patches of sclerosis, have seemed to be the result of multiple embolism.

The **diagnosis** of the condition is not easy. In the acute cases the fever, general malaise, and convulsions not infrequently suggest meningitis, and the head retraction which is occasionally present appears to confirm this view. It is only where these initial symptoms have subsided, and the child is found to be more or less paralysed on each side, that the true nature of the condition becomes obvious. The condition then may closely resemble paraplegia from spinal caries; but the history of the onset, the frequency of mental affection, and the absence of angular curvature will usually be sufficient to allow a distinction to be made. This distinction is more difficult in cases in which the paraplegia is of gradual onset without severe constitutional disturbances at the commencement. A correct diagnosis can then only be arrived at after a careful consideration of the general state and the condition of the spinal column. The spinal form of infantile paralysis is distinguished, not so much by the mode of onset, which may be strikingly similar, but by the flaccidity of the paralysis in the spinal form as contrasted with the rigidity of the cerebral, and the excess of reflexes in the affected limbs in the latter as compared with their diminution or absence in the former.

The **prognosis** in all such cases is bad, and nothing more than arrest of the process can be hoped for. But even if this be effected, the patient is left in a condition of grave disablement, mental, it may be, as well as physical, and is scarcely likely to be in a condition to earn a living. In many cases also the process is progressive, gradually inducing more and more disablement, until some intercurrent trouble—very frequently bronchopneumonia—brings life to an end. In regard to **treatment**, drugs, unfortunately, are useless, except in so far as the bromides may help in controlling convulsions. The convulsions, fortunately, are amenable to these drugs, and, as a rule, yield to treatment. Gentle massage to the limbs may minimise the rigidity, and will often make the patient more comfortable. But apart from these measures, and the strict observance of the hygienic measures so necessary in nursing a helpless and often imbecile patient, no treatment is likely to afford relief.

Amaurotic Family Idiocy, *vide* p. 468.

Infantile Hemiplegia.—Infantile hemiplegia is the name applied to that paralysis of one side of the body which occurs in the first six years of life. Although in its general symptoms, except those occurring at the onset of the attack, it closely resembles the hemiplegia of adults, yet there are some distinct differences. The majority of these, however, are to be referred to the early age at which it occurs, and the consequent interference with development both of the brain and of the affected limbs. A distinction must be drawn—not so much as regards symptomatology, but in reference to pathology—between cases of infantile hemiplegia proper, which is probably a peculiar disease, and cases of hemiplegia occurring in children in connexion with acute disease, especially with rheumatism, scarlet fever, diphtheria, or enteric fever.

The hemiplegia of infancy occurs during the first six years of life, but much more frequently in the first two years than later. Of 42 cases, 19 occurred in the first year, 16 in the second, 3 in the third, 3 in the fourth, and only 1 in the fifth. The illness begins as a rule with general malaise and feverishness, and this culminates in a severe fit, or a series of convulsions. These are generally unilateral, but may spread so as to affect both sides. They may persist with but slight intermission for several days; and after the fits have ceased it is found that the child is paralysed on the side on which the convulsion was most severe, or to which it was limited. The resulting paralysis is permanent, although in degree it may be modified; and as a rule a considerable improvement from the condition immediately subsequent to the fit is to be looked for. If speech were present at the time of onset of the fits, it is usually interfered with at first, being either completely lost or rendered much less perfect than it had been before. Anything like the true aphasia of adult life is rarely if ever present. Permanent speechlessness occurs in some cases, even in children who had already acquired the faculty of speech more or less imperfectly. Permanent aphasia practically never occurs

As a rule, then, there is, after the convulsion or series of convulsions is over, paralysis of one side of the body, affecting face, arm, and leg. The facial affection is usually slight, rarely permanent. The arm is generally more affected than the leg; the knee-jerk is exaggerated, and ankle-clonus is of frequent occurrence. Rigidity is not usually so extreme as in adult cases; but some features exist in infantile hemiplegia which are at any rate much less common in adults. Amongst these, probably the most striking is what is sometimes spoken of as atrophy of the affected side. But this is obviously an erroneous term to use, and the condition is more correctly described as mal-development of the affected side. In a large proportion of cases the smaller size of the structures on the affected side is striking. This defect in development affects not only muscles and other soft structures, but, in a well-marked case, the bones also are much smaller than the corresponding structures on the unaffected side. The arm is usually more strikingly affected than the leg, and this is in accordance with the usual distribution of the paralysis. Not infrequently, when the defect in development is so slight as to be doubtful, a comparison of the two shoulder-blades will make it evident that one is distinctly larger than the other. In rare cases only is the difference in size between one side of the face and the other distinct and striking. The dependence of this feature of infantile hemiplegia on the central lesion is probably indirect. The defective development of the structures on the affected side is more probably the result of the disuse which the paralysis enforces than of any direct neurotrophic influence.

Another condition found in infantile hemiplegia—not so frequently as to be pathognomonic, but much more frequently than in other varieties of hemiplegia—is a form of tremor, the so-called mobile spasm or *athetosis*. This occurs in a small proportion only of cases of infantile hemiplegia (probably about 10 per cent); but it is much more frequent than in hemiplegia occurring in the adult. Indeed, its occurrence in adults, unless the hemiplegia date from an early age, is uncommon; and in most of the cases in which its onset has taken place during adult life it has been in patients in whom the paralysis was the result of accident. Its presence may lead to hypertrophy of certain arm muscles. This form of movement has been already described under birth palsy. In that section also a curious form of tremor, sometimes named "chorea spastica," closely simulating the tremor of disseminated sclerosis in that it is evoked only during voluntary movement, has been mentioned. Of 42 cases of infantile hemiplegia 2 manifested this peculiar tremor; but probably it does not occur nearly so frequently as this proportion would seem to indicate.

As I have said, this condition is usually ushered in with a series of fits beginning on the side subsequently paralysed. Frequently these fits recur at intervals, sometimes short, sometimes prolonged. They are, as a rule, unilateral, affecting the paralysed side. Some observers, however, have stated that the fits affect the non-paralysed side. It is conceivable that this may be the case if the paralysis is severe; whilst the paralysed side may be that affected in the fits only when the paralysis is less complete. The fits are usually accompanied with loss of consciousness, and consist of tonic and clonic spasm, with occasional tongue-biting and relaxation of the sphincters. But sometimes the attacks are of the nature of *petit mal*, and consist in momentary loss of consciousness without convulsion. The cases in which this occurs are cases in which the paralysis is slight. In one the attacks, which occurred frequently, were succeeded by the condition known as automatism, in which the patient, while still unconscious, performed apparently purposive acts. Such a condition is, of course, not only of great interest, but of the highest medico-legal importance (see art. "Epilepsy and Insanity," p. 941).

Besides the evidence of interference with physical functions, in a certain number of cases there is some mental deficiency. Often this is considerable in cases in which the physical functions are but slightly interfered with; and in such cases the mental disturbance not infrequently assumes the form of restless and irresponsible activity. In other patients a dull and apathetic condition of dementia is present; and in them the physical condition is frequently one of considerable, sometimes profound, unilateral weakness. But no constant relation is to be found between the physical and mental impairment, and where the latter exists there is probably damage in the prefrontal region. It is easy to imagine a condition in which it would be considerably affected without any very noticeable degree of interference with motor functions.

Besides these cases of what may be called infantile hemiplegia proper, other cases of hemiplegia of cerebral origin are met with in children, occurring after acute diseases, especially scarlet fever and diphtheria. Such a condition is the result of some intercurrent complication of these diseases. In the former disease it is probably due to embolism, a result of associated endocarditis; in the latter the same process has been alleged, but in 6 cases seen by the writer there was no evidence of any endocarditis; and thrombosis or haemorrhage is to be regarded as a more likely cause. Hemiplegia also sometimes follows enteric fever in a child. As blocking of veins in other parts is not uncommon in this disease, a similar process in the cerebral veins is to be regarded as the probable cause of the palsy. The hemiplegia in the cases of this nature is not generally associated with fits, nor with any profound psychical change, but is accompanied by mal-development of the limbs on the affected side. In regard to diagnosis, prognosis, and treatment, what is to be said in reference to ordinary cases equally applies to cases of this nature.

Pathology and Morbid Anatomy.—The ultimate pathology of this condition is still a matter of conjecture. Two views are held with regard to its morbid anatomy. According to Strümpell and others, the condition of infantile hemiplegia is a result of an acute inflammation of the grey matter of the cortex, analogous to, and perhaps identical with, that in the grey matter of the spinal cord which underlies acute poliomyelitis. This condition he names *acute polioencephalitis*; and if his view be accepted, the ultimate pathology would probably have to be looked for in some infective process (*vide* p. 241). The other view regards the condition of the nervous system as secondary to some vascular obstruction; and this obstruction, according to Sir W. Gowers, is to be looked for in the veins of the surface of the cortex. The probability is that both these hypotheses are true of different cases, and it may be possible later to distinguish the two groups by the clinical features, especially those attending the onset of the condition. In cases dying during the acute stage, areas of inflammation and softening have been found. In cases dying long after the onset, the condition found has been either one of cystic formation (porencephaly), with much atrophy of the affected hemisphere and of the correlated parts of the nervous system, or one of sclerosis of the hemisphere; and both these conditions are, of course, compatible with the initial lesion formulated in each hypothesis. Nothing definite can be said as to the cause of the athetosis present in a number of the cases. It is probably a result of imperfect destruction; but whether in the cortex or in the deeper structures it is still impossible to say. Even the case published by Beevor and Horsley, in which removal of a cortical area in an adult brought about a cessation of the athetosis, is not conclusive.

The **diagnosis** is not ordinarily difficult. A case of birth palsy may closely simulate the clinical condition of infantile hemiplegia; but the history of weakness actually at the time of birth, although only first noticed a few days later, will usually be sufficient to indicate the category to which a given case is to be referred. From infantile spinal paralysis the state of the reflexes and the absence of any electrical change in the muscles of the affected limbs will sufficiently serve as a distinction. Some cases of myopathy may present a superficial resemblance; but the hemiplegic character of the affection, and the presence even of excess of the knee-jerk, as well as the history, especially of convulsions, will usually be sufficient indication to allow of a correct diagnosis being made.

Apart from the danger which the weakness and the liability to convulsions imply, the **prognosis** is good, so far as life is concerned. It is also found that the hemiplegic weakness is usually greater immediately after the onset of paralysis than it is ultimately, so that we may predict a certain degree of improvement. But the weakness will remain; the child will probably be backward, and may have a definite degree of mental weakness. The fits can usually be controlled if the patient is carefully, judiciously, and persistently treated. This, however, is only partly true of cases in which *petit mal* is present. But even in cases in which no fits have occurred, except the one convulsion or the series of fits at the onset, it must always be remembered that fits are prone to occur, although their occurrence may be delayed even for as long as several years. If speech has been impaired, the impairment is rarely if ever persistent; and sensory impairment is not found, although hemianopsia has been described.

Treatment.—Great care and patience must be exercised in cases of mental backwardness to develop the existing mental power cautiously and judiciously. The same is true of the defective physical power; and mild gymnastic exercises and passive movements will do much to increase the power which the patient retains. Massage will also assist in minimising the rigidity; but electricity is not of much use, and its application may do harm by causing an amount of discomfort and alarm quite out of proportion to any benefit likely to be derived. Convulsions will be treated like epileptic fits. They are usually amenable to ordinary treatment by bromides; but the attacks are sometimes of the nature of *petit mal*, which are much more intractable. Fresh air, good food, and the best possible hygienic conditions are, of course, essential.

MICROCEPHALY.—This condition deserves a brief notice. Cases of different kinds have been gathered under this name, but, so far as can be ascertained, only two varieties need be considered: namely, those in which the small or deformed skull is secondary to actual brain disease, such as the birth palsy or infantile hemiplegia just considered; and those in which the smallness of the skull and of the body generally is but a local expression of a general developmental defect in which the brain itself shares. The cases of the former class may be characterised by some local weakness or paralysis; in those of the latter the weakness is universal; in both classes the mental state is very backward. Some years ago operative treatment was recommended in such cases, the object being by removal of the cranial wall to relieve a hypothetical condition of pressure and consequent interference with brain growth. No results, however, have been obtained to justify such procedures; and the cases cannot be regarded as in any degree amenable to treatment other than the educative and disciplinary influences resorted to in the treatment of weak-minded children (*vide* p. 889).

JAMES TAYLOR.

REFERENCES

1. COLLIER, J. S. "Cerebral Diplegia," *Brain*, London, 1899, xxii. 373.—2. CUSHING, H. "Concerning Surgical Intervention for the Intracranial Haemorrhages of the New-born," *Amer. Journ. Med. Sc.*, Phila., 1905, cxxx. 563.—3. FREUD, S. "Die infantile Cerebrallähmung," Nothnagel's *Specielle Pathologie*, Band ix. 8vo, Wien, 1897.—4. GEHUCHTEN, A. VAN. "Maladie de Little et rigidité spasmodique spinale des enfants nés avant terme," *Rev. neurol.*, Paris, 1897, v. 65.—5. GOWERS, Sir W. R. *Manual of Diseases of the Nervous System*, 3rd edit., 8vo, London, 1899.—6. KINGDON, E. C., and RUSSELL, J. S. R. "Infantile Cerebral Degeneration with Symmetrical Changes at the Macula," *Med.-Chir. Trans.*, London, 1897, lxxx. 87.—7. LITTLE, W. J. "On the Influence of Abnormal Parturition, Difficult Labour, Premature Birth, and Asphyxia Neonatorum, on the Mental and Physical Condition of the Child, especially in Relation to Deformities," *Trans. Obstet. Soc.*, London, 1862, iii. 293.—8. MCNUTT, SARAH J. "Double Infantile Spastic Hemiplegia, with the Report of a Case," *Amer. Journ. Med. Sc.*, Phila., 1885, lxxxix. 58.—9. POYNTON, F. J.; PARSONS, J. H.; and HOLMES, G. "A Contribution to the Study of Amaurotic Family Idiocy," *Brain*, London, 1906, xxix. 180.—10. SACHS, B. *A Treatise on Nervous Diseases in Children*, 1895.—11. SPENCER, H. R. "On Visceral Haemorrhages in Stillborn Children; an Analysis of 130 Autopsies; being a Contribution to the Study of the Causation of Still-birth," *Trans. Obstet. Soc.*, London, 1892, xxxiii. 203.—12. TAYLOR, J. *Paralysis and Other Diseases of the Nervous System in Childhood and Early Life*, 8vo, London, 1905.

J. T.

AMAUROTIC FAMILY IDIOCY

SYNONYMS.—*Arrested Cerebral Development; A Family Form of Idiocy; Infantile Cerebral Degeneration; Agenesis Corticalis*

By J. S. RISIEN RUSSELL, M.D., F.R.C.P.

Definition.—A fatal disease of infancy characterised by progressive mental and physical enfeeblement, blindness, and symmetrical changes at the macula lutea which are pathognomonic.

History.—Attention was first called to this disease by Mr. Waren Tay in 1881; and since then a large number of cases have been published. Ophthalmologists have contributed many of these, as the peculiar and characteristic ocular changes form a prominent part of the clinical picture of the disease. To B. Sachs we are especially indebted for much of our earlier knowledge of the affection and the name that has become generally accepted. Sachs, Peterson, Hirsch, and Kingdon in this country were the first to obtain necropsies; the latter observer being particularly fortunate in that he was able to supplement his clinical observations by necropsies and subsequent microscopical examination of the nervous system in three members of a family of seven, five of whom were affected. Our knowledge of the disease has since been greatly advanced by the investigations of Spiller, Schaffer, Gordon Holmes, Vogt, and others.

Causes.—A curious circumstance, which appears to be of etiological significance, to which attention was first called by Carter, is that, of the published cases in which the nationality is stated, the patients have usually been Hebrews. Of 86 cases collected by Heveroch 61 were Jews. The children usually belong to poor families, often immigrants, such as Polish Jews in this country and in America and Germany. In addition to the racial peculiarity, there is distinct evidence that the condition belongs to the category of familial diseases; more than one child in the same family is, as a rule, attacked, though others may remain perfectly healthy.

Syphilis, tuberculosis, and hereditary diatheses do not appear to play any part in its production, and the same may be said of alcoholism; nor is there any evidence that consanguinity of marriage has any great influence in this respect, though it has been a possible factor in some cases. Dementia and psychoses have been recorded in the family history, and one or other parent has been neurotic in some instances. Injury during pregnancy has been noted, but this has been exceptional. No proximate cause after birth has yet been discovered, with the exception of injury in a few cases, but there is no real justification for the belief that trauma is in any way responsible for the disease.

Morbid Anatomy.—The cerebral and spinal meninges are usually free from any abnormal appearances; though in Sachs' first case a few slight adhesions of the former were noted. The convolutions and sulci of the brain are normally arranged; but in the case just referred to there was some abnormal fissuration which was regarded as indicative of a brain of low development, an abnormality present in Peterson's case also. The sulci are wide in some cases, pointing to a certain amount of atrophy of the convolutions; but this atrophy varies in different cases. The brain has sometimes been unduly firm to the touch, especially in the frontal region. The ventricles are normal, and do not contain any excess of cerebrospinal fluid; though there may be some compensatory oedema of the meninges. The essential change met with is degeneration of the cells throughout the central nervous system (*vide* Vol. VII. Plate IV. Fig. 2); they are swollen and often balloon-shaped, and some of them are even vacuolated. The nuclei become eccentric, and are often either very much shrunken or have practically disappeared. The cells of the cerebellum are much less affected than those of other parts of the nervous system, and indeed have sometimes been described as normal, as have also the cells of Clarke's column, whereas Dr. Gordon Holmes found the cells of the dorsal root-ganglion markedly affected. The axis-cylinders are degenerated, as are the neuro-fibrils in the cells, but the dendrites are said to be intact. Schaffer, however, finds that swelling of the dendrites is a special feature of the disease. There is well-marked degeneration of the pyramidal and cortico-pontine tracts, but the others escape. No inflammatory changes are seen, and such slight neuroglial proliferation as is sometimes met with is clearly secondary to degeneration of the nerve elements.

The ganglion-cells of the retina undergo degenerative changes similar to those of the central nervous system; they become swollen and often vacuolated, their nuclei are usually eccentric, and the tigroid disappears. The nerve-fibres of the retina also degenerate, but the cells of the nuclear layers remain unaltered.

Pathology.—While it is possible that the disease may be congenital in origin, neither the clinical history nor the morbid appearances met with after death supply any evidence in favour of this view. Nor do we find evidence that the affection is one of arrested development, as was at first advocated by Sachs; for there is abundant evidence that a progressive degeneration is taking place in the nervous system in these cases. Various hypotheses have been framed to account for the degeneration, but there is nothing to support the suggestions that a toxin or that some alteration of an internal secretion is responsible. It is, accordingly, supposed that an inherited peculiarity of the protoplasm of the cell renders it prone to early degeneration.

Symptoms.—Children who afterwards shew evidences of this disease are born at full term, and are then, as far as can be judged, perfectly normal, and so remain until the end of the third (as a rule), or it may be as late as the sixth month; up to which time development progresses naturally. The first symptom to attract attention is an inability to sit up properly, and to keep the head from falling backwards; in addition to these evidences of weakness of the muscles of the neck and back, there is often a suspicion that vision is impaired; but usually no definite ophthalmoscopic changes can be made out so early.

As the disease progresses the child becomes apathetic, takes little notice of what is going on around it, and wears the vacant expression of mental enfeeblement. It becomes less and less able to sit up, and quite unable to hold up its head, which consequently falls backwards. It lies in bed as it is placed, and has no power of moving from one position to another. Objects placed in its hands are grasped feebly and soon dropped. The muscles become soft and flabby, but in the only instances in which they have been tested electrically in the early stage of the disease they responded to both the interrupted and constant currents. Soon there is evidence that the pyramidal system is implicated; slight extensor or flexor spasm appears in the limbs, and the tendon-jerks become too active.

Later in the course of the illness the enfeebled muscles begin to waste; all the muscles of the body sharing in the atrophy, which eventually becomes extreme. To this muscular wasting, which is general and is not confined to groups of muscles, there may be added an increasing degree of spasmodic rigidity which causes retraction of the head and distortion of the different segments of the limbs,—resulting, for instance, in inward rotation of the arm, pronation of the forearm, flexion of the thigh on the abdomen with marked abduction of the limbs, over-extension with partial luxation at the knee, extension of the foot, and so on. As the contracture increases the child evidently suffers much pain, which is increased by the spasmodic jerking of the limbs that occurs from time to time. Death may result, however, without the occurrence of rigidity; or rigidity may have been occasional, no spasm being permanently present; so that spastic phenomena are not essential to the diagnosis. The mental deterioration is also progressive and ends in complete idiocy.

Convulsions are so rare that they form no part of the usual clinical picture of the affection, but have been observed as terminal manifestations of the illness. Cutaneous sensibility and the superficial reflexes are preserved, and the former has occasionally been found increased, so that the child starts at the slightest touch. The special senses, with the exception of vision and hearing, remain unimpaired throughout the illness. Sight fails gradually until total blindness results from optic atrophy, which has all the characters of being primary, and in the majority of cases is undoubtedly so; though Mr. Waren Tay has observed slight neuritis in the early stage of the optic nerve affection. In addition to this, ophthalmoscopic examination reveals a peculiar and distinctive appearance in the region of each yellow spot. At first a suspicious-looking haze is seen in the macular region; but subsequently a whitish-grey oval patch is seen, almost twice the size of the optic disc, with softened edges, and with its long axis lying horizontally, very slightly raised above the general surface of the retina. The fovea centralis is seen as a dark cherry-red spot in the centre of this patch.

Once established, these appearances remain unaltered throughout the whole course of the affection, and were present in Koller's patient, aged four years. Dr. Parkes Weber, however, has recently recorded a case in which a child, aged fourteen months, blind and presenting the other clinical manifestations of the disease, had neither optic atrophy nor the characteristic changes in the macular region. The parents were Jews, and at least two other members of a family of six appear to have been similarly affected.

There is no evidence that either the cranial nerves—other than the second pair—or their nuclei are affected, speaking generally. The pupils are equal, and, as optic atrophy becomes advanced, they are dilated and inactive to light. Strabismus has been noted exceptionally, as has nystagmus; but both phenomena must be rare: towards the final stages of the illness, however, slow rhythmical movements of the eyes from side to side may occur. The only other points calling for comment, as far as the cranial nerves are concerned, are that hearing sometimes appears to be abnormally acute, a phenomenon too frequently observed to be merely accidental; and there may be difficulty in swallowing for some time before the fatal termination of the case.

Apart from any accidental complications such as bronchitis and gastro-intestinal disorders, the thoracic and abdominal viscera are, as a rule, normal; the appetite good, the bowels regular, the urine natural, and the pulse, respirations, and temperature normal. In Sachs' first case, however, the gastric mucosa early refused to perform its functions properly; and some elevation of temperature has been noted in the terminal stages of a few cases.

The disease has ended fatally in most of the recorded cases, and in those in which this result had not been reached, the condition of the patients at the time of publication made it evident that there was not likely to be any departure from the general rule. The duration of life varies from one and a half to two and a half years, is usually less than two years, and may (quite exceptionally) be prolonged beyond this. The final stage of the affection sometimes resembles the same stage in cases of general paralysis of the insane. Death may occur before much emaciation, and without spasmodic rigidity of the limbs. But usually the child becomes pale and shrunken, more and more lethargic and exhausted until marasmus becomes extreme, consciousness is lost, and death closes the scene. In exceptional cases, instead of this more usual form of death from marasmus and gradual exhaustion, the termination may be unexpectedly sudden.

Spielmeyer, Vogt, and others believe that a juvenile form of the affection exists in which a previously healthy child becomes attacked between the ages of six and fourteen years, and in which there is the same progressive mental and physical deterioration resulting in death in a few years from marasmus, after the patient has become completely blind. The kinship suggested by the similarity of the clinical pictures is made still more probable by the fact that Schaffer finds a close resemblance between the histological changes in the juvenile and infantile forms. Higier, however, adduces sound arguments in favour of the view that these cases are distinct from the infantile affection with which we are now dealing, and argues that the cases described as the juvenile form do not shew the same racial predisposition or the characteristic macular changes.

Diagnosis.—The changes in the region of the macula lutea, when fully displayed, are so characteristic that any uncertainty as to the nature of the case can no longer exist. We do not know of any other condition in which progressive mental and physical enfeeblement are associated with these peculiar and pathognomonic changes. Even before the appearance of the macular changes the diagnosis is not difficult, though the affection may be then confounded with other infantile cerebral palsies. If a trustworthy history can be obtained, the pre-natal and natal affections are readily distinguished from this disease; the earliest evidences of which become manifest at the third month. It may be confounded with inherited syphilis; and the history of syphilis in a parent, and evidences of this disease, ocular or other, in the child must be sought for in attempting to arrive at a diagnosis. Ordinary congenital idiocy is distinguished by the absence of ocular symptoms and fundal changes, and such patients live for many years.

As regards its distinction from other post-natal cerebral palsies, and especially the familial forms of these, reliance must be placed on the absence of convulsions; on the exceedingly gradual and general invasion of the paresis, which does not assume the form of a hemiplegia or a paraplegia; and, above all, on the presence of blindness, with the characteristic fundal changes. The possibility of confounding this disease with acute poliomyelitis is too remote to deserve much attention; but should the question arise before the development of the changes in the fundi, the absence of altered electrical reaction of the muscles would exclude the spinal affection.

Prognosis.—With few exceptions the cases have ended fatally, and this usually within two years. In the vast majority of cases as soon as the diagnosis is certain the prognosis is equally so. Moreover, the occurrence of a case of the kind in a family renders it highly probable that any children born subsequently may become similarly affected; but fortunately this is not invariably the case.

Treatment.—No remedy appears to have any influence on the course of the disease, which is steadily progressive in spite of all the measures that have been tried for its relief. Mercury and iodide of potassium have both failed to influence its course; whether arsenic and similar remedies may prove more successful remains to be seen. If future researches should reveal any defect in connexion with any of the ductless glands in these cases, the administration, in some form, of the particular gland affected might prove of service. Attempts to prevent the occurrence of the affection by administering iodide of potassium to the mother while pregnant have been unsuccessful (Kingdon). This was tried in two pregnancies; one child escaped, the other was attacked by the disease. Some still advocate weaning, although the disease has developed in bottle-fed children, and Frank has supposed that there is a deficiency of lecithin in the mother's milk, and has therefore advocated its administration in the treatment of the affection.

The most careful nursing, feeding, and hygiene of such children are of course essential.

J. S. RISIEN RUSSELL.

REFERENCES

1. APERT et DUBOIS. *Arch. des méd. des enf.*, Paris, 1908, xi. 335.—2. BABONNEIX et BRELET. *Gaz. des hôp. de Paris*, 1908, lxxxi. 675.—2*a*. BING. *Ergeb. der innere Med. u. Kinderheilk.*, 1909, iv. 82.—3. CARTER. *Arch. f. Ophth.*, Leipz., 1894, xxiii. 126.—4. COHEN and DIXON. *Journ. Amer. Med. Assoc.*, Chicago, 1907, xlviii. 1751.—5. FRANK. *Ibid.*, 1906, xlvi. 187.—6. *Idem. Pediatrics*, N.Y., 1906, xviii. 148.—7. GOLDZIEHER. *Centralbl. f. Augenh.*, Leipz., 1885, ix. 219.—8. HEIMAN. *Arch. of Pediat.*, N.Y., 1897, xiv. 268.—9. HIGIER. *Neurol. Centralbl.*, Leipz., 1909, xxviii. 1065.—10. HIRSCH. *Journ. Nerv. and Ment. Dis.*, N.Y., 1898, xxv. 538.—11. HIRSCHBERG. *Centralbl. f. Augenh.*, Leipz., 1888, xii. 14.—12. HOLDEN. *Journ. Nerv. and Ment. Dis.*, N.Y., 1898, xxv. 550.—13. HUISMANS. *Journ. f. Psychol. u. Neurol.*, Leipz., 1908, x. 282.—14. ICHIKAWA. *Klin. Monatsbl. f. Augenh.*, Stuttg., 1909, n.F., vii. 73.—15. KINGDON. *Trans. Ophthal. Soc. U.K.*, Lond., 1892, xii. 125.—16. KINGDON and RUSSELL. *Med.-Chir. Trans.*, Lond., 1897, lxxx. 87.—17. KOB. *Charité-Ann.*, Berl., 1906, xxx. 239.—18. KOLLER. *Med. Rec.*, N.Y., 1896, l. 266.—19. KOPLIK. *Arch. of Pediat.*, N.Y., 1897, xiv. 736.—20. MAGNUS. *Klin. Monatsbl. f. Augenh.*, Stuttg., 1885, xxiii. 357.—21. MAYOU. *Ibid.*, 1904, xlii. 283.—22. MERZ. *Med. Klin.*, Wien, 1908, iv. 1952.—23. MOHR. *Arch. f. Augenh.*, Wiesb., 1900, xli. 285.—24. MÜHLMANN. *Verhandl. deutsch. path. Gesellsch.*, Jena, 1907, Tagung x, 100.—25. PETERSON. *Journ. Nerv. and Ment. Dis.*, N.Y., 1898, xxv. 603.—26. POYNTON. *Brit. Med. Journ.*, 1909, i. 1106.—27. POYNTON, PARSONS, and HOLMES. *Brain*, Lond., 1906, xxix. 180.—28. QUACKENBOSS. *Boston Med. and Surg. Journ.*, 1906, cliv. 238.—29. SACHS, B. *Journ. Nerv. and Ment. Dis.*, N.Y., 1887, xiv. 541.—30. *Idem. Ibid.*, 1892, xix. 603; *New York Med. Journ.*, 1896, lxiii. 697.—31. SARVONAT. *Rev. mens. des mal. des enf.*, Paris, 1906, xxiv. 70.—32. SCHAFFER. *Ztschr. f. die Erforsch. u. Behandl. d. Jugendl. Schwachsinns*, 1909.—33. SPIELMEYER. *Neurol. Centralbl.*, Leipz., 1906, xxv. 51.—34. STOCK. *Klin. Monatsbl. f. Augenh.*, Stuttg., 1908, n.F., vi. 225; *Ibid.*, 1909, n.F., vii. 83.—35. TAY, W. *Trans. Ophth. Soc. U.K.*, Lond., 1881, i. 56.—36. VOGT. *Arch. f. Kinderh.*, Stuttg., 1909, li. 1.—37. *Idem. Monatsbl. f. Psychol. u. Neurol.*, Berl., 1905, xviii. 161, 310.—38. WEBER, F. P. *Proc. Roy. Soc. Med.*, Lond., 1910, iii. (Child. Sect.) 58.—39. WILBRAND und SAENGER. *Die Neurologie des Auges*, Wiesbaden, 1909, iv. Part i. 396.—40. WORDSWORTH. *Trans. Amer. Ophth. Soc.*, N.Y., 1887, iv. 572.

J. S. R. R.

PARALYSIS AGITANS

SYN.—*Shaking Palsy; Parkinson's Disease;* Germ. *Schüttellähmung.*

By SIR WILLIAM R. GOWERS, M.D., F.R.S.

Features.—A shaking of the limbs when they are not moved by the will, sometimes when they are; a loss of the power that can be exerted by the will; a kind of stiffness which, although not often great, tends to fix the frame in a certain posture, which can be changed less speedily than in health, — these are the characteristics of the disease which Parkinson distinguished, and to which he applied the name of "Shaking palsy," "Paralysis agitans," previously used to designate random forms of disease. The name does not recognise the rigidity, which is a more frequent feature than either the tremor or the weakness; nevertheless, in the majority of cases the symptoms indicated by it are sufficiently prominent to make "shaking palsy" one of the best names that have been conferred on any disease. It is an instance of a name based on the simple features of a disease, independent alike of hypothesis that may be wrong and of circumstance that is accidental. It is a name that conveys knowledge, and has not the absolute blankness of a purely arbitrary designation. Charcot and others, because the designation is not entirely comprehensive, proposed to substitute the name "Parkinson's disease,"—to discard, that is, the excellent name which was proposed by its first discerner for one which conveys only a knowledge of an historical accident.

Shaking palsy is a disease chiefly of the early decline of life; but it is also met with in its middle period, and occasionally soon after the achievement of full development. It is a slow, progressive malady, apparently an early senile affection, a failure of nutrition of special character in special structures.

Etiology.—Some inherited influence can be traced in not more than a seventh of the cases, by the occurrence of the same disease, or of more simple forms of tremor, in other members of the family. The proportion of inheritance is the same in each sex. Men suffer twice as frequently as women (156 to 78). The disease begins most frequently during the earlier part of the later period of life. Onset before 30 is extremely rare, and was met with only twice in 236 cases of which I have notes. The rest were thus distributed:—30-39, 13 cases; 40-49, 51 cases; 50-59, 100 cases; 60-69, 60 cases; 70-75, 10 cases. The proportion beginning 60-69 is greater than appears from these figures, because the number of persons living is less by a quarter than in the preceding decade. The reduction of age is shewn by the last six years 70-75 (beyond which no cases of the series commenced). The number was 10, one-sixth of those commencing between 60 and 69 years of age, but the number living between 70 and 75 was more than one-half of those in the decade 60-69. Thus, there is clear evidence that the tendency to suffer does lessen with age, but not in the degree the figures suggest. The onset was during the forties in 51 cases, half that in the fifties, but the number living is a quarter more, so that the tendency is less than half that of the fifties, and during the thirties it is only a tenth, if a similar allowance is made. The age-incidence of women is practically the same as that of men, save that no cases were met with before 30.

The chief direct cause that can be traced is prolonged and depressing mental emotion. It is found in about half the cases; but some allowance must be made for the frequency of anxiety and grief at the time of life at which paralysis agitans generally begins. The influence of emotion receives emphasis, on which too much stress may perhaps be laid, from the fact that it may be distinctly related to the acute emotion of fear. The influence of fear is on the motor centres, to induce the movement necessary for escape, which seems to have made the term "movement from" a name for all such mental states—"emotions." If that movement be impossible, tremor results, and thus we have the word to "tremble" as a synonym for fear. The sudden tremor of alarm has been known to persist, and to assume all the characteristics of paralysis agitans. It may be through this mechanism that local injuries have been the immediate cause of the affection; although in some cases an influence on the nerve-centres from the injured part seems to be associated with it, for in this part the tremor has more than once been known to begin. In some cases it has begun in one arm soon after an injury to it.

In rare cases the disease has followed exposure to cold, acute diseases, and malaria; but the degree in which the malady has been the consequence of these antecedents is not clear. It is curious that the poisons which cause tremor do not seem to cause paralysis agitans.

Morbid Anatomy.—The most careful search has failed to reveal any microscopical changes peculiar to paralysis agitans. Those which have been found are such as are common at the time of life at which its subjects die, and alterations deemed significant in one case have been looked for in others without success. The most frequent are increase of the neuroglia of the spinal cord, especially in islets about the vessels and at the surface; changes in the anterior grey matter, with pigmentation of the motor ganglion-cells, and diminution of the dendritic network, and slight signs of degeneration in the fibres of the pyramidal and posterior tracts. In the brain, besides the necessarily frequent signs of arterio-sclerosis, degenerated Betz-cells have been found in the paracentral lobuli, but little else of importance. The cerebellum, locus caeruleus, and red nucleus have been specially examined with negative results. In the peripheral nerves an increase of interstitial tissue and slight signs of degeneration of the fibres have been the chief changes. The muscles have presented wasting of many fibres, increased longitudinal fission, a diminution in the muscle-spindles, and an increase in the nuclei of the sarcolemma, which have been thought to be unduly dark. None of these can be regarded as significant. The muscular changes have lately received special attention on account of the absence of definite alterations in the nerve-centres; the absence of other changes than those common to the old has been regarded as evidence that the disease does not depend on the nervous system. This conclusion is not warranted, nor is that which has been grafted on the muscular hypothesis—that they are caused by a toxic agent probably produced by the parathyroid glands!

Pathology.—When well developed, tremor is a definite alternate contraction of groups of muscles and their opponents, contractions that should be synchronous for support and steadiness. The alternation develops gradually from the slightest degree ; at first it is only perceptible as apparent arrests of contraction, and it increases slowly, as every established functional state increases by repetition. The contractions, which are thus affected, may be those which are volitional, when tremor is at first confined to voluntary motion, or it may be the tonic contraction which is normally a continuous state, independent of the will. The latter is seen when the tremor occurs at rest, and is often stilled for a short time by a voluntary movement.

For each element, the voluntary action and the continuous state, there are spinal and cerebral mechanisms. This is conspicuous for the voluntary action ; and, for the tonic state, the fact is revealed by functional cerebral diseases which cause an increase of the tonic contraction, such as catalepsy. But the cerebral influence acts through a spinal mechanism, in which the action is maintained and augmented by a reflex process from the afferent muscle-nerves, and it ceases when these are divided. Professor Sherrington has shewn that the impulses from the afferent nerves, when the muscles are extended, inhibit the spinal centres for the active muscles, and the opponents contract in turn ; thus an alternation in the active muscles and their opponents is set up, and goes on. But this only takes place if the motor path from the brain is interrupted higher up the cord. It is evident, therefore, that in the spinal centres these actions are normally combined and rendered simultaneous,—apparently by the effect of the volitional impulse. Tremor, however, does not seem to depend upon a defect of this impulse in paralysis agitans. There is no primary paralysis. At least the tremor bears no relation to failure of volition. It may, more reasonably, be ascribed to a defect in the cerebral tonic influence. This must blend in the spinal centres with the voluntary influence, and both may shew the defect, although in different degree. In the early stage, only an apparent intermission of contraction is recognised, either in rest or on movement, but the defect afterwards attains a degree sufficient to cause a distinct alternation of the acting muscles and their opponents, often greater on volition than at rest. It is generally most marked in the part where volition is elaborate and delicately organised, as in the muscles moving the hand. We cannot yet fathom the conditions which determine its extension. It is noteworthy, however, that the increase of tonic contraction determines the posture and also the slowness of voluntary movement, and that in some cases there is very little tremor, although the fixation by tonic contraction has reached a high degree.

According to the view now held by many authorities of the structure of the nervous system, we can understand that this affection does not involve the degeneration of the fibres which conduct the motor impulses. These fibres depend for their vitality on the cortical motor cells, which, according to this view, have nothing to do with the functional activity of the fibres. The impulses, conducted by their component fibrils, arise at the ends of the fibrils, at the terminal extremities of the subdivided "processes of the cells," the ends of the dendrites, and these impulses merely pass through the cells. It is to those dendritic endings (or beginnings) that we have to look for the mechanism of this disorder. Minute as they are, their number is unknown, but its amount is vast, and multiplicity compensates for minuteness alike in ordered and in disordered action. Until we know more of their action in health, it is useless to speculate on the way it is deranged in disease. But it is a clear advantage to perceive why, even to the end, the symptoms of paralysis agitans do not include excess of the knee-jerk, or foot-clonus, and no degeneration of the pyramidal tracts is found after death.

Whatever the essential change may be in this disease, we cannot doubt that it is of the nature of a senile alteration, a defect of perfect nutrition, which, once established, always progresses, extending and increasing. This is clearly shewn by the age of the commencement. If these dendritic endings are excited by impulses which reach neighbouring endings of other afferent dendrites, neighbouring but discontinuous, we can conceive that atrophic changes may render their excitation intermittent instead of continuous. If their distance is increased during the state of sleep, we can understand that then the intermitting excitation may cease and the tremor may be absent, as it generally is. The disease rarely begins in late senility: if the structures involved have continued unchanged through the danger period, further existence involves little risk.

Symptoms.—The commencement varies according to the degree in which tremor or rigidity is predominant. Commonly one hand is found to shake a little at rest, sometimes only, or more, on movement; the patient finds that it cannot be used so freely, and that slight tremor interferes with prolonged movement, such as writing. The tremor very slowly increases, and after six months or a year is noticed in the leg on the same side, although in very slight degree. As the tremor increases the hand assumes a fixed position, and by the time the leg is involved, if not before, both hands are habitually flexed slightly at all the joints of the fingers, sometimes, however, only at the metacarpo-phalangeal joints. With the tremor, weakness comes on, seldom great; generally, indeed, there is a sense of weakness, partly due to a peculiar slowness of movement. The face presents less play of emotional expression, and wears a fixed look not readily altered. Speech also is less varied in tone, although the sentences are uttered quickly, as if to get them out and done with. There is often a slight forward stoop of the body, and the patient walks in shorter steps than before. Soon after the affection of the corresponding leg the opposite arm begins to shake. By this time the tremor in the hand first attacked has become much greater. Lastly, the leg of the second side is also found to shake occasionally. The tremor in the second hand soon reaches a much greater degree than in the leg of the side first affected; and, almost invariably, the tremor in the legs remains slight compared with that in the hands.

This outline is true of the majority of cases; but the symptoms present considerable variations. Of these the most important is the relative prominence of the general rigidity; the muscles are not rigid to passive movement, but the state is one best described as an apparently increased tonicity of the muscles connected with the sluggishness with which the volitional nerve-impulses are evolved. This stiffness may be great when the loss of power is trifling,. and even when the tremor is scarcely perceptible. The latter can indeed generally be detected on careful observation; but it may be so unobtrusive that the nature of the malady may not occur to the observer unless he has been impressed by the characteristic aspect of the case, and is aware of this occasional predominance of the fixation over the other symptoms.

In some cases the shaking increases, to a high degree, in the limb in which it began, and thereto it may remain limited for years before it spreads. The tremor may begin in a leg instead of in an arm, and pass next to the arm on the same side, or to the other leg; but the arms ultimately display the chief manifestations of the affection, wherever it begins. It may be felt first in the shoulder and descend the arm, although it becomes greatest in the hand. The amount of strength which can be exerted by the limbs varies much in different cases; the weakness does not bear any direct relation to the amount of tremor, and is sometimes for long very slight as measured by the grasp.

Sleep generally stills the limbs; but the constant movement in the waking state becomes a source of great fatigue. With the tendency to tardy response to the will comes a peculiar restlessness, which increases until the patient is constantly desiring to change his posture, or to make some movement. Another common symptom is a sense of internal heat, not often well marked, and absent in a considerable proportion of the cases; but when there is no distinct sensation of heat the sufferers like to be at a distance from a fire, and to have few bedclothes over them even on a cold night. Now and then there is a sense of coldness instead of heat.

Some of these symptoms present features which need more detailed mention.

The tremor is a regular rhythmical contraction of the muscles, alternating in the opposing groups, and having a frequency of from five to seven contractions per second. It is greatest in the forearm muscles which move the fingers and wrist, or in the intrinsic muscles of the hand. It often preponderates in the latter so as to cause a movement like that by which the Arabs beat their small drums. The range of movement of the fingers varies from an $\frac{1}{8}$ to $\frac{3}{4}$ of an inch. In the leg it can often only be discerned by placing the fingers on the muscles; the weight of the limb and the posture at rest may prevent the movement being seen. In the leg it is most marked in the calf muscles and also, very frequently, in the adductors of the thigh. In the hand the tremor is at once arrested by any voluntary effort, but soon returns if this is sustained. Hence it causes the motion of the pen, in writing, to be irregular; lines which should be straight present minute zigzags. Nearly half a minute may elapse after movement before the tremor again returns; but the severer the case the earlier is the return.

This relation to movement is not invariable. It is the common condition and has great diagnostic importance, as it presents a contrast not only to the wilder movements of disseminated sclerosis (which occur only on volitional movement), but also to simple tremor. Yet cases of true paralysis agitans are met with in which tremor is slight and at first confined to voluntary movement. The other symptoms—aspect, posture, and gait—sufficiently indicate the nature of the disease in the cases in which the tremor is not spontaneous. Shaking of the head, so common in "senile tremor," is generally due, in paralysis agitans, to a communicated movement from the arms. But even in shaking palsy there is occasionally a primary tremor of the head, the result of contractions in the muscles of the neck.

The masseters are often affected in the late stage, and sometimes sooner. The tongue may likewise present tremor, either when at rest or on movement.

In cases in which one arm is affected in considerable degree before the other, a very slight but enduring increase of voltaic and faradic irritability has been found.

The *aspect* of the patient, as already mentioned, is most characteristic. It is partly due to the forward stoop, to the expressionless face, to the position of the arms—held in front of the patient—and to the fixation of the hands. The common position of the fingers is in the "rest" position, the phalanges are either slightly flexed at all joints, or in the "interosseal" position, with flexion at the metacarpo-phalangeal joints, and extension at the others, from preponderant contraction of the interossei. These muscles have been known to become permanently shortened, but tissue changes are extremely rare in the muscles, and are not usually met with in other structures. The slow movement of the face and lips, as the disease advances, impairs the retention of saliva, which dribbles from the lips. The sense of restlessness causes a craving for constant change of posture.

Gait.—Associated with the forward stoop is a tendency to take short quick steps, as if running forward to save a fall. The "festination," as it has been called, was one of the symptoms which attracted Parkinson's attention. Its mechanism is obscure; especially because a patient who is made to walk backwards will then also present a similar tendency to move by short quick steps, and may have some difficulty in stopping himself: this symptom has been called "retropulsion." Yet it is curious to observe the ease with which a patient may be made to take proper steps. If some object, such as a sheet of newspaper folded to a narrow band, be placed on the floor, two feet in front of the patient, and he be told to put his right foot on it and walk across the room, he will do so with steps of full and uniform length.

Thermic Symptoms.—The sense of subjective heat may exist without any undue flushing. But this may accompany the feeling, and a tendency to perspiration may be present on any exertion or external warmth.

Mental Symptoms.—As a rule, the only mental changes are the distress and depression produced by the continued discomfort; the restlessness which knows no relief; the fatigue which sleep imperfectly removes, and the depression due to the consciousness of a malady which is found, only too soon, to resist every effort to lessen or arrest it. But in some cases, mental failure—loss of memory, extreme irritability, and even a certain degree of dementia—is associated with the physical symptoms of paralysis agitans. The occurrence of this associated failure of mind is not difficult to understand, if we conceive the morbid process to be a senile degeneration of the cortex; yet it is occasion for many mistakes in diagnosis, especially in cases in which there is little tremor, and rigidity is the chief symptom.

Course and Termination.—As already described, the symptoms may remain limited to one limb for a long time, but more often become general slowly or quickly. Their extension may indeed be so slow that several years pass before the patient experiences real disability. On the other hand, a distressing degree of intensity may be attained in the course of a single year. Whether speedy or slow in course, the malady is progressive. Occasional exceptions are presented by the cases in which the affection is limited in its seat, and reaches a considerable intensity without passing beyond the limb first affected, and those in which the general aspect and rigidity are absent.

Paralysis agitans causes death only by gradual exhaustion after long duration. It presents an illustration of the general rule that chronic diseases do not themselves terminate life. The part they play in causing death is to produce a condition in which some trifling intercurrent malady is adequate to snap the thread which has been reduced to extreme tenuity. The duration of the disease varies from two to twenty years, and is, as a rule, only too long.

Diagnosis.—The characteristic symptoms are the tremor at rest, combined with the general muscular fixation of face and limbs, and the posture. This fixation may, however, be absent when the tremor, although characteristic, is limited to one limb. When tremor is slight, fixation is often present. It is important to remember that the usual relation to voluntary motion may not exist when the tremor is but slight; even in a case in which the general aspect is so characteristic as to leave no doubt of the nature of the disease, and in which the diagnosis is completely justified by its course, shaking may be seen only when the hand is moved. The occurrence of tremor on movement only is a character common to "simple tremor," to "senile tremor," and to all the toxic forms. In these general fixation is absent, and error in diagnosis will be prevented if we remember that simple tremor often begins in youth, and that in it and the senile form the head is generally affected early, and the movement is always small in range.

The relation to movement, and the tremor of the head, have received too much weight as special diagnostic features. It cannot be too frequently urged that the important diagnostic symptom is what may be termed the stiffness of the patient. Once grasped, it is never forgotten and will never mislead. Cases in which this aspect is most marked, and of absolute significance, are sometimes mistaken, for many years, on account of the absence of tremor or its inconspicuous degree. Such an error is specially likely when there is some definite mental change, loss of memory, or slight and occasional wrong ideas. Such changes, indeed, seem to be rather more common in the cases in which tremor is almost absent.

The greatest diagnostic difficulty is presented by two forms of tremor. Cases of simple tremor, on reaching the early senile epoch, 50 to 60, may pass into paralysis agitans. The change is pronounced by the increase in the constant oscillation and the addition to it of rigidity, stiffness, and slowness of movement. An even greater difficulty is presented by cases of senile tremor which begin at this epoch. Often a definite diagnosis cannot be made. As long as the tremor is limited to volitional movement, and rigidity is absent, the hope is justified that it will not increase, but a year or two may have to elapse before a confident opinion can be formed.

Prognosis.—The course of the disease is progressive, slowly or rapidly, in a proportion of cases so vast that the exceptions cannot enter into practical consideration. Neither age, sex, nor causation seems to influence its rate. Only when the symptoms increase to a considerable degree in a single limb, and are confined to this, is there reason to expect that the extension of the symptoms will be very slow.

The energy of vitality is possessed in different degrees by the many structures of which the human frame consists. The failure of vital nutrition seems to be altogether beyond the scope of treatment; and that it should be so is intelligible. The causes that bring about failure seem to be effective by reason of a disposition which is as much beyond our perception as it is beyond our influence.

Treatment.—The peculiar character of the malady, regarded as a local senile degeneration unlike those which occur from other influences, is especially marked in its therapeutical relations. Even in the rare cases which begin before middle life the progress of the disease is not arrested by any treatment hitherto discovered. The statement is of course equally true of the cases which begin in the later years. Indeed, considerable doubt may be reasonably felt regarding the nature of rare cases in which recovery has been said to have occurred, and which have been called by the name of this disease.

But, as in all diseases of the kind, it is equally difficult to affirm that no good has been done by treatment. It is as difficult to discern whether treatment hinders the advance of a progressive malady as it is to ascertain if it accelerates the disappearance of one which tends to pass away.

The subjects of shaking palsy sometimes assert with confidence that they are better for treatment; and occasionally their impression is confirmed by our own observation, although seldom in a degree which can be a source of satisfaction. Hence it is right to try to relieve and retard, and to persevere in so doing; especially since there is much difference in the tendencies of the disease, and therapeutical agencies which are powerless in one case may have some influence on another; or, again, those which are without effect at one stage may be distinctly useful at a subsequent period.

Electricity has no influence on the affection. Neither static, voltaic, faradaic, nor high-frequency has any effect, however applied. All that massage can do is to give slight temporary relief. In the extreme degree of the affection, the movement and discomfort in the hands may be relieved for the time by very gentle upward rubbing of the fingers, hands, and forearms. It should be scarcely more than cutaneous rubbing, but the relief it affords at the time is very definite.

Mental tranquillity is as important as it is difficult to secure. The progressive affection, gradually enforcing the conviction of its incurability, maintains an influence like that which is the most common cause. From medicinal agents chiefly can we expect any good, and from them some effect can at times be perceived. Nervine tonics may be tried, such as we employ in other degenerative diseases; care being taken that the doses are at first sufficiently small to preclude irritation. Quinine, arsenic, strychnine, and the like may be tried. From phosphorus and nitrate of silver I have seen no good result.

Relief is perhaps more often obtained from nervine sedatives, especially if combined with small doses of tonics. Of these also it is wise to try successively those that seem to promise best. One which has a very marked effect on the tremor is hyoscine hydrobromide. Four or five minims of a solution of 1 in 1000 may be given twice a day. The effect depends on the degree of the dose rather than on its frequency. Indian hemp is another; the combination of Indian hemp, arsenic, and a very small dose of strychnine—sometimes with the addition of one-eighth of a grain of cocaine—often seems beneficial. Belladonna and physostigma are seldom useful. Of chloride of aluminium and piscidia erythrina all that can be said is that they deserve further trial.

More potent sedatives may be necessary to procure rest. In a malady so chronic, and in which the need is so recurrent, morphine should be avoided. For sleep it is better to give successively bromide (with or without a little chloral), chloralamide, trional or veronal, and sulphonal. Antipyrin and acetanilide or phenacetin, if given in small doses three times a day, sometimes give relief to the sense of distress.

But of all the degenerative diseases of the nervous system this is least amenable to therapeutic agents, and least capable of arrest.

W. R. GOWERS.

REFERENCES

1. PARKINSON. *Essay on the Shaking Palsy*, London, 1817.—2. BERGER. Art. "Paralysis Agitans," Eulenburg's *Real-Encyclopaedie*, Bd. x.—3. CHARCOT. *Clin. Lect.* and *Progrès méd.*, Par., 1876, iv. 838.—4. ROMBERG. *Lehrb. der Nervenkr.*, 2. Aufl., Berlin, 1851.

W. R. G.

EPILEPSY

By Sir William R. Gowers, M.D., F.R.S.

The Name.—Epilepsy is a disease of which the chief feature is the recurrence of sudden brief attacks in which consciousness is usually lost, and there is often spasm of the muscles. In consequence, the patient falls, and the malady received from our ancestors the apt symptomatic name of the "falling evil" or "falling sickness." The state of the muscles is that of involuntary contraction, sometimes slight, but generally intensely energetic; it fixes the frame in some strained posture, stops the breath until the aspect is that of strangulation. It passes off by intermissions, at first slight and rapid, but increasing to more violent shock-like contractions which cease suddenly, leaving the patient unconscious. To the Greeks it seemed that the attack must be the work of some unseen demoniacal agent, and hence they gave the disease the name of epilepsia, a "falling upon," as by a spiritual agent. This involved a theoretical pathology which was apparently absent from the simpler Anglo-Saxon term. The same idea, however, is preserved to us in the popular designation "a seizure," and is not quite absent from the name applied so widely "an attack."

Definition.—More careful observation has shewn that neither fall nor convulsion is a predominant or even dominant characteristic of the disease, common as each feature is. It has shewn also that the attacks which constitute the malady present variations so great, both in degree and in character, as to baffle every attempt to frame a clinical definition. The only definition which embraces all forms is "recurring attacks, sudden and brief, of disturbance of some of the cerebral functions, acting on consciousness, and not due to a cause outside the brain." But this definition is at once too wide and too narrow. It would exclude cases in which local spasm continues for hours, and it would include many cases which must be classed as pure vertigo and not as epilepsy, whilst it would not include the cases in which convulsions are the result of a local injury to a nerve, cases which are, it is true, rare enough.

A nearer approach to adequacy may be found in the pathological definition that epilepsy is a recurring sudden brief discharge of nerve energy in some part of the cerebral cortex, not due to the normal cause of such discharge. Exceptions may be taken even to this description, and their character will appear in the sequel. A definition of epilepsy would not indeed be worth even this slight attempt, were it not that the failure may make some facts, to be mentioned presently, more instructive. The only use of defining is to enable us to see better what is on each side of the line.

Pathological Definition.—The sudden discharge of nerve force, so conspicuous in a convulsion by the muscular spasm it induces, may be traced also in all the minor forms of the attack. In the sensory centres it acts, as we say, "upwards," so as to influence consciousness only, and may cause, for instance, an appearance of bright stars. When the attack consists of loss of sight, or simply of loss of consciousness, there is an apparent exception. We associate "discharge" with overaction. But overaction in one part may arrest action in another.

The sudden liberation of nerve energy in the grey matter of the cerebral cortex occurs in epilepsy without the normal stimulus. There is much in this statement that will render the facts of the disease more significant, although it is only possible to glance very briefly at that which underlies it.

The function of the nervous system, and especially of the cortex of the brain, depends upon a capacity for instant release of nerve energy. The sudden reflex action, the bound away of a frightened animal, proves that the grey matter must hold nerve force almost released. Released from what? We must regard the action of nerve and muscle, which is excited by a touch upon the skin, as a form of physical energy, peculiar because under the influence of life, released and conducted in living tissues. It is probable that the energy from which nerve force is released is latent chemical energy; this latent chemical energy is stored up inter-atomic and inter-molecular motion; nerve energy is a form of transmitted motion by transmitted chemical processes. The reasons for thinking this I have stated elsewhere (6).

The point for us now is that the nerve force must be ready for instant release in the grey matter. Whatever its source, it must be held in a state of most delicate equilibrium by the restraint which keeps it unreleased but ready. Indeed, there is good reason to think that, to use a simile, the vessels which hold it must be not only filled to the brim and delicately poised, but must be actually overflowing. Both the fact of the constant process of nutritional change, and the evidence of constant activity in the nervous system, lead to the conviction that the readiness of the nerve energy is obtained by its constant overflow, that the chemical processes which induce its sudden generation are always going on in slight degree, and that only thus is it kept so as to be released at once in the needful amount.

But this involves a conception of extreme *delicacy of adjustment* between the chemical relations—the tendency of the atoms to unite in fresh and closer combinations and release their latent energy, and the attraction which keeps them from so doing. This makes it easy to conceive that the slightest deviation from the normal relation between the atoms and the molecules may bring these into a position in which the equilibrium is suddenly overturned, and a quick combination and sudden release of energy occurs. We cannot understand why this occurs abnormally, but we can conceive that it may result from that marvellous inherited tendency which produces such precision of correspondence between parent and child in the process of nutrition, or that it may be the result of acquired causes, from the sudden shock of a concussion, mental or physical, or from a cerebral lesion which has slightly, but only slightly, implicated the grey matter in a certain part of the cortex. Indeed, when we consider what the state of the nervous system must be in its natural function, we cease to wonder that many persons become epileptic without any traceable cause, and our marvel rather is that all persons are not liable to such fits.

A morbid relation of the most delicate and minute character between the molecules may make them susceptible to motion which comes by some other path that we do not discern. Discharge without the normal stimulus may not mean discharge without any stimulus: yet even this is possible; we can conceive that the defective equilibrium may entail a disturbance of the balance, when a certain point is reached, and a discharge may have no other cause than the nutritional augmentation of energy.

The important point thus is that an extremely slight derangement of the constitution of the nerve tissues may cause a liability to sudden discharges of nerve force, slight and local or severe and wide.

Recurring Tendency constitutes the Disease.—But another equally important fact must be recognised. The liberation of nervous energy involves the escape of some atoms of the nerve tissue which are promptly replaced from the plasma adjacent to them. The rearrangement is the same, and yet not quite the same. It differs in just the degree and character to make the same release of nerve energy more easy in the future. Every action paves the way for its own recurrence. This is the secret of the acquisition of skill; it constitutes the physical basis of memory; it is this which facilitates the repetition of a given morbid process, and this causes the recurrence of discharge which makes epilepsy a disease. It is the recurrence of attacks which constitutes epilepsy, and this is in great measure the result of the previous attacks. Every fit is in part, at least, the result of those which have preceded it, and in part a cause of those which follow it. Hence the immediate cause of the first fit must be regarded as the exciting cause of the disease, although the essential cause is the disposition, that is, the state of nerve-nutrition, which makes such discharge possible. The degree of instability may, of course, vary; and when it is not great the first attack may have been due to some cause of considerable energy.

These considerations will make it easier to understand the causes of epilepsy in so far as they are comprehended. But one other important question must also be noticed.

Local and General Change.—In what is called "idiopathic" epilepsy, that which is largely the result of inheritance, the morbid state consists in some trifling alteration in the chemical constitution of the grey matter on which the instability spoken of depends. But a similar local instability may be the result of obvious disease. Beyond the visible disease, whatever its nature, is a zone of altered nerve-tissue in which the slightness of the change permits activity in altered form. In such cases the discharges spoken of may take place. Arising locally, they spread through the brain according to their energy, and leave a tendency to repetition, which involves parts unaffected by the primary disease. It is not customary to include under the term "epilepsy" active progressive brain disease, such as a tumour, even though it cause epileptiform convulsions. But there is a large class of cases in which an old spot of disease induces such discharges, which may be slight or local at first, but spreading when more intense, and ultimately leaving a general disposition which may be hardly distinguishable from idiopathic epilepsy. Although these cases, which may be called "organic epilepsy," are quite distinct in primary causation, they are not practically separable from those of the idiopathic form.

Perpetuation of Reflex Convulsions.—A third group occupies an intermediate position. There are cases in which some local irritation, for instance in the intestinal canal—such as a tapeworm—has excited "reflex convulsions," which had been so many times repeated before their cause was removed, that a persistent habit of morbid brain function was established. This may happen even without any inherited disposition. It is possible that some other influences, acting on the nervous system from the outside, may have a similar result. Such cases do not ultimately differ in any important way from the common idiopathic disease, and the fits may not cease after the removal of their exciting cause.

Source of the Discharge.—Up to the present time it has been a fundamental conception that the nerve-cells from which the fibres proceed are the source of the nerve energy which passes along the fibres. The obvious analogy between the large cell as a generator and the sheathed fibre as a conductor, has aided in fixing the conception. But the labours of histologists, of Max Schultze many years ago, and of Ramón y Cajal, of Schäfer, and others in our own day, coupled with the known facts of physiology, make it necessary to relinquish this simple and attractive notion. We must, it would seem, regard the function of the nerve-cells as nutritional only. On them depends the life both of the processes, short or long, which conduct to them, and the long axis-cylinder process which conducts from them. Through the cell pass the separate fibrils of which the apparently homogeneous axis-cylinder really consists. These arise, without continuity with others, in the spongy grey matter; becoming associated in the "branching (or uniting) processes" they reach the nerve-cell to pass through its protoplasm without interruption. We must regard the dendritic endings, or beginnings, as the substance in which the nerve impulses arise in the spongy grey matter under the influence of other impulses conducted to it; and we must therefore look to this, and to this alone, as the place in which any alteration exists that underlies the phenomena of epilepsy. The eyes of the observer hitherto have been fixed on the cells and on the cells alone. We do not yet know how to unravel the complex structure of the spongy substance so as to discern the normal relations and aspect of its constituents. We are not yet on the threshold of the observation of such disease as must exist in idiopathic epilepsy.

Causes.—The most important impression which the reader should retain from the preceding sketch is that the conditions of the normal action of the brain involve a state of tension ready for release of energy, and a delicate equilibrium which makes the abnormal sudden liberation of energy readily produced, either in consequence of a slight primary perversion of the conditions on which the balance depends, or a sudden and excessive shock to them.

Heredity.—Epilepsy is frequently an inherited disease. Epilepsy and insanity certainly run together in families. The general tendency to one involves also the tendency to the other, although not often in the same individual. In seeking for the family antecedents of epilepsy, it is found that insanity is the only other disease to be taken into consideration. Other neuroses have too many different connexions, and these are of so common a kind as to be comparatively insignificant. But if only epilepsy and insanity are considered it is found that in half the cases of epilepsy there is a family history of one or both of these diseases. If the facts be gathered from the lower classes, who seek relief and yield data at the hospitals, this association can only be traced in a third of such patients. But when cases are carefully investigated among those whose family knowledge is more complete, the proportion rises to one-half; and even this estimate is probably below the truth. Such diseases in antecedent generations are often unmentioned in a family, and the knowledge of them is thus lost.

Of diseases outside the nervous system, it is not possible at present to discern any which stand in a definite relation to epilepsy. Phthisis was once thought to be connected with the disease by some hereditary mechanism, and the cases of phthisis to be heard of in the families of epileptics are many. But the ratio is not found to be larger than in the families of those who are not epileptics, and an investigation from the side of phthisis clearly shewed that the apparent connexion was due only to the frequency of phthisis. In the same manner it is not possible to say whether inherited gout has an influence in the production of epilepsy. It is difficult to resist the evidence of a connexion between inherited gout, migraine, and neuralgia; but the connexion between these diseases and epilepsy is so slight and infrequent as rather to oppose than support the idea that inherited gout is a factor in the production of epilepsy.

The consanguinity of parents necessarily intensifies a family tendency, but there is no evidence that, alone, it has any influence in producing the disease. Such marriages are indeed far too rare to furnish adequate material from which an opinion can be formed.

Sex.—The influence of sex is not great. For every twenty males, twenty-one females suffer from epilepsy. This is the result of a careful comparison of nearly 3000 cases, from which all examples of hysteria, not associated with true epilepsy, have been excluded. In some small collections previously published males have slightly preponderated, possibly from the inclusion of cases of syphilitic disease of the brain.

Age has a great influence on the production of epilepsy. This may indeed be expected from the tendency to spasm and convulsion which is conspicuous in childhood, which lessens as the stability of full development is achieved, and is again increased during the change with which the period of childhood ends. The periods of this change, and of the first three years of life, are those in which epilepsy most frequently commences. One-eighth of all cases begin in the first three years of life, transient infantile convulsions being excluded. Not less than a quarter of the cases begin in the four years, 13, 14, 15, and 16, which, in the majority of cases, include the epoch of puberty. In the second decade, indeed, the malady commences, as a continuous disease, in no less than almost half the cases; and in the first decade in more than a quarter; thus three-quarters of the cases of epilepsy begin before 20 years of age. The third decade is responsible for another sixth, leaving only one-twelfth to commence after 30 years of age. The recurring fits may begin as late as 70 years of age. Yet only one case in 200 commences after 60.

Sex and Age.—During infancy, the existence of a profound difference in the nervous system is shewn by the fact that in cases of epilepsy commencing in the first ten years of life the females exceed the males by 8 per cent, and the difference is greatest in the first year, in which the males are little more than one-half the females. Puberty has a far more profound influence on the nervous system of females than on that of males, and their excess rises, in the second ten years of life, to 20 per cent. With adult life the influence of sex is changed. In the third decennium the disease begins in the same number of males and females. Between 30 and 40 the males are for the first time in excess by no less than 20 per cent. Between 40 and 50 their excess attains nearly 30 per cent. After 50 the same relation persists, but the numbers are too few to permit accurate comparison. Cases of idiopathic epilepsy begin up to 75, but almost only in males. We may find an explanation of these facts in the exposure of the nervous system to more constant strain of anxiety in men, but it may also be that some compensatory indisposition is developed in women who have maintained stability through the trying period of female development. Certainly facts yield no support to the existence of any special danger at "the change of life," the importance of which has been very much exaggerated.

Age, Sex, and Heredity.—The influence of heredity is considerable at every age, although it lessens progressively as life goes on. Combining the cases seen in hospital work with those seen in private, we find the total percentage of heredity is 41 per cent—about 8 per cent less than that found in the cases seen only in private. In the first 20 years the percentage of heredity is 44, in the second 39, in the third 29. Cases with inheritance are met with up to the latest period of life at which epilepsy begins.

The proportion of the sexes in the cases in which heredity can be traced is almost the same throughout life as in the total number at each period.

The large proportion of cases in which no predisposition can be traced need occasion no surprise, if we remember that the conditions for the sudden release of nerve energy essential to the normal action of the brain, must entail a liability to readiness to abnormal discharge on such slight disturbance as may be conceivably brought about by causes which we cannot at present recognise.

Proximate Causes.—So far as we know, and can interpret, the facts of the disease, we regard a disposing condition of the nerve tissues as the chief element in causation. In a considerable proportion of the cases, however, the first attack is referred, with more or less reason, to some immediate cause. In most cases this is too slight to do more than determine an event for which the conditions exist. It is either like the gradual rise of temperature which may bring about sudden combustion, or a spark which immediately excites a discharge of explosive material.

In one-half of the cases such a proximate cause can be traced. It is more frequently met with in males than in females; perhaps because the predisposition is more powerful in the latter and needs an exciting influence less frequently. In infancy, nevertheless, the first fit seems to be excited as frequently in one sex as in the other. After puberty, and to the end of life, a proximate cause is recognised more frequently in males; and after fifty it seems to be rare for the onset not to be distinctly excited.

Particular Causes.—Infantile causes.—A considerable number of cases of epilepsy date from infantile convulsions. In most of these the first fits occur during retarded dentition (commonly with other indications of the state called rickets) as definite convulsions or as very slight "minor" attacks; these persist to puberty, and then become definitely epileptiform. In other cases they may cease after infancy for a year or two, and recur as minor attacks. There is every gradation between persistence and an interval lasting from the first dentition to puberty. This suggests that the early convulsions had produced a lasting change in the brain-tissue, facilitating the tendency to spontaneous discharge at certain developmental epochs, or when some excitation induced an attack. In about one-tenth of the total number of cases of epilepsy, some influence can be ascribed to infantile convulsions.

The popular connexion of these fits with the process of teething has probably a very slight foundation in fact; any local irritation may have an exciting influence. But the chief factor is the general retardation of development. This of necessity has most influence on those nerve structures which are either the least developed or have most recently attained structural completion. The highest controlling centres are latest to develop, and will suffer most from any hindrance. The motor centres which subserve reflex processes in the cord and brain have then an insubordinate excess of function; and among these lower centres we must include the structures of the motor cortex as well as those of the spinal cord and mid-brain. It is not difficult, therefore, to perceive a process by which simple retardation of development shall induce the reflex overaction which gives rise to tetany, to carpo-pedal contractions, to laryngismus stridulus, and to actual convulsions. The influence which is exerted by this process leads to ordinary idiopathic epilepsy; but the attacks which connect the early and later seizures are often those of the minor form, and their character and significance are commonly disregarded by those who have the care of a child so afflicted.

In connexion with the early origin of epilepsy, it should be noted that cases of another class are due to the sudden occurrence of a cortical lesion on the surface of the brain at some time in the first two or three years of life. This may cause hemiplegia, transient or persistent; or an initial weakness so slight as to attract no notice, especially if the lesion occur during some prostrating illness. In the latter case the lesion is near the motor centres, but does not involve them; and disease so situated is especially apt to give rise to subsequent convulsions. These cases are distinguished by the severity of the initial convulsions, by their frequently unilateral character, and by the fact that the slighter fits at a later stage distinctly begin on one side and involve this chiefly. Although such cases are essentially different from those of idiopathic nature, the frequently recurring discharges seem to induce a similar state of the brain; and not uncommonly minor attacks develop that are quite similar to those of the idiopathic form.

In a few cases, which must be referred to infancy, although the attacks may present themselves at a later period, the cause is damage to the cortex, which occurs during the process of birth. The amount of mischief in these cases is seldom sufficient to cause the characteristic bilateral motor affection known as "birth palsy"; and the recognition of the cause is often difficult. Cortical injury is to be suspected, however, when convulsions are manifested in early infancy, or after dentition, in first children whose birth had been tedious; and this is especially probable when symptoms during the first days of life point to some meningeal haemorrhage.

Injury in or near the motor cortex may lead to recurring convulsions; but these begin in particular parts which correspond to the position of the lesion. These cases are rare. In some cases, however, general convulsions, like those of idiopathic epilepsy, follow some more general concussion which has possibly a widespread influence on nutrition. Yet it must be remembered that injury involves a mental as well as a physical shock, and mental shock, as will be seen, is a potent cause of epilepsy.

Toxic Influence.—A first fit may occur during or immediately after an acute specific disease; but only one specific disease has this sequence in any considerable proportion of cases of epilepsy. This is scarlet fever. The first fit may occur during the disease or soon after it, often without any renal sequel with which it can be associated.

In rare cases some other toxic state of the blood is the apparent cause. This may be due to some perverted chemical process within the body, such as that which results from chronic kidney disease, in which recurring convulsions may apparently arise independently of uraemia in the strict sense of this word. It must be remembered that the abnormal chemical process which we can trace must be associated with other chemical changes that we cannot discern, and that these may often have far more toxic results.

Emotional Disturbance.—As a direct excitant of the first fit, intense sudden alarm takes the first place. It can be traced in a considerable proportion of cases. In many instances the cause of the alarm seems absurdly inadequate; but that which seems inadequate at first sight may not seem so on further consideration. An illustration may be given to which particular exception was taken by a French author, when it was first mentioned, on account of its trivial character. Mere minuteness of excitant, however, becomes unimportant if it act as the spark in an explosion of gunpowder; and it must be always kept in mind that the essential cause is the internal predisposition. The instance referred to is that of a nervous sentinel who was on duty one dark night near a churchyard. A white goat suddenly ran across the churchyard and jumped upon the low wall. The man, convinced it was a ghost, shook with terror, but was unable to desert his post. An epileptic fit soon followed, which was succeeded by others.

The disturbing effect of sudden fear seems the greater when it cannot have its normal consequence, namely, the energetic discharge of the motor centres to escape from danger. There is action upon them, but it is perverted in effect; it disturbs their nutrition and deranges their function. The immediate effect is seen in the trembling, which has thus become a synonym for fear; the remote effect is seen in such maladies as epilepsy, chorea, paralysis agitans.

A true epileptic fit seldom follows the sudden fright instantly. An instant fit is more commonly hysterical, and sometimes perhaps preserves the patient from the more enduring malady. An epileptic fit generally occurs within a week; if a longer interval elapses the relation to the alleged cause is doubtful.

Prolonged anxiety seems also to be a definite cause, although the more gradual mode of its influence makes it less easy to trace with certainty. Acting upon a predisposed person it seems to be influential at all ages, and in the later period of life chiefly in the male sex; it is rare for a senile sufferer not to have been subjected to a long mental strain. The way in which this cause acts is less perceptible than in the case of sudden fear. It doubtless depresses the nutrition of the whole nervous system, for there are few chronic degenerative diseases of which it may not be a cause; but probably its influence in producing epilepsy, as with the other causes in earlier life, is the result of a definite predisposition on the part of the structure concerned; how this predisposition arises we have no knowledge. In senile subjects disease of the smaller vessels often exists, although we do not know what part it plays in causing epilepsy.

Reflex Causes.—The chief peripheral irritation which can be regarded as a cause is that of intestinal worms. It seems to be only from irritation in the intestine that general convulsions result. These cases illustrate the manner in which convulsions are self-perpetuating by the primary disposition already mentioned.

Irritation of the nerves of the limbs is such a rare cause of epilepsy that doubt as to the occurrence of fits from this is not unreasonable. It is certain that they occur too seldom to merit special description.

Pathology.—The chief inferences regarding the pathology of epilepsy have been mentioned in the introductory section. The nature of the disease can only be inferred indirectly from evidence, otherwise obtained, regarding the functions of the nervous system and from the negative effects of the disease itself. At present there is no morbid anatomy of the disease, nor is it likely that any conspicuous changes will be found, except such as may result from the excessive functional activity in the status epilepticus. The effects of the attacks must be too widely distributed to afford any clue to the primary disturbance.

It would serve no useful purpose to describe the conceptions of epilepsy which have been current during the last fifty or sixty years. Fifty years ago it was generally believed that the epileptic fit was the result of a spasm of the arteries of the brain, due to the discharge of a vasomotor centre in the medulla. This view has almost disappeared before the simple but conclusive facts which connect the manifestations of the disease with a morbid action in the cortex, facts which were first discerned, with clear vision, by Dr. Hughlings Jackson.

(i.) Sudden liberation of nervous energy in the brain is one certain fact of the epileptic attack. (ii.) The first indication of this liberation, which consciousness perceives, proceeds from the cortex of the cerebral hemisphere. (iii.) We have no evidence that the initial symptoms ever proceed from any other part. (iv.) It is probable, then, that the nerve discharge which causes muscular spasm proceeds from the cortex of the brain. (v.) We have no evidence that the sudden liberation of energy in the cortex is the result of any preceding morbid action. (vi.) All that has been learnt of the action of the nerve-centres is in harmony with the primary disturbance being in the seat to which the symptoms point. (vii.) The essential element in the disease, as such, is the effect of morbid function or nutrition, by which its recurrence is facilitated.

The explosive liberation of nerve energy in the cortex has been hitherto supposed to occur in the nerve-cells. But recent perceptions of structure, and consequent conceptions of function, throw doubt on the opinion that it occurs in these enclosed structures, through which the nerve-fibrils seem to pass without interruption. They point rather to the spongy grey matter, in which nerve material is finely divided, and to the ends of the branching processes of the nerve-cells as the element in which latent chemical energy is transformed into the nerve force which is transmitted by the nerves and stimulates the muscles. Through the nerve-cells the impulses pass along the separate fibrillae which, as continuous paths, traverse the cell and compose the axis-cylinder of the nerve-fibre. To go farther into the region of speculative pathology would not serve any useful purpose.

Symptoms.—The severe epileptic fit is that which has always been the recognised type of the disorder, and therefore may be first described. The sufferer, with or without some momentary sensation—a sensation at the epigastrium, or of some special sense of vertigo or the like—becomes unconscious and falls, often with violence, as if hurled down. The fall is sometimes such as to cause injury to the patient, or to involve him in some danger, as from fire or water. The fall is due to involuntary spasm, inconsistent with the maintenance of the upright posture, whether of standing or sitting. The spasm is tonic, and affects all the muscles of the body; in those of the chest it occasions an expiration which, if it coincide with spasm of the larynx, may cause a strange cry.

Although general, the spasm is seldom equal on the two sides; the head therefore deviates to the side on which the spasm is most intense, and the arms are not moved equally. They are often slightly flexed at the elbow, which is separated from the trunk; and the fingers are in strong "interosseal flexion." Occasionally the hands and wrists are strongly flexed, in which case sometimes the elbow is also flexed and the hands are brought in front of the body. The legs are usually extended, although often not completely. In some cases, with strong flexion of the arms, the legs are drawn up. The mouth generally deviates a little towards the side of the greater spasm, towards which also the face and eyes are often directed. After about thirty seconds, during which time the arrest of breathing renders the face cyanotic, tremulous variation in the tonic spasm is observed; this soon increases to definite remissions, and to the jerks of the clonic stage. The remissions deepen until they become intermissions, and soon afterwards, at the end of about another minute, occurs the last jerk, which is often as violent as those which have preceded it; the patient then lies in a state of exhausted relaxation. During the stage of clonic spasm the cyanosis lessens, because there is movement of air into and out of the chest; this causes saliva to be frothed out of the mouth, often attended with blood in consequence of the tongue being bitten. The tongue shares the clonic spasm, and on account of the inequality of this the tongue is pushed to one side, so that the edge is cut between the jaws. As it is very rarely pushed between the teeth before the jaws are brought together by tonic spasm, the tongue is not usually bitten in this stage. Some patients never bite the tongue, apparently because of some special character of the spasm. Urine is often passed during the attack, a symptom which is probably due to extension of convulsive spasm to the wall of the bladder, seeing that it is discharged with violence, and in no proportion to the degree of general muscular contraction.

Attacks occur of less severity in every degree, even to that in which the occurrence of muscular contraction can scarcely be recognised. In idiopathic epilepsy the initial tonic spasm is a general characteristic, and in the slight fit it may be the only spasm. A brief stiffness may indeed alone represent an attack. Cases in which a general convulsion of very slight degree consists only of clonic spasm are exceedingly rare.

In this respect convulsions which begin locally, from local disease of the brain, stationary or active, present an absolute contrast to the idiopathic form. In the former the attacks begin by local clonic spasm in the part related to the spot in the brain at which the disease has raised the excitability to a morbid degree. The spasm thence spreads throughout the side, and then may involve the other side. If the discharge is intense the spasm may extend with such rapidity that the second side seems to be involved almost from the outset. Moreover, the first clonic convulsion may quickly change to tonic spasm, which then runs a course similar to that presented by the idiopathic form. This change is apparently promoted by the effect of repeated discharges on the whole motor grey matter of the brain, which produces a tendency to general discharge that may ultimately be almost as intense as in idiopathic epilepsy, and must be considered in assigning such cases to their proper position. This view is emphasised by the fact that the minor attacks, presently to be described as occurring in the idiopathic form, may appear in the cases of old stationary organic disease.

In such severe fits consciousness is not always lost at the onset. In a small proportion of them some abnormal state or symptom exists for an hour or two before the attack. In a much larger proportion the onset of the attack is preceded, for a second or so, by some sensation or other disturbance of consciousness. This is called the "warning" of the attack, or, in medical terminology, the "aura." This old Latin name for the premonition seems to have arisen from special notice of the cases in which local disease of the brain and local discharge cause such partial commencements of the attack as twitching contractions, the clonic spasm above mentioned, or some sensation in the part, generally tingling, which begins in an extremity and swiftly passes up a limb. But it is probable that the origin of the term was connected with the idea, dominant then and for many a century afterwards, that the arteries contain air; and the ascent of the sensation was attributed to the ascent of an unnatural vapour in these vessels. The term "aura," like "hysteria" and many others, has survived its original associations, and persists as a simple synonym for the immediate warning of the fit.

This immediate warning is of great importance. Except in the cases of extreme local instability of the cortex, generally due to local disease, the warning is some sensation. The sensation is the effect on consciousness of the commencing process of discharge, which begins in structures by which those are influenced that are highest in function, related closely to consciousness. The warning is thus an indication of the part of the brain in which discharge begins, because the place where it first attains such an intensity as to cause a "sensation" must be assumed to be the place at which it starts. In general the sensation is uniform in the same case; occasionally it undergoes a change in the course of time.

The importance of these warnings is increased by the fact that they are perceived more frequently and in greater degree the slighter the attack. This is true of the whole range of degrees downwards, until at last the lowest level is reached, in which attacks may consist only of the "warning" or "sensation"; sometimes with imperfect loss of consciousness, sometimes with no appreciable impairment of it. This has led to the popular use of the word "sensation" as a synonym for the minor attacks. As a rule the warning of the slighter attack, with definite loss of consciousness, is the same as that of the severer forms, should consciousness persist long enough for its perception. Sometimes, indeed, the slighter attacks are heralded by a different warning, but the common correspondence makes it well to consider the warnings of attacks of all degrees together.

The relative amount of the process of discharge that is perceived by consciousness varies. As already intimated, it may be the whole, the disturbance ceasing with the warning, consciousness not being lost. In other cases there is merely a partial impairment of consciousness, enough to prevent perfect recognition of the surroundings, so that they seem to be unfamiliar and strange. Every form of aura may, however, be followed by definite loss of consciousness, which in slight attacks is usually momentary. It is prolonged in many cases in appearance, and in reality so far as the self-consciousness which involves memory is concerned. There may be a moment in which the patient is obviously unconscious; this is succeeded by a few minutes of automatic action, rambling talk, attempt to undress, and the like, which in another person would not imply unconsciousness; although the patient may remember absolutely nothing of that which has occurred. Such a state may follow an attack in which there was slight spasm, but it hardly ever follows an attack with severe convulsion. This sequel will be considered presently.

Prodromes.—Besides the immediate warning, or when there is none, a few patients are conscious of some unusual symptom which tells them that an attack is impending. There may be peculiar mental irritability, or sudden jerks, or definite minor seizures. It is important to keep these distinct from the immediate warning, since a patient when asked if he or she knows that an attack is coming on, will often say "Yes," and describe such a prodrome even when there is no immediate warning.

Forms of Aura.—The chief forms of warning may be briefly considered. Their interest is great, but their practical value is almost limited to the detection of attacks, which might otherwise be unperceived, by the evidence this character affords, and by their frequent indication that the instability is the result of a local lesion of the brain.

Motor Phenomena.—Local clonic spasm in the limbs is too rare in idiopathic epilepsy to be worth mention, but it is important in connexion with organic brain disease.

A definite but unusual commencement is by the act of running. A patient may run forward several yards, even a hundred, and then fall in a fit. Another may turn round and walk back, and then fall. Another may turn round two or three times, and then fall unconscious in an epileptic convulsion. We cannot really explain these co-ordinated motor symptoms, but the last is obviously connected with the most common warning of which a memory is retained by consciousness, namely, vertigo.

The giddiness which is a common warning of an epileptic fit seems to be a purely motor symptom, the result of the influence on consciousness of the greater energy of the motor centres of one hemisphere. Hence as a rule the patient feels as if turning towards the side which is most convulsed; and it seems to him that other things are turning in that direction, because he imputes to objects the movement he seems to have. If this terse description seems not to be clear, it will probably become clear if carefully thought over. Occasionally the features of the vertigo described are difficult of explanation.

Vertigo involves a false perception of the relation of the individual to his environment, and this constitutes inaccurate, that is, imperfect consciousness.

But in simple vertigo, apart from epilepsy, definite loss of consciousness is extremely rare, and clearly is the result of the intensity of the vertigo. In epilepsy it is frequent, even when the vertigo is slight. The sensation of turning may be associated with a visual aura, the appearance of an object which moves from one side of the field of vision to the other, and which the patient seems compelled to follow until its disappearance. Often the side on which the object appears is that chiefly convulsed, the side of the body, that is, opposite to that of the brain in which the chief discharge takes place; and the disappearance of the object on the opposite edge of the field of vision is followed by a turning of the head in the convulsion. This fact illustrates the extreme complexity of the features of the vertigo in the epileptic discharge. Those who desire to pursue the subject further will find some information about it in my Bowman lecture (7). The leading fact, however, is the evidence of the motor character of the warning, associated as it is with deviation of the head and sometimes with rotation of the body. Moreover, as sudden vertigo is often the only subjective indication of a minor attack in which there is a moment of absolute unconsciousness, this symptom is of great practical importance.

Local Onset.—In idiopathic epilepsy there is seldom more definite evidence that the discharge occurs earlier in one hemisphere, than is afforded by the evidence of excess on one side which is shewn by deviation of the head; but in other cases the onset may be indicated by a sensation in the extremity of one limb only, usually by a tingling which passes up the limb; or there may be clonic spasm associated with the sensation, or occurring alone. The sensation or spasm may begin in some part of the hand, for instance in the thumb and finger. There may be a similar beginning in the foot, or in some part of the face, generally the angle of the mouth. The spasm may remain local, or may spread through the side, or to the other side, with varying rapidity. In all such cases there is excessive local instability in the cortex of the brain, in the centre related to the local result. Such excess of local instability is very seldom part of idiopathic epilepsy. It is commonly the result of an old organic lesion, which has damaged the grey matter locally, and made it functionally unstable. An infantile lesion is the most common cause, but such attacks may be due to thrombosis or embolism in later childhood or early adult life. The grey matter of the brain is normally so sensitive that discharge spreads rapidly and widely in proportion to its intensity. Repeated discharges establish a widespread habit which is practically identical with the general brain state in idiopathic epilepsy. The local disease which causes the instability is often such as to cause some degree of hemiplegia, especially at the onset. Persistent hemiplegia is usually due to destruction too extensive to permit discharges; but these cases are often called "post-hemiplegic epilepsy." It is important, however, to note the manner in which the tendency to discharge becomes established. After removal of a small superficial scar attacks have continued; moreover, the convulsions which begin locally, in the hand, for instance, and spread thence, may be accompanied, after a time, by minor attacks, quite like those of idiopathic epilepsy. We can separate such cases by their causation; but we cannot always, or indeed often, separate them by their course or characters.

One point regarding this warning deserves note. It may be purely sensory, apparently a gentle discharge in the sensory elements of the central region. This aura may be the result of organic disease, and is one of the facts which shew that the central region, whence the motor impulses proceed, has also sensory functions. A gentle discharge of this kind, causing, for instance, a sensation of tingling in the foot, may pass up the leg, up the side of the body, down the arm to the hand, and when it reaches the fingers clonic spasm may occur, which passes up the arm; at this point, as a rule, consciousness is lost. It is as if the discharge in the sensory elements of the centre passed gently through the sensory structures until it reached a functional limit, and was there turned aside to the motor structures, in which it quickly gathered force. Again, such a sensory discharge—in the arm, for instance—may be accompanied by sudden powerlessness of the limb without spasm. Apparently the discharge in the sensory elements simply inhibits the motor structures. If a motor discharge begins in the centre for the foot and causes clonic spasm, this, passing up the leg and side, may begin again in the hand on reaching the level of this; it does not descend the arm from the shoulder.

Bilateral and Trunk Auras.—Occasionally the warning is some sensation referred to both legs, which seems to ascend, often to the head; or it may be a sensation in the spine. From such trunk warnings it is necessary to separate those which are included in the next class.

Visceral Auras.—The onset of an attack is often announced by some sensation referred to the viscera, the epigastric region, the cardiac region, the throat, or the head. With the exception of the cephalic warning, these visceral auras may be referred to the region whence impressions reach the centre through the pneumogastric nerve; the chief functions of this nerve are represented in the gastric, cardiac, and throat sensations.

The epigastric aura is one of the most common. It may be a deep-seated pain, sometimes associated with nausea. More often it is a peculiar undescribable sensation, which often ascends to the throat, rarely to the head. When it seems to pass to the head there is immediate loss of consciousness. If it passes up to the throat it becomes there a sense of constriction or fulness, and of breathlessness—similar, apparently, to the globus hystericus. The pain which remains at the epigastrium, associated with nausea, may reasonably be regarded as due to the central representation of the gastric function of the vagus. This is compatible with another fact, namely, that the sensation is occasionally referred to a lower part of the abdomen, since some fibres of the vagus pass to the intestines. On the other hand, the sensation which ascends to the throat and is associated with a feeling of want of breath, may be referred to the central representation of the respiratory function of the nerve. In the same class may be included cardiac sensations; these are not common and are seldom well defined. The most common of these is pain, palpitation, or a sense of arrest of action.

Cephalic Sensations.—When an epigastric aura ascends to the head, on reaching it consciousness is usually lost before the character of the cephalic sensation can impress itself so as to be revived. This is an interesting fact in relation to the representation in the sensory centres of the brain itself. Occasionally it is a definite physical sensation in the head. It can seldom be described except as a "rushing," or "fulness," or "movement in the head." This last presents a gradation to vertigo by a feeling as if something were turning round in the head, now and then associated with a sense of movement of external objects.

Special-sense Warnings.—These are not uncommon. The order of frequency is visual, auditory, olfactory, gustatory.

Olfactory and gustatory.—As an aura of epilepsy, a smell is always distinguished from a flavour; although both are normally produced through the olfactory nerve, as the cause gains access to it through the anterior or posterior nares respectively. The latter often seems to blend with a true gustatory sensation, as a sensation of "taste." A pure taste, sweet, bitter, etc., is seldom described. These warnings are almost always unpleasant, but generally transcend the powers of description and even of comparison. Our vocabulary is quite inadequate to the expression of normal sensations, and still more for those which are due to processes of abnormal character. In many minor attacks, especially those attended with a "dreamy" feeling, there is smacking of the lips, of which the subject is unconscious. It suggests a sensation of taste, but this is very seldom to be heard of in such cases.

Auditory Warnings.—These are less uncommon. There is occasionally some simple sound at the moment of onset, such as a whistle, the sound of a bell, or a crash. Occasionally there is a more elaborate sound, music, or voices, and even distinct words. The latter are important for the reason to be mentioned in connexion with the more elaborate visual warnings with which the similar auditory aura is sometimes associated. Inhibition of the auditory centre may precede the fit; there is sudden silence, which may be associated with sudden darkness, before consciousness is lost. The loss of hearing may precede or accompany the subjective sound, an association of inhibition of response to external stimulation, with a spontaneous action in the centres related to consciousness.

Visual warnings are more common than any others connected with the special senses. The extreme delicacy of the structure which responds to the most rapid form of wave motion of which we have direct perception, must be associated with a corresponding delicacy of action in the central structures that receive the nerve impulses produced by the waves of light. It is not surprising that, when central instability exists, the visual centre should often lead the way in the sudden derangement. The warning may be a flash of light, stars, sparks, simple or coloured light. There may be more complex sensations of objects, faces, persons, scenes; and with the last an auditory aura of corresponding elaboration may be associated. It is important to note that in those patients who experience such psycho-sensory auras there is often a strong tendency to mental derangement.

Inhibition of the visual centre—sudden darkness—is a frequent warning. It may be followed (it is seldom preceded) by a "discharge" in the centre; first all becomes dark, then stars or a luminous object appear. This is an important indication of the close relation of arrest of action and spontaneous action, of inhibition and discharge.

The various special-sense warnings are occasionally associated, most often the auditory and visual. The degree of elaboration in each usually corresponds, although sometimes an aura of low degree is succeeded by one of greater elaboration. Two lights, in one case, were always succeeded by the figure of a woman. In the case of both the visual and auditory sensations there may be a progressive increase or diminution; lights which suddenly appear may become fainter and seem more distant, or may become large and seem to approach; sounds may become slighter, or may seem to become louder and louder. In each case, with a considerable degree of change, consciousness is lost.

Psychical Auras.—A sudden abnormal mental state may constitute the first symptom, and may leave a definite impression upon the memory. The brain processes which attend mental states may leave a residual effect which permits their subsequent revival; and their perception may be called, somewhat loosely, "psychical sensations." That which precedes an epileptic fit may be an emotion or an idea. The emotion is almost always some form of fear. It may be definite alarm, and

KING'S COLLEGE HOSPITAL
MEDICAL SCHOOL.

associated with the conception of some cause for it, as that of a pursuer from whom the patient has to run away, and does actually run, sometimes looking back in terror. It may be more vague in character although definite in nature, as a sense that whatever is being done is morally wrong.

Those warnings which take the form of "ideas" are generally of the nature of complex special-sense conceptions, especially related to vision. Not only may a sense of fear be accompanied by the vision of some cause for it, but the more elaborate visual sensations are such as to involve a psychical process. A remarkable instance is that of a woman who saw London in ruins, the Thames emptied to receive them, and herself the lonely survivor—a manifest psycho-sensory warning. Indeed the same combination may be discerned in many other forms. Its details have no other importance regarding the disease than as an indication of a special instability of the centres concerned with psychical processes, which often takes the form of definite mania.

Somnolence.—These warnings illustrate the many-sided relations of the condition which subserves subjective consciousness. Consciousness may be lost and return with such suddenness that no trace of either process is retained by the memory, and the patient is absolutely unaware of the gap. But it may also be lost deliberately by a gradual process, which is described as "sleepiness," or sometimes by an analogous state with a slight psychical character, called "dreamy." This warning is more common in minor attacks, and especially in those which are slight in degree and slow in evolution. Closely allied to these is a more definite mental state, especially common at the onset of minor attacks, which sometimes apparently constitutes them, in which there is a sense of strangeness, of unfamiliarity—it may be with a place which the patient knows well.

Minor Attacks.—"Epilepsia minor" is the name applied to the attacks in which there is no visible convulsion. *Petit mal* is often used in the same sense, because the French made us familiar with such attacks, and we have therefore called them by the French name. There is no definite distinction between the two classes; spasm may occur and yet be unseen, and slight momentary visible tonic spasm may easily raise a doubt into which class the attack should be placed. It would indeed be well to recognise an intermediate class, which might be called *epilepsia media*, in which there is muscular spasm of tonic character, without the clonic spasm which follows when the tonic spasm is more severe. In the epilepsy which is due to organic disease the slight attacks often consist of clonic or tonic spasm only, but these differ in so many particulars from the slight attacks of the idiopathic disease that it is not practicable to consider the two together.

In the popular nomenclature various names are given to these attacks, according to their chief characteristic. When they begin with a sensory aura, of which only the patient may be conscious, they are called "sensations." If loss of consciousness occur they are called "faints." "Turns" is another name for them, which seems only due to the common use of the word as a designation for any sudden unusual falling. It is always important to ascertain what word is commonly used. It should be a matter of habit never to use the word "fit" or "epilepsy" unless it be known that it has been employed before in the patient's hearing. Friends often conceal the nature of the attacks, and when their nature is not known by the patient, it is wise to avoid so painful a word.

Varieties.—The forms of minor attacks are so various as to baffle classification and preclude full description. The typical attack is a momentary loss of consciousness, causing a momentary cessation of conversation; often a vacant look, or the dropping of anything from the hand, and occasionally a fall. There is no pallor of the face at the moment, although this often succeeds the attack, which is so brief that a patient is only observed after it. Hence the erroneous idea that pallor is a symptom of the attack itself. Nor is there any change in the pulse at the time, or in the aspect of the retinal arteries, as I have been able to observe. Of such loss of consciousness a patient may be wholly unaware, so sudden is its onset and ending. He may go on with the sentence he is uttering, and be astonished to observe that those about him are looking at him with concern.

Such change in consciousness may be incomplete. It may not be lost, but only rendered for the most part imperfect, and the character of the imperfection varies in different cases. It may be purely mental; the patient sees that which is about him, but, as already mentioned, it seems strange. Much less frequent is the curious sense that he is doing something wrong. There may be, in other cases, a more complete but more partial change; there may be sudden darkness for a moment, or a sudden silence, sometimes followed by sudden darkness, from the inhibition of the centres for sight or hearing, or both.

In a large number of cases the onset of the minor attack is accompanied by some sensation which the patient remembers. This must be ascribed, of course, to a discharge in the part of the brain with related function. There may be a flash of light, stars, a gleam of colour, or some more complex sensation such as has been described. This may precede loss or impairment of consciousness; the sufferer may even go on with his conversation, and others may be unaware of the attack. If there are also severe attacks, the sensation may be the same as that which precedes these; but occasionally it is not the same, even if the severe fits are preceded by a well-marked aura. A common warning of both forms is the epigastric sensation.

Vertigo is a frequent momentary symptom of the minor attacks. It involves, by its nature, impairment of consciousness in so much as it depends upon an erroneous sense of the relation of the individual to his environment. It may be various in character, subjective or objective, but is of great importance on account of its frequency, and in that it may not be associated with severer attacks in the same person. Indeed this is true of all varieties of minor epilepsy, and makes it necessary to put leading questions to ascertain their occurrence. The important characteristic is that they occur without any exciting cause; vertigo, for instance, may arise without any sudden movement to produce it. In all attacks in which there is loss of consciousness there may be micturition during the attack.

Local discharges, which cause the local commencement of convulsions that become general in severe fits, constitute, when they occur alone, the minor form of these attacks. If severe fits begin by clonic spasm in the hand, extending through the whole side and then to the other, the slight attacks consist of convulsion limited to the arm. Again, if the attacks begin with a sensation of tingling which passes up the arm and is followed by spasm, the minor form consists of the sensation alone, which often has the effect of inhibiting the related motor centre, so that the arm hangs powerless by the side. So likewise with the initiatory phenomena in other parts.

Such slight local discharges, which do not spread beyond the centre in which they arise, are commonly unattended with loss of consciousness. Their frequency is often great, especially when they are due to an active morbid process which irritates, but progresses slowly. In such a case (of cerebral tumour) I have known 19,000 attacks to occur during eleven months.

Post-epileptic Symptoms.—After a severe attack the patient lies exhausted and prostrate. The exhaustion may involve the spinal cord so that the knee-jerk cannot be obtained. When it returns it is excessive, apparently from the lack of higher restraint; and after attacks of moderate severity there is commonly a foot-clonus. It lasts longer on the side most convulsed, that is, the side towards which the head has been turned in the fit. When a convulsion is unilateral, the resulting feebleness may be very conspicuous and last for hours, "post-epileptic hemiplegia." It is important, however, to note that the sensory discharge may inhibit the motor structures, and it is probable, in cases that are slight, that inhibition enters into the condition which is generally regarded as solely due to exhaustion. A deep sleep follows, into which the coma may pass, and often headache succeeds it. In many cases there is little sleep but much headache, and this is often severe if the patient is prematurely roused. There is general sense of intense fatigue. Occasionally vomiting occurs before consciousness is recovered, or before the comatose state has passed into a sleep not too intense for the vomiting to awake the sufferer. Food which is then brought up may easily get into the larynx and cause fatal suffocation.

One other action after an epileptic fit, although rare, is important. A patient who is in bed or lying down has occasionally a tendency, immediately after the fit, to turn over on the face. The post-epileptic slumber may be so deep that even the progress of suffocation does not rouse him; asphyxia intensifies the stupor, and death results. Rare as it is, this incident is of much practical importance. Whenever a person, supposed to be in good health, is found dead in bed, with his face against the pillow, death was probably the result of such suffocation after a fit, even if the individual was not known to be the subject of epilepsy. The post-epileptic coma may also be intensified to a fatal degree by the administration of even $\frac{1}{4}$th of a grain of morphine before the fit. For this reason extreme care is necessary in the administration of morphine to epileptics, and if a fit follow an injection the patient should be carefully watched.

Automatism.—After slighter attacks, and indeed in proportion to the slightness of the attack, provided there be loss of consciousness, a peculiar state of automatic action is apt to ensue. This is apparently due, as Dr. Hughlings Jackson has suggested, to the weakening of the control normally exerted by the highest centres. It is not certain, however, that the abnormal action of the lower psychical centres may not depend on a primary insubordination; for attacks occur in which, however carefully observed, such automatic action does not seem to be preceded by any indication of a fit.

The simplest form is that in which a person goes on with some action on which he was engaged at the time, and with every manifestation of consciousness, yet after suddenly recovering himself, cannot remember anything of what has occurred. Thus, a man drove a waggon across London, and then found himself six miles from the place where he was, as it seemed to him, a moment before. The "solution of continuity" in the self-consciousness may occur after the slightest form of minor attack. Often some simple action occurs, unconnected with that which has preceded the attack—irrelevant speech which has no relation to the subjects engaging the attention, or some unintelligible words. There is often a fumbling action of the hands, or some apparently purposive action which may be highly inconvenient. Taking off the clothes is especially common, perhaps from some vague sense of indisposition and the propriety of going to bed. A music master had to give up his profession on account of the liability to this equivocal proceeding when he was giving music lessons to young ladies, by whom the momentary attack was unperceived. It is in this state that acts of violence may occur, as in the case of a patient, who in the out-patient room of the hospital attacked the physician with a poker. One man had a sudden slight attack, which caused him to stand still while crossing a crowded street. A passer-by took his arm and assisted him to the pavement, but no sooner had they reached it than the helper received a violent blow from the man whom he had assisted. In this state crimes may be committed, and when the excitement is pronounced the state has received the name of "epileptic mania."

Post-epileptic Hysteroid Convulsion.—This condition of brief insanity, the sequel of an attack of minor epilepsy, is pathologically related to another sequel. In those who are of the age and sex in which that state of the brain exists which underlies the peculiar symptoms termed "hysteria," namely, a few young men and most women, a slight and even a moderate epileptic fit may be instantly followed by hysteroid convulsion, quasi-purposive energetic movements, throwing about of the arms, kicking of the legs, arching of the back, varied with wild talking or manifestations of horror or fear. Such a state may last for a few minutes or for an hour or more. The initial attack is often unnoticed. Minor epileptic attacks in childhood, to which this sequel is added at puberty, are often then thought to have become severe epileptic attacks. These cases are extremely common, and almost always misunderstood. From the character of the obtrusive symptoms they are regarded as purely hysterical: yet whenever a patient has such attacks, recurring for years, it is practically certain that the essential disease is epilepsy. The difficulty of the diagnosis is, moreover, increased by the fact that, under the training which the brain thus undergoes, such patients may occasionally have independent purely hysterical attacks.

These cases are the only representatives of the hystero-epilepsy of the French which we meet with in this country. In the purely hysterical convulsion there is nothing, even in the initial stage, which an observer accustomed to epileptic fits does not recognise at once as wholly different. But the term "hystero-epilepsy" should not be applied to post-epileptic hysteria.

Excitants of Attacks.—In idiopathic epilepsy attacks usually occur without obvious exciting cause. Nevertheless sometimes gastric irritation, an excess in alcohol, or sudden mental emotion may induce a seizure. It is important to know that, even in true epilepsy, annoyance or a sudden fright may cause a fit. There is a strong but unjustified tendency to regard fits excited by emotion as hysterical.

Arrest of Attacks.—The commencing local convulsion, or sensory aura, which is due to local organic disease of the brain, may often be arrested by a strong sensory impression on the limb above the part. A ligature is the easiest and apparently the most effective; perhaps partly because the sensory impression is produced on the entire circumference of the limb. This arrest may be obtained even in cases of cerebral tumour. Apparently the excitation of the sensory structures causes a motor inhibition in the path of the discharge, just as a sensory aura in the arm, too slight to excite motor discharge, may arrest the action and render the arm helpless.

In idiopathic epilepsy the warning seldom endures long enough to permit any measures for arrest to be adopted. Vigorous movement occasionally stops a commencing attack—an effect which may be due to a diversion or dissipation of some of the nerve energy of the motor structures of the cortex ready for liberation. A strong sensory impression may be effective in such cases also, especially one which acts upon the fifth nerve. Smelling-salts, a mouthful of common salt, or even a splash of water upon the face may be sufficient.

The only available drug, which acts with sufficient rapidity, is nitrite of amyl by inhalation. It is intelligible that the sudden flushing of the brain with arterial blood and the sudden influence of the mechanical distension of the arteries, should disturb and arrest the abnormal action. It is only available when the warning is prolonged, and it does not always succeed.

Course of Epilepsy.—State.—Severe attacks occur in the waking state alone twice as frequently as in the sleeping state alone. About 0·3 occur in both waking and sleeping, about 0·2 in sleeping only, and nearly 0·5 only when the patient is awake. Minor attacks are almost confined to the waking state. Attacks which have occurred only in one state seldom change to the other, although the first attack may occur in the state in which subsequent attacks do not occur. Attacks which occur only in sleep may so change as to occur also in waking hours; it is rare for attacks which have occurred only by day afterwards to occur in the night also. Infrequent attacks during the night, in those who sleep alone, may go on for twenty years without their occurrence being suspected.

Menstruation.—It is common for attacks in women to occur either before, during, or just after the menstrual period, simply because the nervous system is then generally disturbed. It affords no proof of a relation to uterine or ovarian disease, of which indeed definite evidence is very rare.

Pregnancy.—In most women who are subjects of epilepsy, the fits occur during pregnancy with the frequency unchanged. Occasionally they cease during pregnancy, and if they have commenced during pregnancy they may sometimes be confined to that state for two or three successive periods. Afterwards they usually occur at other times. They do not seem to have much tendency to cause abortion. Attacks may or may not occur after delivery; when they do, they seldom have any unfavourable effect.

General Course.—Puberty.—In very intimate connexion with the occurrence of fits at the menstrual period is the relation to the course of the disease of the commencement of menstruation. When a girl has become subject to fits in childhood, the hope that they will stop on the establishment of the catamenia is generally entertained, and is sometimes encouraged by doctors. But we have seen that this period is one at which there is the greatest tendency for epilepsy itself to commence. The conditions favourable to its onset are not likely to be favourable to its cessation. This is entirely borne out by facts. If the disease does not cease before puberty it is not likely to be arrested for some years afterwards.

Tendency.—The facts of epilepsy shew it to be a self-perpetuating disease, maintained and increased by a sort of habit developed in the brain. We have seen also that from what we know or conjecture of the minute processes which underlie these and other functional actions of the nerve-centres, it is easy to conceive that this self-perpetuation is inevitable. Thus the natural tendency is for attacks to become more frequent or more severe. And yet discerning, as we do, how much there is in the conditions of action and nutrition of the central nervous system that is quite unknown to us, we shall be prepared to meet with facts which seem inconsistent with our most careful inductions.

The disease often begins by minor attacks which may exist for years without severe seizures. The slight attacks may pass gradually into severe fits, or severe fits may commence and the minor attacks may cease. Conversely, minor attacks may replace convulsive seizures, and often do so as the result of treatment. In some cases, only too rare, all attacks may cease: we know not why. This event is scarcely ever spontaneous, except in adult life. Since attacks go on through adult life, and the disease may begin at any period of adult life, it is clear that such cessation must be due to conditions and influences as yet unknown, which may neutralise the self-perpetuation, and may invalidate our forecasts.

The tendency to the cessation of attacks is greatest in the period between the completion of the structural development of the nervous system and the establishment of its chief functions; that is to say, in the period between the first three years of life and the second dentition. There is a slight tendency to cessation (manifested chiefly by the greater influence of treatment) between 10 and 13. In the following three years attacks are less likely to cease. After 16, however, treatment has a better chance of doing definite good. The more favourable conditions seem to persist through adult life, and at any time the malady may be arrested. There seems to be more prospect of arrest, however, between 30 and 45 than in later life.

It will be noticed that much of what is said of the course of the disease properly belongs to the prognosis. This is inevitable, because in the vast majority of cases the common treatment relieves when it does not cure. When it is intermitted the attacks become more severe or more frequent, or both. Observations on the course of the disease are thus of necessity observations on its course as influenced by treatment.

Frequency of Attacks.—Severe attacks occur at intervals which vary from a few days to a year or even more. In the majority of cases the interval is between a week and a month. The interval is irregular in most cases. It is not uncommon for only one or two attacks to occur each year. But this fact is also true of the disease as influenced by treatment. In many patients the fits occur in groups; from two or three to ten or more may occur in a period of two or three days, and then no more for weeks or even months.

Minor attacks also present extreme variation in frequency. They may occur daily, and even many times a day; or at intervals of days, weeks, or months. In some patients they occur especially before or after a severe attack. They often become more frequent when severe fits are less frequent and vice versa.

Status Epilepticus.—The subjects of epilepsy, chiefly those who have passed early childhood and are not far beyond the completion of full development, occasionally have a series of fits in close succession. Before recovery of consciousness after one fit, another occurs, and the rapidly recurring attacks may go on for several days. The temperature rises two, three, or four degrees, and the patient is reduced to a state of extreme exhaustion, which may result in death. Fortunately this "epileptic state" is very rare. Nothing is known of its immediate causes.

Mental State.—The relation of mental disturbance to epilepsy is discussed in another article (p. 941), and the post-epileptic state has already been spoken of. Many sufferers from the disease are naturally weak-minded. The predisposition which underlies the instability of the motor and other structures, and is the remote and chief cause of the disease, is also manifested in imperfect function. It is not difficult to understand that this should be. But persistent mental feebleness may be a result of the attacks. Severe fits seldom cause it, but the result is common from minor seizures. It is not a necessary effect; several minor attacks may occur every day for years without the slightest influence on the mental power. We cannot yet say why the effect sometimes results. It especially follows attacks in which there is momentary loss of consciousness only, and this may be referred to the influence of the discharges on the structures of the brain which subserve the highest cerebral function; yet precisely the same attacks may occur for years with no apparent influence. The most important practical fact connected with mental feebleness is the frequency with which it is manifested when attacks cease, which have been going on for a long time. It is met with when the cessation is spontaneous, and therefore, although it is commonly ascribed to any medicine which has caused the cessation, its chief cause is probably the repression of the discharges to which the brain has been accustomed. Nerve energy must be evolved, ready for escape, as the result of previous liberation. If this escape be prevented, and yet the processes for the production of nerve energy continue, it is easy to understand that there may be a widespread interference with the activity of the brain. As far as we can perceive, medicines act by repressing the discharge, and it is only after a time that the energy for the discharge ceases to be generated. The depression of function of the brain influences the whole nervous system and the whole body. It is a temporary effect, but often a source of undue concern to the friends, and it may be so severe as to necessitate diminution of the treatment. After a severe convulsion the patient is at once better, as if a depressing cloud had passed away.

Causes of Death.—Alarming as is the aspect of a patient in a severe attack of epilepsy, death very seldom results from the violence of a fit, and the danger in any given fit is negligible. This is true even of patients with grave heart disease. In the status epilepticus death does sometimes occur from exhaustion. When epilepsy ends life it is generally by indirect means. A fit may cause a fatal fall or burn or death by drowning, even in a shallow pond; it may cause death by suffocation in consequence of vomited food getting into the larynx, or through the patient turning over with the mouth against a pillow. Yet when all these causes are taken into account, it is remarkable how few persons die in consequence of this common disease. We have, indeed, imperfect knowledge of the causes of death of epileptics, but it is certain that even severe epilepsy may persist through life and may not prevent the attainment of old age.

Secondary Forms.—We have seen that convulsions, however produced, become perpetuated by the influence on the cerebral nutrition and functional tendency of the recurring discharges. By reason of this, the convulsions which are not idiopathic may become recurrent, after their cause has ceased, as a malady, indistinguishable from idiopathic epilepsy.

Reflex Epilepsy.—One class of such secondary attacks is that which results from some peripheral irritation, and is called "reflex epilepsy." Convulsions thus produced are almost confined to the first half of life and to irritation of the gastro-intestinal canal. The most common cause is tapeworm. If the attacks persist after the expulsion of the worm, they do so in consequence of the tendency established in the brain which cannot then be considered as essentially different from that of idiopathic epilepsy. Recurring convulsions from irritation of one of the cerebro-spinal nerves are extremely rare; and it is enough to say that the same statement is true also of these. Of epilepsy due to disease of the uterus or ovaries nothing is seen except by gynaecologists.

Toxaemic Epilepsy.—The blood states which cause convulsions generally also cause death. It is possible, however, that some chronic blood states give rise to fits quite similar in character and course to those of idiopathic epilepsy. There is reason to think that such fits occur in some cases of chronic renal disease, and are due to a variety of toxaemia which differs from common uraemia. Lead poisoning may have the same effect. But of these events, also, it is often true that, if the patient recover from the blood state, the fits continue by the same induced state of the brain as in the idiopathic disease.

Chronic Alcoholism is an occasional cause. Often the signs of chronic cortical meningitis are also present. In other cases, of occasional alcoholic excess, each outbreak is followed by a series of convulsions. *Absinthe* drinking in France is found to be a frequent cause of epilepsy.

Organic Disease of the Brain.—The convulsions due to active brain disease, generally to a tumour, cannot be regarded as properly within the subject of epilepsy. Nevertheless a very slowly-growing tumour may produce attacks which are difficult to distinguish from those of epilepsy, and occasionally indeed cannot be distinguished from them. This is especially true of tumours of the frontal lobe, which may cause attacks resembling those of minor epilepsy. But stationary lesions of the brain are frequent causes of recurring fits. The disease is now and then a tuberculous tumour which has ceased to grow; occasionally such fits are due to old chronic meningitis on the cortex of the brain. In the vast majority of cases the lesion is a spot of old softening in the cortex, the result of a vascular lesion, generally a thrombosis which occurred in early life, or of an injury. If this involve the motor centres there is initial hemiplegia, partial or at first complete. But more often, as already stated, the lesion is not in but near a motor centre, and damages the grey matter only in slight degree. It is the slight damage, one which impairs power only slightly or not at all, which is the chief cause of subsequent discharges. Hence it is not often that such convulsions are met with in association with persistent severe hemiplegia; they may succeed slight hemiplegia, but often there is no history even of that. Cases of this class may be called "organic epilepsy."

The convulsions of this class are generally unilateral when slight, bilateral when severe. The secondary tendency to extension of the discharge through the brain, after a few years, may be so great as to cause the fits to become so quickly general that local or one-sided commencement is seldom observed. The effect of such repeated discharges is to induce a general state of the brain similar to that of idiopathic epilepsy, with forms of minor epilepsy, such as momentary loss of consciousness, met with in idiopathic cases. This secondary state is further illustrated, in an important manner, by the fact that, after the convulsions have thus become quickly general, or minor attacks have been established, the removal of the part of the cortex from which the discharges proceed may not arrest the occurrence of the fits, or may only do so for a time. Hence the operation does not justify any promise that the attacks will cease.

Diagnosis.—The diagnosis of epilepsy involves, first, the recognition of attacks; and, secondly, the recognition of their nature.

Occurrence of Attacks.—(*a*) During sleep. A severe epileptic fit is a conspicuous event if it is observed. But a solitary sleeper may know nothing of the occurrence of an attack, and it is important to remember the indications that one has taken place during sleep. They are—waking in the morning with a bad headache and a general sense of fatigue or soreness; evidence that there has been micturition in the night without the patient's knowledge, provided he is not a young child; considerable and unusual disarrangement of clothes; soreness on the side of the tongue; moisture or spots of blood on the pillow. Such evidence of night attacks may occur for many years without suspicion of their significance.

Minor attacks often escape recognition. They may be so slight as to be noticed only by the patient; if observed by others they are thought to be simply giddiness or faints. It is generally necessary to mention to the patient several common symptoms of such attacks, and to ascertain if he can remember experiencing any one of them. If there are such brief suspicious symptoms, their significance depends largely on their occurrence when a patient is tranquil and not subjected to any adequate cause. It should never be forgotten that it is very rare for a patient or the friends to suspect that there is any relation between the minor and major attacks, or that the minor attacks have the slightest importance.

From Hysteroid Fits.—The convulsions of hysteria and epilepsy can scarcely be mistaken by any one who is acquainted with their features, and has an opportunity of observation. In hysteroid convulsions the spasmodic movements are such as might be produced by the will, they present what may be called a "purposive" aspect, quite different from the strong warping tonic spasm which constitutes the first part of a severe epileptic fit, and changes gradually to the clonic spasm of shock-like character. Nor is there in hysteroid fits the strong deviation of the head and eyes to one side, or the frothing of saliva, often blood-stained, from the lips—except possibly in rare cases of voluntary imitation of attacks that have been seen. If a severe epileptic attack begin with clonic spasm, this is local at the onset, and the quick jerks, spreading from the place where they begin, bear no resemblance to the muscular contractions of a hysteroid fit. In the former the patient has only to be kept from accidental injury; in the hysteroid attack, forcible restraint is often necessary. The patient has to be "held down," and in this the essential difference in the character of the convulsion is revealed. Moreover, the duration of a single epileptic fit seldom exceeds three or four minutes, whereas most hysteroid attacks last a quarter of an hour, and often continue much longer.

A special diagnostic difficulty is involved in the occurrence of hysteroid attacks of characteristic form, as the immediate sequel to minor attacks of epilepsy. These may be so slight in degree as to be unnoticed, whilst the hysteroid symptoms are obtrusive and are alone described. It is, however, common for such patients to have, at other times, attacks of *petit mal* without the hysteroid sequel. This can be ascertained by inquiry, although their relation to the other attacks is seldom suspected by the friends of the patient. It is also common for more severe epileptic fits, with tongue-biting or micturition, to have occurred at some time, a history of which is conclusive. That which should always cause a suspicion of the real nature of the case is the course of the disease. Pure hysterical attacks do not recur in such a way as to constitute a prolonged disease. Whenever a patient has been subject for years to attacks, hysteroid in their obtrusive characters, the malady is epilepsy with the hysteroid sequel to minor attacks.

It is most important to know that at puberty the hysteroid condition is often superadded to the simple minor attacks of childhood. The account given by the friends seems to mean that previous attacks of *petit mal* have become severe convulsive fits. Sometimes it is so, but in many cases there is only an addition of the hysteroid to the epileptic attack. The practical importance of the diagnosis is great. To do good the epileptic element must be treated; the hysteroid may be disregarded.

Vertigo is a common subjective symptom of a commencing epileptic seizure when loss of consciousness is not the first effect. When the vertigo is obtrusive, it may be a question whether the attack is one of epilepsy or of labyrinthine vertigo. The difficulty is increased by the fact that in rare cases the vertigo of epilepsy may be attended with a sudden noise in the ear. In aural vertigo loss of consciousness is very unusual, and even impairment only occurs in attacks of extreme severity. These present vomiting and persistence of the vertigo long after consciousness has become normal, a symptom which alone excludes epilepsy.

Syncope, simple fainting, is generally due to a definite cause, such as emotion, a hot room, and the like. It occurs in those who are deficient in strength. Loss of consciousness is not instantaneous, and on its return consciousness is perfect in character from the first, although its degree may only slowly reach the normal. There is not the "dazed" condition, which is a characteristic of the recovery from the minor epileptic seizure. Involuntary micturition never occurs during the unconsciousness of syncope, but is not rare in that of epilepsy. There is never any automatic action after simple syncope.

Other diagnostic problems occur so rarely that it is needless to discuss them, especially since they are for the most part included in this aphorism, that the great characteristic of epileptic attacks is suddenness of onset and brevity of duration. To this may be added spontaneity of onset, provided it be remembered that a sudden emotion may excite an epileptic attack in a patient whose seizures are generally without excitation.

Prognosis.—The prognosis of epilepsy is a question of therapeutics. That which we can perceive of its course and infer regarding its nature shews it to be a self-perpetuating disease, in which no expectation of spontaneous cessation can be entertained. This conclusion is undisturbed by the fact that the disease may now and then cease spontaneously; this issue is so rare, and the conditions under which it occurs are so little known, that it cannot enter into a practical forecast.

The recovery from the disease is freedom from fits without treatment, yet in no disease does treatment enter into the prognosis in the same degree. Hence in most of the cases in which the prognostic problem involves that of cure, the prognosis is determined by the conditions of the patient, and especially by the probability of adequate treatment. In this disease, as in many other diseases of the nervous system, but more than in most, observation of the present alone can afford an accurate forecast of that which is to be. Only when every kind of attack is prevented is a patient on the road to a cure. As long as the slightest seizures occur, severe attacks would be certain were the patient to leave off taking the medicine which has arrested them.

But treatment may produce a state which, however far short of a cure it may be, often involves a vast difference to the subject of the disease. The ability to work and the capacity to enjoy life may be regained, with, as a consequence, the relief of the friends from the pressing anxiety that is involved in the frequent occurrence of a fit. By treatment epilepsy is sometimes cured, but life is often transformed; and how precious this imperfect result frequently is can only be discerned by those who have witnessed the gratitude it evokes.

The shorter the duration of the disease, and the less frequent the attacks, the brighter is the prospect of their permanent arrest. It must be remembered, however, that the less frequent the attacks the longer has treatment to be maintained before its influence can be seen, and therefore the more difficult is it to secure perseverance. The prognosis is better if attacks occur only in the night than if they occur only in the day, or in both sleeping and waking states. It is better in idiopathic epilepsy than in the form which is the result of some old cerebral lesion.

The most unfavourable feature is the occurrence of minor attacks.

These are often not influenced in the least by the treatment which stops the severe ones, indeed they may become more frequent. So long as they continue, if medicine is stopped, severe attacks will soon recur. Hence the extreme importance of their recognition.

Treatment.—The tendency to the recurrence of convulsions, which constitutes the disease we call "epilepsy," can only be influenced by the administration of drugs; that is, by bringing certain chemical substances into artificial relation with the nerve elements concerned in the disease. The sketch of the nature of the disease which was given in the introduction should make the fact intelligible. Action is due to chemical union; instability to too ready union; stability is due to some unknown mode of restraint of the tendency on the part of the atoms of the nerve elements to unite with the oxygen brought to them in the plasma with which the nerve structures are bathed. From this plasma come the molecules which they appropriate for the maintenance of their nutrition and of their capacity for action. The nutrition of the elements and the replacement of lost elements is under the inscrutable influence of life, and so also is the liberation of the latent chemical energy when atoms escape.

Mysterious as is the process, its control is not less so. The influence of the alkaline bromides of potassium, sodium, and ammonium is one of the most remarkable and definite therapeutical discoveries of the last century, and it was purely empirical. We have no evidence that the bromide salt is decomposed in the system in appreciable degree, or that its influence depends on any separate influence of the bromine. We can discern no room in the constitution of the nerve substances for the assimilation by it either of bromine or of its salt. We may conjecture that the salt exerts its peculiar influence as such; as such it restrains the sudden explosive activity. How this restraint is exerted we cannot comprehend. It may, however, be connected with the important part played by chloride of potassium in the intimate function of the central nervous system, and in the electrical processes which are induced by injury to nerves.

The three alkaline bromides present little difference in the influence they exert. In general, of those in common use, the greater influence seems to be in proportion to the amount of bromine in the salt; that which is most effective is bromide of sodium: and bromide of ammonium sometimes seems to have a greater influence than bromide of potassium. But bromide of calcium contains a larger proportion of bromine than the others. The difference, however, is very slight, and is, moreover, of small practical importance. A combination of the salts has been recommended to prevent deleterious influence of the bases, but there is no evidence of such influence, and we do not know that there is enough decomposition of the compound to permit the base to have a special effect. It is strange, indeed, to note how little evidence there is for opinions that are widely acted upon. Bromide of strontium has been recommended and widely used, but any evidence of the superior value of this salt is difficult to discern on extensive trial. Ammonium bromide is thought to be least depressing. Calcium bromide may be given with reason in cases with adolescent albuminuria, and often seems most effective in the young; sometimes magnesium bromide answers well, especially when the digestive system is easily disturbed.

Whichever bromide be employed, it must be given continuously; that is, the influence must be maintained without interruption. It is necessary to continue the regular administration of it for two years after the occurrence of the last attack of any kind, and then to spend another year in gradually diminishing the dose. Not until the end of the third year is it reasonably safe to omit the medicine altogether. This precaution applies to every system of treatment; there is no short method of cure. The temptation to leave off the medicine is very great when the attacks have ceased; but it is then that the prospect of a cure makes persistence of the greatest importance. The duty of perseverance must be enforced upon the patient or his friends. Moreover, it is necessary to arrest all attacks, the slight as well as the severe; so long as minor attacks occur, it is practically certain that a severe attack will soon follow discontinuance of the treatment. During any acute febrile illness, while the temperature is raised above 100° F., there is little tendency to the occurrence of attacks, and the bromide may be reduced or omitted with impunity, but it must be resumed as soon as the pyrexia ceases. Relapses are frequently due to a delay in its resumption.

The bromide may be given once, twice, or three times a day according to the frequency of the attacks. A single dose will generally suffice if the interval between them is several months; but if it be less than two months two doses should be given. There is little difference in effect between two doses and three doses a day provided the amount of bromide administered is the same. Two doses of 30 grains have generally the same effect as three doses of 20 grains. A single dose may be given either at bedtime or after breakfast, according as the attacks occur in the night or in the day, or even on first waking if the attacks occur soon after rising, as is often the case.

The object is to arrest the attacks, and also to afford a margin of stability sufficient to resist an occasional exciting influence. On the other hand, it is important that the dose given regularly should not be larger than is necessary to achieve this object. In many cases that which is necessary and not excessive can only be ascertained by experience in the individual case. But the largest daily quantity which, as a rule, can be continuously borne with impunity is 90 grains—half a dram three times a day; and for many, perhaps for most patients, this quantity is too large. The ordinary maximum may be put at 50 or 60 grains a day. Curable cases do not need more; and when more has to be given, the tendency to the attacks is so strong that bromide alone only relieves and cannot arrest the disease. Relief may indeed be almost as important as cure.

The same daily quantity is sometimes better given in three doses, but it is not so easy to secure uniform regularity with three doses as with two. The system of increasing the dose when an attack is supposed to be impending is not to be commended. If such an increase be really necessary the constant dose is insufficient.

Some years ago I tried carefully the system of putting a patient through a course of increasing doses of bromide, in the hope of producing an effect on the nutrition and functional tendency of the nerve structures sufficiently great to be enduring. The dose and interval were increased until an ounce of bromide was given in nearly a pint of water every fifth day; a larger quantity was vomited. The result was not encouraging. After such a course of increasing doses, reaching the maximum at the end of three weeks, maintained there for about a fortnight, and then diminished through another three weeks, it was found that no lasting amelioration was produced. It was still necessary to use ordinary doses to prevent recurrence, and it was doubtful if any more good had been achieved than would have resulted from the administration of ordinary doses for the same time.

Most persons can bear 60 grains of bromide daily, for years, without the slightest interference with the functions of the nervous system. The arrest of attacks is indeed often followed by some depression of the functions of the brain and of the general system, naturally ascribed to the bromide. But if the dose prove insufficient, and another attack occur, the depression generally passes off. The action of the nerve-centres suggests that the repeated liberation of nerve energy in the epileptic fit induces an increased generation of latent energy, ready for similar release, which is only prevented by repression of its escape. We can understand that this may depress the general function of the brain until nutrition has become accommodated to the new conditions. It is important to realise this, in order not to misunderstand the effect which follows the arrest of attacks. The direct influence of bromide cannot, indeed, be excluded, but in most cases it is not the chief cause of the depression; this is in many cases an inevitable concomitant of the process of cure. But it is not enduring. It should be met by tonics; if these do not suffice, by a diminution in the dose of the bromide; only as a last resource should bromide be omitted, for this involves a risk of fresh attacks, which are less easily arrested a second time. It is important that this should be generally understood, because physicians are generally disposed to put all ill-effects down to bromide, and to advise its discontinuance, without any adequate reason; but the cessation of treatment may mean the destruction of the prospect of a cure.

But bromide has its drawbacks. It is not surprising that it should often interfere with the normal function of the brain; the marvel is that it should so often arrest the abnormal and leave the normal almost or completely undisturbed. However, such interference is especially evident in the large number of cases in which the occurrence of epileptic fits is the expression of a general qualitative deficiency of the brain. In these cases the weakness of the intellect and backwardness of development manifest a state in which bromide cannot restrain the abnormal without interfering with the normal.

These are the patients in whom it is sometimes necessary to lessen the medicine, and even to permit the recurrence of attacks on account of the depression and failure of memory that may result. There is no reason to believe that any other drug would be free from the same disadvantages: we have no reason to think that epilepsy could be cured by any agent except in the way that bromide cures it.

Another drawback is the familiar influence of bromide on the skin. Foci of suppurative inflammation, in which the formation of pus is small in proportion to the inflammation (bromide acne), are the common effect; but the process may extend over a large area, and give rise to a local dermatitis with the production of a semi-purulent secretion which dries into scabs. In children large and deep ulcers may be formed on the limbs and elsewhere. Beyond the fact that bromine has been found in the contents of the pustules, we have no knowledge of their precise causation. The tendency of the several bromides to produce this effect is in proportion to the amount of bromine they contain, but of the way in which the presence of bromine produces them we know nothing. Their occurrence, especially on the face and back, is doubtless due to the same conditions which make common acne most frequent in those situations. On the other hand, some persons present a perfect immunity from these lesions, and effects are chiefly seen in thick-skinned persons with abundant "comedones" upon the face. Why some persons never suffer is a problem which deserves more attention than it has yet received. The tendency to the acne rapidly diminishes after middle life.

The only preventive of the bromide rash is arsenic, in the proportion of 2, 3, or 4 minims of Fowler's solution to each scruple of bromide. No other expedient of any kind is even worth mentioning. In most persons the arsenic prevents the rash, and in others may reduce it to an endurable degree. But it must be continued as long as the bromide is taken, and it has its own evils. After some years arsenical pigmentation of the skin may develop, and sometimes we must allow the patient to choose between pigmentation and acne: the pigmentation is always preferred. It is extremely rare for symptoms of arsenical neuritis to result, so rare as to suggest that the bromide prevents the metabolic entrance of arsenic into the nutrition of the nerves.

Fifty years have passed since the influence of bromine on epilepsy became known, and for the latter two-thirds of that time its success and failure have alike stimulated an energetic search for that which should succeed when it fails, or reinforce it when inadequate. The repertory of therapeutics in the pre-bromidic days, and all the pharmacological ingenuity of the present time, have been brought to bear upon the problem. The result is not unimportant, and yet it increases by contrast the mystery of that which is obtained by the administration of the simple salt.

But the influence of bromide is sometimes increased by the addition of other agents. Of these the most important are belladonna, in doses of

5 minims of the tincture, and digitalis in the same dose. At present it is not possible to give trustworthy indications for the use of either. It deserves note that in the West of England digitalis is an old popular remedy for epilepsy. The addition of either to an unchanged dose of bromide sometimes causes the attacks to cease. Indian hemp seems also to be occasionally useful.

Biborate of sodium is perhaps the most effective of the drugs available in the inveterate cases in which bromide partially or wholly fails. It is one of a large number of drugs of which I have made careful comparative trials; it was first recommended by me in 1880.[1] In doses of 4 to 10 grains three times a day, it often very much diminishes the frequency of attacks; but it does not seem to be capable of arresting them. In this incapacity it corresponds with all other drugs. The only drawbacks to borax are trifling intestinal irritation, and occasionally a very typical psoriasis which readily ceases if arsenic is added.

Of the agents of repute in the pre-bromidic days little need be said of the chief one, nitrate of silver. Forty years ago patients with skins stained by silver were occasionally still suffering until treated by bromide. We may reasonably conclude that silver does not offer us benefits commensurate with the inconvenience of it. The chief drug which has survived the introduction of the bromides is zinc, although alone it rarely succeeds. The lactate of zinc, advocated by Herpin, is often well borne in doses of 5 to 7 grains three times a day, and it seems sometimes to add definitely to the effect of bromide. The bromide of zinc is more soluble, but is not so well tolerated. It may be given for the sake of the zinc. On the whole the oxide and citrate cause least irritation.

The salts of zinc, however, seem to have more influence upon the minor attacks than on those that are severe. The influence is not great, but sometimes definite; and nothing is to be despised which can influence these seizures.

Belladonna and Indian hemp, also, in some cases have a definite influence on minor seizures; so likewise has morphine in doses of 5 to 10 minims of the solution, but it is undesirable. The hydrobromide of hyoscine is rarely useful. Nitroglycerin deserves trial in many cases, especially in children with frequent minor seizures, and in any cases in which symptoms of vascular disturbance are unusually prominent. The dose may be from ½ to 1½ drops of the 1 per cent solution. It may be combined with bromide provided some hydrobromic acid is added to prevent alkalinity. Strychnine or nux vomica harmonises well as a tonic. The immense number of other therapeutic agents which have been extolled may be passed in silence. Most of them I have tried in vain, as well as a large number of drugs of which nothing has been heard.

Operative Treatment.—It would be a waste of space to describe the various operations that have been advocated, whether on arteries or on the sympathetic nerves, "which have their day and cease to be," not much to the credit of the profession.

---

[1] Goulstonian Lectures (8). The fact is scarcely worth mention, but is referred to because its use seems to be thought, by most of those who have written upon it, to be a relic of antiquity.

One surgical procedure, however, stands on a different level. Trephining the skull is an ancient measure, undertaken ages ago, perhaps to let out an evil spirit. It has been revived of late to permit the removal of an evil substance. When the disease can be removed, and there is a reasonable prospect that the effects will not survive the primary cause, the operation is justified alike by reason and result. Such is the case when there is an irritating body which can be removed, be it a spiculum of bone or a stationary tumour. Thus it is in the case of a spot of softening, which causes only local discharges manifested by local convulsion. But when such disease, although removable, has so trained the whole brain into a habit of discharge that the attacks differ little from those of idiopathic epilepsy, the chances of the success of an operation are small. A temporary arrest is followed by recurrence, and the only result is to add more or less hemiplegic weakness.

In cases of idiopathic epilepsy trephining has been known to be followed by cessation of the attacks, but the effect has been seldom proved to be permanent. Most cases are published before a sufficient time has elapsed to permit the effect of the operation to be estimated. It is doubtful if its action is other than as an energetic counter-irritation. Failures, often unregarded, are too numerous to bring the procedure within reasonable range, except perhaps in cases in which nothing is too grave to counterbalance the influence of the disease on the life of its subject.

In cases in which some disease in the region of the cerebrospinal nerves is distinctly the starting-place of fits, and the disease is such as can be removed, it would be certainly right to rid the patient of the apparent cause, or to arrest its influence by resection of the nerve so implicated. Such cases may be met with, but they are extremely rare. The few cases on record have impressed themselves unduly on medical literature.

The status epilepticus seldom yields to bromide, or indeed to any drug except to chloroform, or to the hypodermic injection of morphine or of hyoscine. The dose of morphine should be small, not more than $\frac{1}{12}$th of a grain, repeated, if necessary, every three hours. Hyoscine hydrobromide is often effective, $\frac{1}{150}$, $\frac{1}{100}$, or $\frac{1}{70}$ gr. being injected. Among other measures which deserve trial, however, are antipyrin and nitroglycerin. The latter is less transient in its action than nitrite of amyl, and seems to change the functional state by flushing the nerve-centres with arterial blood. Inhalation of oxygen may also do good, but the means are seldom at hand.

General Management.—The diet of the epileptic should be free from all that is obviously indigestible—raw fruit of firm texture, such as raw apples; the skin of dried fruit, including currants; pastry, except in small quantities, and tough meat. It has been recommended, on theoretical grounds, that meat should not be taken, but a careful comparison of a number of suitable cases for successive periods with and without meat, shewed that the only difference to be observed was that the attacks were, on the average, a little more frequent and severe when meat was withheld. It may certainly be allowed with advantage, in moderate quantity, once a day. But I have met with more than one patient who could not take beef without having an attack, although he could take other meat, and many patients find it wise to abstain from beef.

It is important that food should be taken slowly, and that it should not be too large in amount. Many young epileptics have a peculiarly ravenous appetite, which should be restrained. Stimulants are better avoided. Regular action of the bowels is of great importance, and should be secured by a daily mild laxative. The omission from the diet of common salt has been recommended, but I have never seen any benefit therefrom.

The life of the epileptic should be as uniform as possible ; irregularity in meals, over-strain in work, and excitement of every kind should be carefully avoided. Exercise is important, but over-exertion should be avoided. So with the mental training of the young, which is often an anxious matter. Education may go on, without injury and even with advantage, if all strain is excluded, and if it be remembered that the excitement of the playground may be more harmful than the mental work of the school. The same general rule applies to the life of older persons.

As little difference as possible should be made in the mode of life, but it is generally necessary to avoid crowded rooms and the vertiginous influence of the dance.

A knowledge of the nature of the malady is often carefully withheld from the sufferer. But such ignorance is undesirable if the needful care cannot be secured unless the patient knows from what he is suffering.

Occupation.—The first point is that the occupation of an epileptic should be one in which attacks shall not involve personal danger to life. This precludes a life at sea, on scaffolding, or about machinery. For the most part choice is restricted to sedentary occupations. The only wise rule is to assume that the attacks will persist, however strong is the hope of their arrest. If this be done, the course of the disease is unimportant so far as the life-work is concerned ; if it be disregarded, years of training may be wasted, and life may have to be recommenced too late.

Marriage.—The question of marriage of epileptics is one of great difficulty. The influence on the individual sufferer is unimportant. The chief consideration is the probability of the transmission of the disease, or of its congeners, insanity and idiocy. The subsequent influence of such transmission on its subject, on the happiness of the parents, and on the welfare of the race, has also to be thought of. When epilepsy is the result of an old organic cerebral lesion, however early in life this has occurred, it may be disregarded so far as transmission is concerned. The difficulty chiefly arises when there is evidence that the disease is distinctly inherited. Regarding the risk of transmission in such cases, the patient may be informed in general terms that there is no certainty that the offspring will suffer; that the chances are against the appearance of the disease in any individual child; but that they are also against the escape of all of several children. With the statement of these facts the decision must be left to those immediately concerned. The general welfare of the community would preclude the marriage of any member of a family in which there is hereditary disease; but the welfare of the community has seldom a definite influence on personal action. Minute as must be the effect of any individual action, it is a part of that which makes for the well-being or ill-being of the race through years we cannot number.

W. R. Gowers.

REFERENCES

1. Beevor, C. E. "On the Relation of aural Giddiness to Epileptic Seizures," *Brain*, London, 1883-84, vi. 487.—2. Broadbent, W. H. *Brit. and For. Med.-Chir. Rev.*, 1866, xxxvii.—3. Echeverina, M. G. *On Epilepsy*, New York, 1870.—4. *Idem.* "Syphilitic Epilepsy," *Journ. Ment. Sc.*, London, 1880, N.S. xxvi. 165.—5. Gowers, Sir W. R. *Epilepsy and other Chronic Convulsive Diseases*, London, 1880; 2nd ed., 1901.—6. *Idem.* *Dynamics of Life*, London, 1894.—7. *Idem.* Bowman Lecture *Trans. Ophth. Soc. U.K.*, London, 1894-95, xvi.—8. *Idem.* "Goulstonian Lectures on Epilepsy," *Brit. Med. Journ.*, 1880, i. 548.—9. Hasse, K. E. Virchow's *Handbuch*, Bd. iv. Abt. i. S. 264.—10. Herpin. *Du prognostic et du traitement de l'épilepsie*, Paris, 1852.—11. Jackson, Hughlings. *Med. Times and Gaz.*, 1868, August 5; 1872, June 6.—12. *Idem.* *Lancet*, London, 1873, i.—13. *Idem.* *Brain*, London, 1899, xxii. 534.—14. Spratling, N. P. *Epilepsy and its Treatment*, 1904, Phila. —15. Turner, W. Aldren. *Epilepsy*, 1907, London.—16. *Idem.* *Brit. Med. Journ.*, 1910, i.

W. R. G.

CHOREA

Synonyms.—*St. Vitus's Dance; Chorea Minor; Chorea Anglorum; Chorea Vulgaris; Sydenham's Chorea.*

By J. S. Risien Russell, M.D., F.R.C.P.

Chorea is a spasmodic affection of the nervous system, characterised by involuntary movements which are sudden, jerky, and irregular; by a variable degree of muscular weakness accompanied by inco-ordination of voluntary movements; and by psychical disturbances, usually of the nature of hebetude and mental weakness: to these may be added phenomena indicative of arthritis and endocarditis.

No more inappropriate name could have been chosen from an etymological point of view than that of chorea,—which was applied to the disease by Sydenham,—for χορεία means dancing, and "Chorea Sancti Viti" denoted a dancing mania prevalent in certain parts of Germany during the fourteenth and fifteenth centuries—an affection totally distinct from the disease that we have now to describe. The association of the name with the dancing mania dates from 1418, when an epidemic occurred at Strassburg, and the Magistrate sent the victims to the chapel of St. Vitus at Zabern to be calmed by religious ceremonies and the influence of the saint. As pilgrimages were made to other shrines also the epidemic became variously known as St. Anthony's dance, St. John's dance, and so on.

Sydenham's use of the name Chorea for an affection totally different from that to which it had been formerly applied, naturally led at first to a great deal of confusion; so that the use of such names as "Chorea minor," "Chorea Anglorum," and the like, were introduced to distinguish the disease now before us from the dancing mania; but as the latter outbreaks no longer occur there is now no likelihood of ambiguity. Nevertheless, even yet the name "Chorea minor" is commonly employed to distinguish the affection, as described by Sydenham, from a hysterical manifestation known as "Chorea major," which is related to the dancing epidemics of the Middle Ages.

Chorea minor, as now known to us, was first described by Sydenham in 1686, and again seven years later. Many contributions to the subject are to be found among the writings of authors between that time and the beginning of the last century; and references to their publications are made in a paper by Bernt, published in 1810; in which year another important monograph on the subject came from the pen of Bouteille in France. Since then the writings of Trousseau, Sée, Roger, and Charcot in the same country have added much to our knowledge of the subject; similar credit is due to Romberg, Steiner, and von Ziemssen in Germany, and to Weir Mitchell, Jacobi, Putnam, Osler, and others in America; but the most important contributions to the subject have been made in this country—by Bright, Todd, Wilks, Fagge, Hughlings Jackson, Gowers, Poynton, and many others.

Causation.—*Race.*—According to Weir Mitchell and others chorea is rare in negroes; and the same may be said of Indians, for although it occurs in half-breeds, Prof. Osler never traced it in full-blooded Indians.

Hereditary Influences.—Though in some instances heredity plays an important part in the etiology, the proportion of cases in which chorea itself has existed in the parent is small; an indirect neuropathic heredity, in the shape of epilepsy or insanity, being more common. It frequently happens that more than one member of the same family is affected with chorea.

Temperament.—The disease is especially apt to occur in nervous, highly strung, excitable children, while it is rare in the phlegmatic.

Social Status.—Such statistics as are at our disposal point to the occurrence of chorea in the lower much more frequently than in the upper classes; the percentage of the former, according to the Report of the Collective Investigation Committee of the British Medical Association, being 72·27.

Sex.—The statistics of different authors vary somewhat as regards the relative frequency of chorea in the two sexes; but all are agreed that it is more common in girls than in boys. Sir W. Gowers found the relative proportion to be almost three girls to one boy; Prof. Osler found that women are affected in the proportion of rather more than two to one.

Age.—Chorea is a disease of childhood and adolescence, and it is especially prone to occur in the later years of childhood. In the Collective Investigation Committee's Report, 34 per cent of the cases occurred between five and ten years of age; 43 per cent between ten and fifteen; and 16 per cent between fifteen and twenty: but it is not stated how many of these were first attacks. Prof. Osler has analysed 522 cases as regards age, and found that 33 occurred in the first hemi-decade, 228 in the second, 212 in the third, and 62 in the fourth. The disease is rare before the age of five years; though, so far as we may judge from Prof. Osler's statistics, this does not apply so much to cases in America as in this country; but even this observer agrees that the disease is rare before the fourth year.

On the other hand, but few cases occur after the age of twenty; according to Sir W. Gowers not more than 5 per cent of the total number: though a form of chorea may be met with in quite old people. This form, however, is probably quite distinct from that which occurs in younger people.

Sex plays a part in relation to the age of incidence of the disease, which falls rather earlier in girls than in boys. According to Prof. Osler's statistics the largest number in boys is in the second hemi-decade, and the largest number in girls in the third.

Climate, Season, Locality.—Chorea does not appear to be influenced by climate; but the investigations of Morris Lewis, in Philadelphia, have been largely serviceable in establishing a distinct relation to the time of year. In an analysis of 717 separate attacks of chorea, the smallest number of cases occurred in November (24, or 3·3 per cent). A rapid rise in the number was observed in December (56, or 7·8 per cent); the number remained about the same during January and February, rose to its highest in March (107, or 14 per cent), fell somewhat in April (63, or 8·7 per cent), to rise again in May (80, or 11·1 per cent); after which time there was a steady fall to the lowest point of the curve in November. Lewis accordingly concluded that weather is probably an important factor in the etiology of chorea. Putnam, however, failed to establish a seasonal variation in Boston; and in this country season appears to have no decided effect, for of 100 cases Sir W. Gowers found that 33 occurred in the first three months of the year, 25 in the second, 20 in the third, and 27 in the fourth.

Although more cases of chorea are met with in towns than in the country, Sir Isambard Owen's statistics shew that the disease is widely distributed throughout Great Britain, and is not more apt to occur in any particular parts.

Rheumatism.—That there is a close relation between chorea and rheumatic fever is now universally recognised, and some regard the former condition as a manifestation of the latter. Indeed there is abundant evidence that both diseases are microbic in origin, and that the same micro-organism is responsible for the clinical manifestations of rheumatism and chorea. Their association attracted the attention of earlier writers. Hughes, G. Sée, and H. Roger did most to establish the relationship of the two diseases; indeed, the last-named observer, writing in 1866, regarded rheumatic fever, chorea, and endocarditis as different names applied to one and the same pathological condition. Other French authors have also urged the rheumatic causation of chorea; but the Germans have not attached nearly so much importance to it, except Mayer, who attributes 80 per cent of his cases of chorea to rheumatic infection. In this country, among those who early recognised the association of the two diseases, the physicians at Guy's Hospital appear to have been especially prominent. Fagge states that, according to Bright, rheumatism was regarded as the cause of chorea as early as 1802. Babington noted the occasional association of chorea with rheumatic affections of the heart and pericardium, and was of opinion that the credit of the discovery of this association belongs to Addison.

Statistics have been collected, by various observers, bearing both upon the actual occurrence of rheumatism in the individual prior or subsequent to the attack of chorea, and upon a personal or family history of the arthritic affection. As regards the latter, Tylden found the proportion as high as 72 per cent, but the largest recorded proportion in the former category is 50 per cent. The British Medical Association Collective Investigation Committee found that rheumatism preceded the chorea in 26 per cent; in 32 per cent the rheumatism accompanied the chorea or occurred subsequently; this number rose to 46 if patients who had been the subjects of vague rheumatic pains were included. Among the statistics collected in America with regard to this point, those of Prof. Osler, based respectively on 554 and 175 cases, gave a percentage of 15·8 among the former and 18·24 among the latter, of cases in which articular rheumatism had been present prior or subsequently to the attack of chorea. Allen Starr found, as regards actual rheumatism, that 385 cases yielded a percentage of 18; and C. W. Townsend found that in the 148 cases of his collection the percentage was 21; but by far the largest percentage is that of 54, arrived at by Crandall.

In the great majority of cases in which the two diseases are associated the arthritic manifestations precede the chorea, which occurs either as the rheumatism is subsiding or after convalescence is established. According to Dr. T. T. Whipham, in less than 2 per cent of cases chorea precedes rheumatism; whilst Dr. F. E. Batten found that 11·3 per cent of chorea patients acquired rheumatism within three years, and 20 per cent within six years.

The percentage of cases preceded by rheumatism varies with the age of the patient; thus, Sir W. Gowers met with one instance only among his cases in which there was a history of previous rheumatism in children under nine years of age; between the ages of ten and fifteen, on the other hand, there was such a history in more than one-fourth of the cases. It is obvious from this that, if attention be not paid to the ages of the patients from whom the statistics are compiled, an incorrect estimate may be formed of the frequency of the association of the two diseases. It must be remembered, however, that the manifestations of rheumatism in a child may be so slight as readily to escape detection, and that this may possibly account in part for the small number of cases in which a history of previous rheumatism can be obtained during the first decade of life. An analysis of 327 cases by Dr. Morley Fletcher shews that in 26 per cent of cases chorea is preceded by rheumatism, his figures agreeing closely with those of Sir S. Mackenzie (29 per cent). Sée and Hughes both make the percentage much higher, whilst according to Sturges it is only 20 per cent.

Inseparable from this inquiry is the unquestionable affinity which exists between chorea and cardiac affections. Bright regarded inflammation of the pericardium as the link between rheumatism and chorea, but his views were based on an erroneous conception of the pathology of the latter disease.

Endocardial changes are common in chorea; so much so, that Fagge found such changes in 17 out of 18 necropsies of chorea at Guy's Hospital; in 5 of them death was accidental and due to some intercurrent affection or complication, not to the severity of the disease. The presence of such valvular changes in the heart in a case of chorea raises the question whether the endocarditis be of choreic origin, or dependent on pre-existing rheumatism. It may be impossible to answer such a question when an attack of chorea has preceded that in which the patient is seen; but, otherwise, even in the absence of a history of previous arthritic rheumatism, it may be possible to form a comparatively accurate estimate on the subject. In 40 cases of chorea with organic heart disease, Sir W. Gowers had strong grounds for attributing the cardiac affection to previous rheumatism in 18—a proportion which he regards as below the actual number as he only admitted conclusive evidence.

Those observers who disbelieve the rheumatic origin of chorea insist that rheumatism is not an invariable precursor or concomitant of chorea; and there are those who, like Féré, regard the rheumatic infection only as a contingent cause, comparable to the action of other infectious diseases. The observations of Drs. Poynton and Paine, however, appear to outweigh all such objections, for they have succeeded in isolating and cultivating from the cerebrospinal fluid of patients suffering from chorea a diplococcus identical with the *Diplococcus rheumaticus.* Inoculation of the diplococcus in the rabbit has produced twitching movements, endocarditis, myocarditis, pericarditis, cardiac dilatation, arthritis, pleurisy, pneumonia, subcutaneous nodules, and teno-synovitis; whilst with a culture of the same organism, Dr. Vernon Shaw produced cardiac lesions and multiple arthritis in monkeys. Drs. Poynton and Paine have found the diplococcus in the pia mater and brain, both in choreic patients and in the rabbit. Drs. Poynton and Gordon Holmes have had a like experience in rheumatic chorea and in chorea gravidarum, and succeeded in isolating the diplococcus from the blood of these patients. (For the bacteriology of chorea and for a critical consideration of the *Micrococcus rheumaticus*, *vide* Vol. II. Part I. p. 612.)

Infective Diseases.—Of the various infective diseases which have been known to precede chorea, scarlet fever, which may be attended with arthritis, is the most important. So rarely are any of the infective diseases associated with chorea, however, that most observers have been led to regard them rather as indirect exciting causes of the attack. Out of 533 cases of scarlet fever, collected by Carslaw, chorea was noted in 3 only; and though Prof. Osler obtained a history of scarlet fever in 141 of his 554 cases of chorea, in not a single instance did the latter disease immediately follow the former. Ross, however, held that chorea follows scarlet fever frequently, and thought that the association might probably be explained by the fact that rheumatism frequently follows scarlet fever.

Measles, variola, diphtheria, enteric fever, cholera, and pneumonia have all been known to be followed by chorea, and, according to Sturges, whooping-cough frequently precedes it. Another class of infective diseases, in the course of which chorea may arise are pyaemia (with or without suppurative polyarthritis) and gonorrhoea. Straton has insisted on the possibility of micro-organisms gaining entrance through erosions in the mucous membrane of the naso-pharynx in children, and thus causing the affection. Chorea has also appeared in the course of secondary syphilis, which disease Féré considers has been justly regarded as a causative factor in chorea. Harrison Mettler justifies a similar view by this association, and the further observation that the chorea has been cured by antisyphilitic treatment.

Instances of the association of chorea and malaria are on record, but there is no proof that the former affection is more common where the latter is prevalent than elsewhere (see "Electric Chorea," p. 554). It is interesting to note that with the onset of fever the spasms cease; a fact observed since the time of Hippocrates and frequently seen in connexion with epilepsy. When one of the acute specific diseases occurs in the course of chorea the irregular movements usually cease.

Maragliano attributes chorea to the action on the nervous system of the toxins of various micro-organisms, notably of staphylococci, while Siegert considers that subacute bacterial invasions in acute articular rheumatism, angina lacunaris, and gonorrhoea are the most important etiological factors.

Intoxications.—Closely associated with the last inquiry is that of the part played by poisons introduced into the system. The most convincing case of the kind on record is one, by Demme, in which chorea appeared after the use of iodoform in a fistula connected with caries of the cervical vertebrae; it ceased when the iodoform was suspended, and returned on resumption of the treatment.

Impoverished States of the System.—Any weakening influence seems to favour the occurrence of chorea; but most has been made of that of anaemia, as advocated by Bouchut, Trousseau, and others. The importance of an impoverished blood state as a causative factor has been insisted on more especially by Rachford. On the other hand, Prof. Osler dissents entirely from this view, though he admits that he has seen several cases in which chorea occurred in chlorotic girls at the time of puberty. Possibly the supposed etiological relationship of the two conditions is rather to be explained by the common occurrence of both during adolescence; nevertheless, Litten alludes to 2 cases in which chorea appeared in the course of pernicious anaemia, and in which recent endocarditis was found after death. So, too, Roeser had a case of fatal epistaxis in which similar endocarditis was found at the necropsy, and in which chorea occurred during life.

Pregnancy.—The relationship of pregnancy to chorea is both important and interesting. Chorea is usually manifested during first pregnancies, and in most cases there is no other cause than the pregnancy to which it can be attributed. In other cases, however, there is a history of a previous attack of chorea, perhaps in childhood; or there may be a distant history of rheumatism; or the latter disease may immediately precede the chorea. Those who have suffered from chorea in a first pregnancy may suffer also in a second, and more rarely in a third; Féré, indeed, refers to an instance in which a woman had an attack with each of five pregnancies. A patient may escape during her first pregnancy and have chorea in a subsequent pregnancy; in 9 out of 29 cases recorded by Drs. French and Hicks the first pregnancy was free, and yet chorea occurred during a later pregnancy; in one instance chorea first appeared in a fourth pregnancy. The disease scarcely ever occurs for the first time after the age of twenty-five. From his own cases, and some selected from those collected by R. Barnes, Sir W. Gowers found that of 28 cases 8 occurred at the age of twenty; 3 at the age of seventeen, eighteen, and nineteen respectively; 2 at twenty-one and twenty-two; 6 at twenty-three, and 1 at twenty-four. In Drs. French and Hicks' cases there were 3 aged thirty years.

The onset of the chorea is most commonly between the first and the third months of pregnancy, and appears specially prone to occur at the third month. It may, however, appear at any period of pregnancy, though very rarely in the ninth month. In a few rare cases the chorea has not appeared until after parturition; it has been known to follow an abortion. According to Drs. French and Hicks, when chorea occurs in successive pregnancies it is prone to occur at the same month on each occasion; this, however, is not universally accepted.

An interesting question, which arises out of the association of pregnancy and chorea, is how much has emotion to do with the matter? A woman who discovers for the first time that she is pregnant is more likely to be the subject of emotional excitement than one who has been in this condition before. This view finds support in Sir S. Wilks's observation that a large proportion of the cases occur in unmarried girls.

Nevertheless, Drs. Poynton and Gordon Holmes are of opinion that this form of chorea is also of rheumatic origin, for they have obtained the *Diplococcus rheumaticus* from patients suffering from this disease.

Emotion.—The form of emotion to which chorea has been most frequently attributed is fright; and as many mothers know this to be a recognised cause of St. Vitus's dance, probably the number of instances in which a history of fright has been obtained must be discounted. In a considerable proportion of such cases several days have elapsed between the fright and the chorea; but there are well-authenticated cases on record in which the disease followed so immediately on the emotion as to leave no reasonable room for doubting the relationship of cause and effect. One of the most striking instances of this kind is a case, recorded by Romberg, in which a little girl was attacked the same day after a severe fright caused by a dog jumping and barking at her. Von Ziemssen refers to a case in a boy in whom the disease became severe within a few hours after a fright; Trousseau to a case of a girl who was terrified into a nervous fit, and immediately became affected with chorea. It is rare for chorea thus to follow a fright without any interval; but among other recorded instances of the kind are 2 cases quoted by Sir W. Gowers, both in boys; in one it was the result of an unexpected pistol shot close to the ear, the other boy was caught in an apple tree and fell in his hasty descent. On the other hand, it is rare for the interval to exceed a week, and when several weeks have intervened the efficiency of the fright becomes a matter of doubt.

Other mental emotions may likewise be responsible in some cases; notably grief, worry, or various forms of excitement: but none of these is nearly so frequently operative as a fright. Mental strain, such as high pressure at school, was also urged as a cause of chorea by Sturges.

Hysteria.—As chorea is so much more common in girls than in young men, and as many cases occur in adolescents, it is scarcely surprising that the two conditions should sometimes be associated in the same individual. Duchateau has collected 34 cases of the kind. On the other hand, a few cases have been recorded in which hysteria, the great simulator, has reproduced the clinical picture of chorea with wonderful exactness.

Imitation.—So impressed were some of the older physicians with the effect of imitation as a cause of chorea, that, according to Fagge, Addison and physicians who succeeded him at Guy's Hospital did not allow more than one or two cases of chorea to be warded with other children. It is, however, improbable that true chorea is ever produced in this way; and in none of Prof. Osler's 554 cases did this factor appear to have been operative. It is the hysterical variety which often arises in this way, and most of the epidemics of chorea have been made up of hysterical cases. Such epidemics have been reported, notably by Bricheteau, Steiner, Weir Mitchell, and Wichmann; but Steiner regarded the epidemic at Prague as the result of atmospheric influences, and in no way attributable to imitation.

Errors of Refraction.—Closely associated with overwork at school is the part played in the production of chorea by eye-strain consequent on anomalies of refraction—a causal influence more especially insisted on by Stevens. De Schweinitz finds that there is hypermetropia, or hypermetropic astigmatism, in about 77 per cent of children affected with chorea; this proportion, however, corresponds exactly with what is usually found in childhood, for hypermetropia is present in 76 per cent of the eyes of children at the elementary schools; he concludes, therefore, that the evidence that hypermetropia is a fundamental cause of chorea is not sufficient. He, however, admits that cases of chorea are benefited by the correction of refractive errors, and he supposes that in a person predisposed to chorea eye-strain may foster attacks, or even provoke them.

Reflex Irritation.—Intestinal worms, gastric disturbances, dentition, and the like have been regarded as causes by many, whilst by others their influence has been denied. They are so rarely met with that it is doubtful if they play any part in the production of the disease; at the same time they certainly may act as proximate causes in persons predisposed to chorea.

Injury.—Blows, falls, surgical lesions, the successful and unsuccessful extraction of a tooth, and such shocks, have all been assigned as causes of chorea; but the concomitant emotion is probably the potent causative factor in such cases.

Morbid Anatomy.—Macroscopical examinations of the central nervous system in uncomplicated cases of chorea have usually been negative, as have many microscopical examinations in the past; but many of these have not been conducted with the necessary thoroughness. From a study of 79 necropsies Raymond concludes that the condition most commonly met with is hyperaemia; the next in frequency of occurrence being softening consequent on embolic plugging of cerebral vessels, and then chronic encephalitis. In a similar analysis Dana found only 39 necropsies reported with sufficient fulness to be of value. In 19 of these, careful microscopical examinations were made; the lesions found in 16 were pronounced hyperaemia, punctiform haemorrhages, perivascular exudation, foci of softening, and, in some instances, emboli; all of which changes were most marked in the basal ganglia. Cramer and Többen found distension of the vessels of the brain, perivascular haemorrhages, and increase of glial nuclei. Orzechowsky determined foci of degeneration in the motor region of the brain, but does not consider them characteristic of chorea. He regards them as due to septicaemia, which, according to him, is the cause of death in chorea. Delcourt and Sand are of opinion that foci of exudative inflammation are much more numerous in the basal ganglia than in the cortex, and found the vessels surrounded by lacunae, zones of necrosis, and abundant exudation. Gross lesions are met with exceptionally, as, for instance, softening consequent on plugging of a large cerebral artery with consequent hemiplegia during life, and a large haemorrhage which may have proved fatal.

Discrepant results have been obtained on examination of the motor neurons of the cerebral cortex. Charlewood Turner, in 5 cases, found some of the large pyramidal cells swollen, and their protoplasm cloudy and dense-looking. Berkley, in a case of Osler's which he examined, found no special changes in these motor neurons. Dana, on the other hand, found the pyramidal cells in a state of hyaline degeneration; but his case was complicated by chronic leptomeningitis. Changes in the neurons in other parts of the brain have also been described; thus Meynert found swelling and hyaline degeneration in the cells of the central ganglia, and Elischer similar changes in the claustrum and island of Reil. Peculiar round hyaline bodies, concentrically laminated and strongly refractile, to which the name "chorea-corpuscles" has been applied, have been found in the perivascular sheaths of the vessels of the corpora striata and internal capsule, and have been described by Elischer and Jakowenks; but Wollenberg, who carried out control investigations in forty-six brains of persons not affected with chorea, besides the brains of six persons so affected, concluded that, though these bodies are present in some cases of chorea, they have no special significance, as they are also to be found in persons who have never had this disease.

The changes which have been found in the pons, medulla, and spinal cord are no more characteristic than those already described as sometimes met with in the brain; they consist of hyperaemia, punctiform haemorrhages, and perivascular round-celled exudation. The supposed degenerative changes described by Elischer in the cells of the neurons of the spinal cord are in no way characteristic; and Berkley, in the case which he examined, failed to detect any changes in these cells, or in those of various nuclei in the medulla. Haemorrhage into the central canal of the spinal cord has been recorded (Steiner).

Meningitis, either cerebral or spinal, has been met with as a complication in exceptional cases.

The changes described in the peripheral nerves by Elischer—hyaline swelling of the axis-cylinders, with increase of interstitial tissue—are of questionable significance; but Frey has found changes characteristic of multiple neuritis.

Several observers have attempted to isolate micro-organisms in chorea, but the investigations of Drs. Poynton, Paine, and Gordon Holmes with the *Diplococcus rheumaticus* are the most complete. Many other micro-organisms have been detected, by different observers, in the blood, cerebrospinal fluid, brain, and heart, notably staphylococci and streptococci (*vide* Vol. II. Part I. p. 612).

Pathology.—Does chorea occur in a number of circumstances, owning no indispensable cause? From the widely different etiological factors it might seem that such is the case; but on closer scrutiny we find that these diverse factors may be grouped in three classes. The first may be unstable conditions of the nervous system, of congenital origin, or induced by developmental changes; the second, similarly unstable conditions of the nervous system induced by accidental causes, such as shock or great emotion, acute illness, or overwork, mental or physical; and in the third class we may place the immediate causes of the particular attack. We have seen that the phenomena of chorea may be due to some toxic agent; or, on the other hand, they may be evoked in a reflex manner through some source of peripheral irritation, such as eye-strain. Yet, however effective a contingent cause, such reflex influences cannot possibly generate a disease of which the nervous phenomena form but a part. Far more potent factors are defective nutrition, bad hygiene, and the like, which favour the action of microbic infection. There is much in favour of the belief that chorea is an infective disease, and its kinship to rheumatism strengthens this view. The two diseases are now generally regarded as microbic in origin, and according to some the *Diplococcus rheumaticus* is the infective agent in both, but others consider them the outcome of mixed infections rather than the result of a specific virus.

The old view that chorea was due to cerebral emboli necessitated the assumption that endocarditis always precedes the nervous manifestations of chorea, whereas very few instances of this sequence have been met with; moreover, the nervous manifestations have resulted in death without any endocarditis having been afterwards detected.

That such remote antecedents as temperament, age, sex, season, and debilitating influences affecting the nervous system, may favour the action of an infective agent, is in keeping with what we know of infective processes generally; but it is less easy to reconcile with the infective doctrine those cases in which chorea has followed so closely on a fright as to make it difficult to believe that we are not dealing with cause and effect. Widely different views have been expressed as to the part of the nervous system which is probably deranged and permits of the manifestations of chorea, so that although the motor cortex has many advocates, numerous other seats have been suggested. Sir W. Gowers is of opinion that as the source of nerve impulses is referred beyond the cell to the dendrites, chorea may be due to affection of the dendrites, as irregularity both in time and extent of dendritic action would account for most of the motor manifestations of chorea. Delcourt and Sand favour derangement of the basal ganglia; Charcot and Raymond special fibres in the internal capsule; Kahler and Pick assume irritation of the cortico-spinal tracts by lesions at the base; whilst von Monakow believes that the irritation is transmitted to the motor centres of the cortex by way of the sensory tracts which pass to them from the corpus striatum. Yet another view, which finds support in Bonhoeffer's statement that in symptomatic chorea there are often foci near the superior peduncle of the cerebellum and red nucleus, is that the manifestations of chorea are due to alteration of the nerve impulses which pass from the cerebellum to the motor cortex. The observations of von Halban and Infeld tend to confirm this view, which has been accepted by a good many observers; in addition to which Ecomono and Karplus observed choreiform movements in cats in whom the red nucleus had been injured in the operation of dividing the pes pedunculi.

Symptoms.—In chorea a disordered condition of the motor centres is evidenced by the involuntary spontaneous movements, and by the defective power and precision of voluntary movement. Each of these defects is seen in different degrees of intensity and in various combinations in different cases. The first indication often is that the patient is always fidgeting, though the movements are so similar to those seen in nervous children without chorea that they may fail to suggest any other interpretation. These movements usually begin in the arms or face, and exceptionally in the legs. Thus, various grimaces and contortions are seen; at one moment the eyebrows are raised, at the next an eye is closed and then opened, the upper lip is drawn up on one side and then falls again, the angle of the mouth is drawn to one side, or the mouth is first opened then closed—all of which movements are carried out in a purposeless manner. Or it may be that the head is suddenly twisted to one side, or jerked back; or the patient shrugs one shoulder; or the whole arm is jerked forward from this joint. So, too, when sitting down with the hands on the lap, the hand may be resting naturally when it is suddenly turned palm upwards, and as abruptly back to its original position; or the fingers are irregularly extended and flexed without purpose. Implication of the trunk muscles causes writhing movements, or swaying from side to side, as the patient sits or stands. When the legs are affected the momentary contractions of their muscles cause jerking of the trunk first to one side and then to the other; or in walking the leg is rotated, it may be first in and then out: or one leg is thrown across the other, as if about to become entangled with its fellow.

The spasmodic movements are sudden, and each is of very short duration; they may be simple or complex, and are always irregular, both as regards the degree of resulting movement and as regards the intervals at which they are repeated. At first the patient may have to be closely watched before the movements are noticed, but later they require no such close scrutiny for their detection; so, too, the intervals between the spasms are much longer at first, and become shorter as the disease progresses, until there may be scarcely a single moment during the patient's waking hours when some part of the body is not in movement. Mental excitement and any attempt to perform a voluntary movement aggravate the spasms; whilst mental and physical quietude diminishes, and sleep, whether natural or artificially induced, arrests them; except in rare instances of very severe spasms, in the majority of which cases sleep is prevented. According to Jaccoud, in less severe cases the spasms sometimes remain in abeyance for a little time after the patient wakes from sleep. Weir Mitchell and Rhein find that in some cases there may be severe choreiform movements during repose which entirely disappear on some muscular act being performed. Dr. J. W. Russell has confirmed these observations, and has, moreover, pointed out that the power of inhibiting the choreiform movements can be well demonstrated by making the child attempt to write; then it will be seen that the patient can, for a time, inhibit choreic movements of the most pronounced kind, and write, it may be, even a word composed of six or seven letters with perfect steadiness. Like Weir Mitchell and Rhein, Russell divides cases of chorea into certain groups according to the character of the handwriting:—(i.) the writing gives evidence of power of control over the movements, inco-ordination being probably present in many of the cases; (ii.) the power of control is practically complete and there is no inco-ordination; (iii.) choreic movements are either absent or very slight, and yet much inco-ordination is revealed by the handwriting; (iv.) control over choreic movements is perfect, but the handwriting betrays great inco-ordination; and (v.) choreic movements are moderate, but writing impossible from mental defect.

According to von Ziemssen, all the movements are not involuntary, some being voluntary and quickly added for the purpose of concealing an immediately preceding involuntary movement.

We have seen that all degrees of severity of the muscular contractions are met with; and not only may the most grotesque grimaces be constantly occurring, but the ocular muscles may be involved, the eyes turning concomitantly with the head and preserving their parallelism or resulting in squint when the degrees of spasm in the muscles of the two eyes are unequal. The globes return to their normal position, or are jerked into some other abnormal position.

In a case recorded by Tuckwell, so powerful was the spasm of the muscles which raise the lower jaw that several teeth were broken. The head may be banged against adjacent objects, and the limbs greatly bruised as they are violently thrown about. The arms are always affected in greater degree than the legs; the jerking movements may be so severe as to make it quite impossible for the patient to effect any purposive movement with the arms; thus, in attempting to raise a glass or cup to the lips the arm is thrown in various directions before the vessel reaches its destination, and most of its contents are probably spilled before it is grasped by the teeth and the remains of its contents gulped down in the most hasty manner possible.

When the trunk muscles are involved their spontaneous contraction may be so powerful as to throw the patient out of bed. Similarly voluntary movements of the lower limbs may be so interfered with by the muscular contractions that walking may be made quite impossible. In cases of moderate intensity the spontaneous movements may be limited to one side of the body; but Dr. Hughlings Jackson has shewn that this is true only of the limbs, the muscles of the face and trunk being always bilaterally affected. In these cases of hemi-chorea Dr. Jackson is of opinion that the right side is the more frequently affected; according to Jaccoud, it is the left, and the figures quoted by Dr. Pye-Smith and Sir W. Gowers both shew a small balance in favour of the left side. The movements may remain limited to one side or part of one side throughout the attack; or the opposite side may be attacked, with or without cessation of the spasm on the side first affected; so, too, movements involving the arm and leg on one side may spread to the opposite leg without implication of the corresponding arm.

But, as was stated at the outset, not only may voluntary movement be interfered with by these spontaneous movements, but two other factors may be in operation : an inco-ordination or want of precision in the performance of voluntary movements, or an actual want of power due to muscular weakness. In the first of these cases, instead of some jerky spontaneous movement being the first to attract attention, this failure to co-ordinate movement may be noticed first : the motor centres do not obey the dictates of the will, so that when the patient tries to relax some set of muscles, for the due completion, it may be, of some muscular act, there is a marked delay, which, of course, seriously interferes with its efficacy. Or the motor centres may bring about relaxation of a group of muscles during the performance of some voluntary act without having received any command to do so, and oftentimes with disastrous results, as objects in the hand may be suddenly dropped and broken. The inco-ordination of movement may be shewn in a different way, the patient overshooting the mark in the attempt to lay hold of or pick up some object.

It will be readily understood that the spontaneous movements and the inco-ordination of movement may in any given case be so combined as to make it difficult to distinguish the operative factor ; for, of course, a voluntary act may well be interrupted, not in consequence of the sudden unwilled relaxation of a group of muscles, but of the unexpected intrusion of some spontaneous movement. Voluntary movements are irregular, and are performed in a sudden and spasmodic manner ; partly on account of the involuntary movements, and partly to dodge them. If the patient be told to protrude the tongue, the act may be delayed in accomplishment, but the organ is then shot out of the mouth and as suddenly withdrawn, perhaps just in time to escape the snap of the closing jaws. In severe attacks deglutition is much interfered with. Speech is often affected, words are spoken rapidly and in an explosive sort of way, the sentence being frequently interrupted by a hasty inspiration which may clip off the last part of the word which immediately precedes it ; or there may be an inspiration of this kind after each word that is uttered ; or a word can only be uttered spasmodically in parts. Owing to the spasmodic movements of the tongue and muscles of articulation, speech may indeed become quite unintelligible, or the patient may be deterred from making the attempt. Von Ziemssen, on laryngoscopic examination, has observed irregular movements of the vocal cords ; and that the tension of the vocal cords is defective is evidenced by the fact that a note cannot be sustained for any length of time ; moreover, the voice is low pitched and monotonous.

The movements of the thorax in respiration are also made disorderly, chiefly owing to implication of the diaphragm. Inspiratory efforts do not occur in any regular order, either as regards time or depth of movement ; thus irregular pauses intervene, and one inspiration is deep, perhaps excessively so, whilst another is shallow or abortive. The inco-ordination of respiratory movements sometimes shews itself in an alternation between abdominal and thoracic respiration. Romberg refers to 2 cases in which a whistling or snapping sound accompanied inspiration; and in one of them spasm of the intercostal muscles caused sudden drawing in of the thorax. The same observer speaks of a case of troublesome choreic hiccup. Apart from any independent cardiac affection, the heart's rhythm may be made irregular by the disorderly respiratory movements.

A third factor which may interfere with voluntary movement is muscular weakness, a feature of the disease which was clearly recognised by the earlier writers on the subject. As a rule, this loss of power is most marked in cases in which there is little if any spasm, when it is commonly of hemiplegic distribution. Though the weakness amounts to definite paresis there is never anything approaching complete loss of power. Todd fully described this type of the disease, to which C. West applied the term "chorea mollis," and for which Sir W. Gowers has proposed the name "paralytic chorea." This element of muscular weakness probably accounts for the slovenly way in which some choreic patients shuffle their feet along the ground in walking. In 28 of Prof. Osler's cases, in which muscular enfeeblement was noted, it was monoplegic in 16, the arm being affected in each instance; paraplegic in 6; and hemiplegic in 4: in 1 case all four extremities were affected, and in 1 both arms without the legs.

The weakness may come on gradually during the course of an attack of chorea with spasms; or it may come on in a similar manner or quite suddenly before any spasms have been detected; in a third group of cases the paresis is noted after the spasms have ceased. When spontaneous movements and paresis both exist they usually disappear together; but in some instances the paresis persists, it may be even in considerable degree, for a long time after the cessation of the spasmodic movements. Prof. Osler speaks of a case in which wrist-drop persisted for two years after an attack of chorea of the right arm, with which it came on, and Dr. Judson Bury has recorded a still more striking case in which paralysis of the dorsi-flexors of the ankle, with consequent foot-drop, in a case of chorea was still in evidence four years after the onset of the paralysis.

Electrical Reactions.—These have been studied more especially by Benedikt, Rosenthal, Schmitt, and Sir W. Gowers, all of whom are agreed that in some cases electrical irritability may be increased, both in the nerves and muscles; and to both galvanism and faradism. So, too, an altered qualitative mode of response to galvanism has been described, in which the anodal closure has resulted in contraction of the muscles with as weak a current as the cathodal closure, instead of the response being more readily obtained with the latter than the former, as obtains normally. Thus KCC = ACC, instead of KCC > ACC.

Sensory Symptoms.—Headache is common; it may precede the spontaneous movements, or appear later; and is either more or less continuous or is paroxysmal. Sir T. Watson met with some cases in which the pain was limited to the side of the head opposite to the affected limbs.

Pain in the nerves and muscles of the affected limbs is rare, and, of the exceptional instances in which they have been described, most of the cases have been hemi-chorea, and have been designated "painful choreas" by Weir Mitchell. Instances have also been recorded in which pain has occurred in parts subsequently involved in the spontaneous movements. The nerve-trunks are said in some cases to be tender on pressure, and tender points have been described along the spine at the points of emergence of the spinal nerves from deeper structures. Cartier, Triboulet, and Marie have especially insisted on these, and it has been said that they are limited to the affected side in hemi-chorea; however, such phenomena, though carefully searched for by Prof. Osler in a large number of cases, were very rarely met with; and there can be no question that in the large majority of instances chorea is an affection unattended by pain. Trousseau insisted on the occurrence of tingling and formication in the affected parts, and others have described similar paraesthesias and numbness. The latter symptom Fagge found not uncommonly noted in cases at Guy's Hospital.

Diametrically opposite opinions have been, and still are, held with regard to the sensibility in chorea. Most observers are of opinion that if there be any impairment of sensibility it is probably an hysterical manifestation; but Féré asserts that blunting of sensibility is common, and goes so far as to say that this has been found in almost all cases in which sensibility has been carefully studied, especially in parts where the spasms are most pronounced. Sir W. Broadbent also considered that sensibility is frequently diminished, and Dr. Purves Stewart detected it in 10 out of 41 cases.

Reflexes.—Discrepant statements are made by different authors on this subject; some consider that the reflexes are usually normal, others that they are usually exaggerated; the truth being that in many cases they are normal, in others they are exaggerated, whilst in yet another group they are diminished; a condition especially likely to be met with in the paralytic form of chorea, in which they may even be abolished. In 50 cases of chorea, examined by Sinkler with the object of ascertaining the state of the reflexes, the knee-jerks were found normal in 26, diminished in 15, and absent in 9. Oddo's figures shew that of 147 cases examined, the knee-jerks were normal in 13 per cent, increased on one or both sides in 25 per cent, and similarly diminished in 62 per cent of the cases. Dr. W. Gordon has insisted on the importance of a peculiar way in which the knee-jerk is altered so that the limb is suspended in mid-air too long after the blow to the patellar tendon, owing to prolonged contraction of the quadriceps muscle. It is probable that the explanation of this, as suggested by Dr. Judson Bury, is to be found in Oddo's observation that a choreic movement can often be excited by percussion of a muscle or tendon. The superficial reflexes are said to be commonly diminished, and the plantar reflex has occasionally been of the extensor type, in the absence of organic hemiplegia the outcome of cerebral embolism.

The sphincters are not affected, though in severe cases the evacuations may be expelled involuntarily; and a similar result may be met with in cases in which there is much mental hebetude present.

Trophic Disturbances.—There is no spontaneous tendency to bed-sores; but the incessant rubbing of certain parts against the bed in severe cases and self-inflicted cutaneous abrasions may lead to their formation. Such sores are especially apt to form in advanced stages of severe cases, when general nutrition is much lowered, and when, as often is the case, mental hebetude is so great that the evacuations are passed under the patient.

Mental Condition.—The psychical disturbances in chorea have been studied especially by Marcé, Buccelli, and Burr. Though bright and intelligent at the outset, there are few patients in whom there is not some mental affection, though fortunately in the majority it is slight. The alteration may be in the form of diminution of attention, mental dulness, and loss of memory; not only is the child unable to learn lessons, but it no longer cares about story-books, and the face wears a dull heavy look, singularly wanting in variety of expression. Or there may be marked perversion of character; the child becomes irritable and whimsical, is liable to emotional attacks, and does all sorts of odd things. Then again, there may be hallucinations (Axenfeld and Huchard), most commonly of sight, though sometimes of one of the other special senses; or it may be of common sensibility, or in rare circumstances even of the genital sense. Such hallucinations may be originated in dreams and perpetuated during the waking hours. Buccelli is of opinion that the psychical changes are the direct outcome of the motor disorders, and that perversion of psycho-motor functions exercises a harmful effect on all mental processes. The degree of mental change, however, bears no necessary relation to the severity of the other symptoms; and the symptoms may progress to dementia, melancholia, perhaps with suicidal intent, or to mania in which the patient jabbers incoherently, shouts, and sings. It is to this class of cases that the name "chorea insaniens" has been applied; in many such patients sent to asylums the physical condition is entirely overlooked. The delirium is usually recovered from in acute chorea; but where there is any hereditary taint of insanity it may be the starting-point of progressive and permanent mental deterioration. Burr has insisted that there are no mental symptoms pathognomonic of chorea, and that there is no such disease as chorea insaniens.

Convulsions are rare in the course of chorea, and epileptiform convulsions of unilateral distribution still more rare; nevertheless, originating in this way, convulsive attacks have been known to persist, and to constitute ordinary attacks of epilepsy. Certain peculiarities of the convulsions have been noted in some cases—such as an admixture of choreiform spasms in the attacks, or an associated condition of cataleptic rigidity.

Ocular Phenomena.—In addition to the transitory squints and the like, which result from irregular spasms of the eye muscles, the following ocular phenomena also may be met with. The pupils are frequently dilated, and this has been observed to be more marked on the side on which the spasmodic movements have been most severe. According to Dr. Langmead, the following alterations of the pupils are comparatively common in chorea—hippus, peculiar movements on accommodation, contraction, inequality, and eccentric pupils. No special significance is to be attached to inequality in children, but the only other general affections in which the other abnormalities appear to be met with are articular and cardiac rheumatism. The optic discs are usually normal; but neuritis is met with exceptionally, and as hypermetropic errors of refraction have been also present in many such cases, it has been supposed that the hypermetropia may be responsible for the changes in the discs; but that it is not the only factor in the production of the change is proved by the disappearance of the neuritis without correction of the hypermetropia. This condition of the optic nerves lends support to the view that chorea is of infective origin, for it is more probable that the neuritis depends on some blood state than that it is secondary to an intracranial lesion.

Circulatory System.—The pulse is frequently rapid, especially where the spontaneous movements are severe; in some cases it is irregular, according to some observers, owing to a veritable chorea of the heart; whilst others regard it as depending on the disturbance of the respiratory movements of the thoracic cage. Sir W. Gowers has observed several instances in which posture had less effect than in health, there being little difference, if any, between the pulse-rate in the upright and recumbent postures.

The most important defect in the circulatory system, however, is in the condition of the heart itself. Apart from rapidity and, possibly, irregularity of action, murmurs may be heard over the precordial area, the significance of which has been much discussed. Three explanations have been offered for these—(*a*) that they are haemic, depending on associated anaemia; (*b*) that they are the result of regurgitation at the mitral orifice consequent on irregular contraction of the papillary muscles not closing the mitral flaps effectively; (*c*) that they are due to organic valvular lesions. The second of these explanations is one about which little need be said, in that we have no means of proving or disproving it. With regard to the other two there can be no question that both are operative in different cases, or it may be even in the same one. Whilst it may be difficult in some cases to be certain which of these two factors is responsible for the murmurs, as a rule the following considerations will lead to a correct interpretation. In the first place, doubt can only arise where the murmur in question is systolic in time; a presystolic or diastolic murmur being unequivocal evidence of organic disease. I cannot concur with those who hold that diastolic and even presystolic murmurs may be haemic in origin. In the next place, it is comparatively rare for organic disease to be found at the aortic orifice in chorea, the mitral valves, as we have already seen, being affected in the large majority of cases. In anaemia the most common murmurs have their seat of maximum intensity at the base; or if, as sometimes happens, the point of maximum intensity is in the fourth intercostal space close to the left border of the sternum, it is heard up to, but not beyond, the apex of the heart; whereas in the organic cases by far the most common murmur is a systolic one having its seat of maximum intensity at the apex and conducted into the axilla, and it may be round to the angle of the left scapula. The presence of a murmur at the apex conducted into the axilla may, however, depend on dilatation of the left ventricle in anaemia, without the presence of organic valvular disease; so that it is necessary to take other points into consideration before arriving at a conclusion, such, for instance, as other evidences of anaemia in the patient, the presence or absence of a haemic murmur at the base having its point of maximum intensity in the left second intercostal space, the presence or absence of a venous hum in the neck, and so on. But as organic valvular defect and anaemia may coexist, some cases may have to remain unsettled until the anaemia has been removed. Even this does not entirely settle the question, however, for, as Fagge has insisted, even murmurs observed in rheumatism may similarly pass away; Kirkes, Wilks, Baxter, and Frank have all recorded cases in which no murmur was present during life, and yet vegetations were found on the valves after death.

Apart from rapidity and possible irregularity of action, there may be definite organic disease of the heart. According to Sir W. Gowers, in 9 out of every 10 fatal cases of chorea, disease of the valves of the heart is found. Sturges collected 80 cases from four of the large London hospitals, in only 5 of which were the heart and pericardium free from disease. Of 34 cases collected by Raymond, in addition to the cases reported by Sturges, the condition of the heart is only given in 19, in all of which there was endocarditis. Prof. Osler has since collected from more recent records 73 cases with endocarditis in 62 of them.

Murmurs indicating the existence of organic disease of the heart may be present before the attack of chorea, and may then be due to a previous attack of this disease or of acute rheumatism. On the other hand, primary attacks of chorea may be met with in which there had not been any antecedent rheumatism, and in which no murmur is present. In some such cases a murmur may appear during the attack, and may persist after the patient is convalescent; in other instances, as the patient recovers from the chorea, the murmur may disappear. In another class of cases no murmur is heard throughout the attack, but presents itself at some subsequent period.

The most common murmur is a systolic one produced at the mitral orifice; but sometimes, where the murmur existed before the onset of the chorea, a presystolic murmur is heard alone, or in combination with one systolic in time. Murmurs indicating the existence of aortic disease are much more rare, as may be judged from the fact that the Collective Investigation Committee, previously referred to, found 116 cases of mitral disease as opposed to only 6 aortic cases. So, too, Sir W. Gowers states that out of 250 cases of chorea he only met with 2 cases of aortic regurgitation; and Prof. Osler, out of 72 in which there was evidence of organic disease of the heart two years and more after chorea, found 4 cases only in which the aortic valves were affected.

With regard to the association of pericarditis with chorea we have already seen that Bright, and some others of the older physicians, especially at Guy's Hospital, recognised this, and that Bright founded a hypothesis of chorea on this association. The pericarditis is usually associated with endocarditis, though it sometimes occurs alone. Thus, of 21 cases of chorea occurring in acute rheumatism Sibson found that 15 had pericarditis, 14 endocarditis, 3 doubtful endocarditis; 6 had no pericarditis, and 3 no endocarditis. So, too, of 73 necropsies in chorea, collected by Prof. Osler, there were 19 cases of pericarditis, in only 2 of which endocarditis was absent.

Cutaneous Affections.—The more important of these serve as another link in the chain of evidence in support of the close affinity between chorea and rheumatism. Erythema nodosum, arthritic purpura, purpuric urticaria, and subcutaneous fibrous nodules belong to this class. Herpes zoster is sometimes seen, but probably owes its origin to the arsenic which is administered to such patients. Excessive pigmentation of the skin, when present, is probably always due to the same drug; but absence of pigment in the skin in patches has been described. So, too, alopecia areata is said to have been observed; bald patches on the head, the result of the incessant rubbing against the pillow, must not, however, be mistaken for the baldness of alopecia.

Urine.—An excess of urea was found by Walshe and others; and, according to Todd, uric acid may be deposited in considerable amount. Handfield Jones described a similar increase of phosphates. These conditions of the urine have been found proportional to the severity of the attack of chorea. The pigment urohaemato-porphyrin, discovered by Dr. M'Munn in the urine of rheumatic subjects, was found by Dr. A. E. Garrod in the urine of 14 out of 20 cases of chorea; this he regards as additional evidence of the close relationship between rheumatism and chorea. Albumin, when present, usually indicates nephritis; according to some observers, it may be produced by renal embolism also; of this statement, however, satisfactory proof is wanting. Glycosuria has also been recorded.

Temperature.—The milder forms of chorea may run an apyretic course; and even when the movements are severe no elevation of temperature may be detected. Other cases present a slight degree of pyrexia, irrespective of any detectable complication; there may be a rise of a degree or two in severe cases, and in chorea insaniens a temperature of 104° F. may be reached. Elevation of temperature may, however, occur as a result of some complication, usually arthritic or cardiac; and, though a temperature of 105° F. has preceded death where no complication was detected, hyperpyrexia is usually the result of some complication, and even then in chorea is a rare event, as stated by G. Carpenter in recording the case of a child three and a half years old who died of hyperpyrexia.

Recurrence.—A patient who has once had an attack of chorea is liable to be similarly affected on one or more subsequent occasions. This tendency of the disease to recur was known to Sydenham, and has been insisted on by all subsequent writers. Sir W. Gowers found that a third of his patients had more than one attack of chorea, and he mentions one in whom there had been as many as nine attacks. Prof. Osler's statistics shew that, out of 410 cases, there were two attacks in 110, three in 35, four in 10, five in 12, and six in 3.

The interval between a first and a second attack of chorea varies from a few months to several years, but is most commonly about a year. Instances in which recurrence has been reported within a few weeks of the original attack ought probably to be regarded as relapses of an attack the course of which is not fully spent, rather than as true recurrences. The effect of season on the recurrence of chorea has been noted by a good many observers, but notably by Weir Mitchell. Such fresh attacks occur most commonly in the spring; but instances of their occurrence in the same subject during successive autumns have also been observed. Rheumatism does not appear to have any influence on recurrences; though Prof. Osler reports a higher percentage of arthritic changes in cases of three attacks or more than in those of one or two attacks. As the heart has been more frequently affected in recurrences than in primary attacks, some have supposed that cardiac disease favours such recurrences. It is far more probable, however, that the true explanation of this association is that, as endocarditis tends to occur in chorea, the greater the number of attacks of chorea the more likely is endocarditis to be found. Indeed, evidence of the occurrence of endocarditis during a recurrent attack has been met with in cases in which there were no signs of it at the beginning of the attack.

Females are more prone to a recurrence than are males in about the same proportion as they are more liable to original attacks; but in cases of recurrence there is an increasing tendency for the number of females affected to preponderate over the males; so that in cases of four or more attacks the subjects are nearly always females.

In many cases no cause for the recurrence can be detected; in some it has been ascribed to fright. The relationship between a fright and the onset of the symptoms of chorea is in some such cases so close as to compel us to regard them as cause and effect (p. 526); but it is equally certain that in many cases no evidence whatever of fright can be obtained. Overwork at school has been also regarded as a cause of recurrences. On the relations between pregnancy and chorea sufficient has already been said to shew that the former condition is a potent factor in exciting second and, more rarely, third attacks of chorea in women; and that, exceptionally, pregnancy may be responsible for several recurrences (*vide* p. 525).

As a rule the manifestations of the disease in second and subsequent attacks are not so pronounced as in the first; but many notable exceptions occur in which the symptoms in a recurrence are much more severe than in the original attack. The parts affected by the spasms may be more or less the same in recurrence as in the primary attack; or there may be no such correspondence, the spasm being either more widespread than in the first attack, affecting totally different groups of muscles, or, while involving those originally affected, preponderating in others that formerly escaped. Thus, in some cases successive attacks are more or less restricted to the same side of the body, whilst in others such limitation occurs on the opposite or on the same side indiscriminately; or, again, both sides may become affected, but the opposite to a much greater degree than that which suffered, it may be exclusively, in one or more former attacks.

Diagnosis.—There is no difficulty in diagnosing typical chorea; the spasmodic movements might, however, be mistaken for those seen in some cases of cerebral diseases in infancy; indeed this mistake has probably been made in some of the alleged instances of chorea in very young children. The history serves to distinguish the two conditions: chorea is usually of recent onset, whilst the cerebral condition is one of earliest infancy.

So-called "paralytic chorea" may be very difficult to recognise with certainty; spontaneous movements may be almost entirely absent, and thus the case may come to be regarded as some other form of paralysis. As a rule the history suffices to exclude other forms of paralysis, for the weakness in chorea usually comes on gradually in the course of some weeks. We have seen, however, that there are exceptions, and that considerable paresis may be of sudden onset in this disease. When, moreover, the weakness is hemiplegic in distribution, and there is organic disease of the heart, the diagnostic problem may be one of considerable difficulty; in such cases, however, there is no history of convulsions or loss of consciousness, the paralysis is never so well marked as in occlusion of a cerebral vessel, and is not spastic; moreover, in chorea, even when the leg is affected in conjunction with the arm, the face escapes.

In the case of monoplegic weakness, as for instance of one arm—the most common form of paresis in chorea—the slow onset distinguishes the condition from organic affections; or, if the onset happen to be sudden, the escape of both leg and face and the absence of convulsions and other grave symptoms at the onset exclude an organic cerebral affection; the absence of atrophy and the reaction of degeneration exclude acute anterior poliomyelitis; and they also exclude peripheral neuritis, the exclusion of which may possibly be aided by the absence of any defect of cutaneous sensibility, which is certainly the rule in this class of cases of chorea. Moreover, by carefully watching such patients, very slight spontaneous movements may possibly be detected from time to time, either in the paretic limb or elsewhere; or some such defect may be detected by making the patient hold the arms above the head or straight out in front of her, with or without the tongue protruded at the same time; again, if the child be told to grasp an object with the affected hand, and to keep the hand tightly closed on the object for a little time, she may fail to do so, the muscles involuntarily relaxing in the manner already described as so characteristic of chorea; or, once more, delay may be detected when the patient voluntarily attempts to relax some group of muscles. Sir Clifford Allbutt has told me of a case of paresis of the forefinger of the left hand in a boy of fourteen which proved to be chorea, as was suspected by himself and by an eminent physician to whom he referred the case. The paresis, which came on gradually in a very healthy boy, and was chiefly manifest as a disability to hold the reins of his pony, stood, as a symptom, absolutely alone for many weeks. Then very slight choreic movements and a transient systolic murmur clinched the diagnosis of a case which for a while could only be guessed at on an estimate of probabilities. In a second case under his care, a girl was admitted into the Leeds Infirmary completely or at any rate very deeply paralysed in all four limbs: she lay motionless, but the movements of organic life persisted. This state was said to have come on rather quickly—in a few days. The negative features of the case led to a provisional diagnosis of chorea, and within a week twitches appeared and chorea became unmistakably manifest.

It may be no easy task to distinguish true chorea from the hysterical simulation of it. In cases of the latter kind a history of imitation can often be obtained; the subjects are generally girls about the age of puberty or adolescence; the movements are more sudden, and single muscles contract; furthermore, the movements are often purposive or rhythmical, whereas in true chorea they are always irregular.

As was said when discussing the symptomatology, in cases with pronounced mental symptoms chorea may never be suspected. This is especially apt to be the case in the maniacal form of delirium in which the choreic spasms, though present, are masked by the mental state. Moreover, in some of these cases the choreic spasms cease when the mania is at its height. Acute mania does not usually attack such young subjects, and in it the patients jabber more incessantly than in the delirium of chorea. Careful watch must, of course, be kept for any choreiform movements, and the state of the heart must also be taken into consideration.

Senile chorea (p. 552) is distinguished by reason of the patient's age at the time when it first manifests itself; by the spasm being as a rule slighter in degree than in cases occurring in earlier life; by its usually chronic progressive course, often persisting until the end of life; and by the absence of any relation to rheumatism or endocarditis.

Huntington's chorea (p. 549) has, in common with the senile form, the distinguishing features just described; besides which there is an hereditary history, often dating back several generations. Such cases are even more progressive than the senile form; they invariably persist to the end of life, and are commonly attended by mental deterioration.

In electric chorea (Dubini's disease, p. 554) the spasms are much more sudden and shock-like; and cases of the kind have been met with only in Lombardy and adjacent parts of Italy. The course is progressive; a large proportion of cases end in death, and the more acute are attended with rise of temperature. Epileptiform convulsions are common; and not only does paresis follow, but the muscles waste, and lose their faradic irritability.

Paramyoclonus (p. 649) is distinguished by the fact that the spasms are much more sudden and shock-like; as a rule they affect similar muscles on the two sides of the body, and are commonly restricted to the muscles of the trunk and proximal segments of the limbs; though in some cases they may be much more generalised. This affection is more harmless than chorea, being in no way related to rheumatism or endocarditis, yet more persistent, lasting in most cases for years.

In double athetosis the movements are slow as a rule, are writhing or undulating, and are attended with permanent stiffness of the limbs; there is also, in some cases, an increase in the volume of the muscles. Such patients commonly suffer from convulsive attacks, and as a rule the intellect is permanently defective.

The prognosis of chorea varies with the age of the patient; in children the large majority, even of severe cases, end favourably; the report of the British Medical Association Investigation Committee shewed the death-rate to be about 2 per cent—nine deaths in 439 cases of the disease. According to Anstie, chorea is much more serious after puberty, and its occurrence in pregnancy has been regarded as especially grave. Drs. French and Hicks, however, find no justification for this view, for out of 29 consecutive cases of chorea gravidarum treated at Guy's Hospital, only 3 died. Rise of temperature above 100° F. without complication is regarded by them as of bad omen, and occurred in all of the fatal cases. The majority of the deaths from chorea in pregnancy result from abortion, whether spontaneous or artificially induced; or indeed, in consequence of the extreme debility which commonly ensues in such cases, a natural labour at full term may prove fatal.

As a rule the more pronounced the psychical disturbances the more serious the prognosis; but this is not always so, for patients with marked mental affection may recover completely, whilst on the other hand the most severe spasmodic movements may be present, and may end in death, without definite mental disturbance. It must be remembered that where there is much psychical disturbance, though there may be recovery from the attack of chorea, progressive mental deterioration may follow, and may be permanent. Yet mental defects have been known to persist for months and even years, and ultimately to pass away. It must also be remembered that epileptoid attacks in the course of chorea may subsequently become genuine epilepsy.

Apart from complications, the chief peril is exhaustion from want of sleep and the never-ceasing violent movements. Little less important is the interference with the patient's nutrition, and even with the very acts of taking food. Death rarely occurs in a recurrent attack. These points must, therefore, all be kept clearly before us in attempting to formulate a prognosis in any case of chorea.

Not only have we to express an opinion as to the ultimate result, but also as to the duration of the attack; yet nothing is more uncertain than the latter, for whereas in the majority of cases the attack will come to an end in six weeks, in others it may last as many months. First attacks usually last longer than subsequent ones; the more severe the spasmodic movements the longer is the condition likely to persist; and cases of "paralytic chorea," though presenting little or no danger to life, are often most obstinate. Post-choreic paralysis is sometimes very well marked; but, in the absence of evidence of occlusion of a cerebral vessel, complete restoration of function in the affected parts may be confidently expected.

In some cases the spasmodic movements persist, and result in a chronic chorea which does not appear to affect the general health of the patient materially. Such cases are much more commonly met with in males than in females. The hysterical form, which never ends fatally, often persists for a long time.

The presence of endocarditis does not affect the immediate prognosis, for embolic attacks are singularly rare; but it affects the ultimate prognosis in the two following ways. It may lead to serious structural damage of the affected valves and to the train of phenomena which attend chronic valvular diseases of the heart, ultimately terminating in failure of compensation with its usual consequences; and if a repeated attack of chorea occurs in a person the subject of former heart disease, the prognosis is less favourable in proportion to the gravity of the latter. This, however, is a factor of minor importance in prognosis. It is not a little curious that although chorea and rheumatism are so closely associated, and pericarditis occurs in a fair proportion of cases of chorea, hyperpyrexia is very rarely met with in the latter disease.

Treatment.—Acceptance of the view that chorea is due to microbic infection implies the necessity for a specific serum therapy, as Siegert suggests; but he points to many difficulties which militate against this, and considers that by the time the infective toxin could be determined, the lesions of the nervous system would have already resulted.

It is of primary importance that any possible exciting cause of the affection should be sought for, and combated, if possible, by appropriate treatment. A sedative and calming line of treatment must be adopted where shock appears to have been operative. Where reflex factors, such as errors of refraction, dental irritation, intestinal worms, constipation, phimosis, and the like, are present, the particular source of irritation must be removed. In cases of chorea gravidarum the question of artificial abortion or premature delivery may arise, and this procedure, grave as it is, may in severe cases be imperative. Drs. French and Hicks, however, maintain that this form of chorea does not require different treatment to any other; that the induction of labour is not a specific cure, that it is rarely indicated, for the majority of patients get well without it, and that it is too late to induce labour after pyrexia has set in, rise of temperature being of bad omen in their experience.

Rest is one of the most important factors in the treatment of chorea. Even in mild cases, occurring in children, it is important that all lessons should be discontinued, and that the child should not be allowed to make any mental effort. So, too, it is well to begin the treatment with a few days of absolute rest in bed; this must, however, largely depend on the effect which it has on the child, for in cases in which bed is irksome the consequent mental depression may more than counteract the good effect of the means. Where such absolute rest is not deemed advisable, much good may be obtained by getting the child to lie on a bed or couch for an hour or two in the middle of the morning, and again in the afternoon; and various means may be devised to keep it interested and amused during these periods. Although more important in the severer cases of the disease, the amount of rest must be regulated on similar lines; yet, if possible, the child should be kept in bed until all the choreiform movements have ceased. In severe cases this rule should have no exception. Though the value of rest in modifying or possibly warding off the endocardial lesions in rheumatic fever is fully recognised, it is scarcely realised that this element of treatment may be equally important in similarly modifying or warding off such lesions in chorea. Quite apart from the great good which must result to the motor neurons which are in an exalted state of irritability, there can be little doubt that rest is none the less needed as a prophylactic or curative measure in the treatment of the cardiac manifestations of the disease.

In cases in which the disease is at all pronounced it is well to combine isolation with the rest treatment. We have already seen how highly strung and excitable the subjects of chorea commonly are, so that it will readily be understood how important it is to reduce all mental excitement to a minimum. This can best be effected by keeping the child in a bright airy room with its mother or a sensible nurse to look after it and to keep it interested and amused. An important question which frequently arises in private practice is whether it is advisable to engage a trained hospital nurse in such cases. In the treatment of adults there can be no question that this is the best plan, but no general rule can be laid down on this point with regard to children; each case must be dealt with on its own conditions. If the mother be not herself highly strung or of neurotic temperament, and if the child's own nurse is a sensible and soothing person, it is better not to risk the introduction of a stranger; but where, as so commonly happens, the mother is neurotic, and the nurse has no notion how to keep the child quiet and yet amused, it is better, even at the expense of a little disturbance at the outset, to place a carefully selected trained hospital nurse in charge of the child, and to keep all other people away as much as possible. It need scarcely be added that in selecting the nurse attention should be paid not only to her general qualities, but also to her special experience in the management of children.

If the spasmodic movements are at all severe the patient must be guarded against injury to the limbs. The patient should be on a soft mattress, and any adjacent hard structure carefully padded; prominent parts, like the elbows and knees, may be enveloped in cotton-wool. When the spasms are so severe as to throw the patient out of bed, a mattress should be placed on the floor in the corner of the room, and the adjacent wall padded with cushions or mattresses. If there be the slightest tendency to bed-sore a water-bed should be provided.

Of no less importance than mental and physical rest is an abundant supply of nutritious and easily digested food. Where the spasms of the head and neck are severe, feeding may be attended with great difficulty, and it may be only possible to administer liquids, and these, perhaps, only by a feeding-bottle. The nutrition of the muscles may be improved by massage—a measure which cannot be employed where the spasms are severe, but which is useful in milder cases, and especially in the paralytic forms of chorea.

Owing to the liability to bed-sores scrupulous cleanliness must be observed, especially in those patients who pass their evacuations in the bed; there is commonly so much mental apathy in these cases that the patient may be unaware of mishaps, the early detection of which must depend upon the nurse's watchfulness.

When the movements are not too violent, warm baths do good and are grateful to the patients; care must, of course, be taken to prevent any injury by self-inflicted blows. Though satisfactory results have been obtained by the method of producing diaphoresis by antimonial wine, supplemented by the hot-air bath, it is not a measure to be recommended in the majority of cases of chorea, in which the degree of debility does not permit of further depressing influences. In the severer forms of chorea attended with high temperature the cold pack is useful; or the cold bath may be substituted where the spasmodic movements do not prevent this.

Two classes of drugs are chiefly useful in the treatment of chorea—tonics and sedatives. Of the former class arsenic has a deservedly high reputation. The general condition of the patient much improves under its use; and, whether the drug have any power of controlling the spasms directly or not, we may surmise that it improves the nutrition of the neuron. Whatever be the true explanation of its action, the fact remains that a large number of cases of chorea do well under its use. The drug is usually given by the mouth, in gradually increasing doses, after meals; 3 to 5 minims of liquor arsenicalis may be given three times a day as an initial dose, and the amount increased by 1 or 2 minims every third or fourth day until a dose of 15 minims three times a day is reached, or until the stomach becomes intolerant of the remedy. Koplik is, however, opposed to the use of arsenic in this way owing to its dangers, notably its effects on the kidneys, and contends that if used at all it should be only given in ordinary tonic doses. Owing to the great intolerance of the stomach in some cases, the use of arsenic subcutaneously has been recommended by Eulenburg, Hammond, Wiederhofer, and others—a procedure which is feasible in the treatment of adults, but which is attended with obvious drawbacks in the treatment of children. An advantage claimed for the method is that much larger doses can be given without the intolerance which appears when similar doses are given by the stomach; indeed the administration of the drug is sometimes satisfactorily continued by subcutaneous injection after the gastro-intestinal tract has become intolerant. It must be remembered, however, that such large doses of arsenic as these cannot be given with impunity; for an attack of peripheral neuritis may be the consequence, the treatment thus substituting one disease for another, a state of things hardly to be desired. A much less important effect of the arsenical treatment which sometimes occurs is pigmentation of the skin, an appearance apt to be attributed to an insufficient use of soap and water.

In anaemic subjects of chorea iron may be advantageously combined with arsenic, and if arsenic has to be suspended from any cause, it is a useful substitute. In weakly children the syrup of the phosphate of iron, or some similar preparation, alone or in conjunction with cod-liver oil, is of advantage. Phosphate of lime, cod-liver oil, preparations of malt, and the like, are all of value as adjuvants or temporary substitutes during a course of treatment by arsenic or some other nervine tonic.

Zinc, in the form of either the sulphate or oxide, was formerly much used, the dose being gradually increased up to 20 grains. The valerianate has also been employed, and of late the bromo-valerianate has been recommended.

No good comes of the use of strychnine in the earlier stages or in the acuter manifestations of chorea; but in chronic cases, especially where motor paresis is prominent and the spasmodic movements absent or slight, this drug is often of distinct service.

Good results have been obtained by H. C. Wood with large doses of quinine, a mode of treatment which has found favour more especially with many American physicians. In cases in which there is a personal or family history of rheumatism, salicylate of sodium may be given with advantage in the earlier stages of the attack. Dr. Lees especially advocates its use in conjunction with sodium bicarbonate if cardiac symptoms or the slightest sign of rheumatism are present. Aspirin may be similarly employed, and Dr. Williamson in advocating the use of this drug, in 10 to 15 grain doses four times a day, refers to cases, previously rebellious, which quickly recovered under its use. Antipyrin, in doses gradually increased up to 2 drams in the twenty-four hours, has been recommended.

Its chief use is during the acute stage, and even children tolerate it for weeks. Apart from the more usual signs of intolerance of the remedy, the appearance of albumin in the urine must be watched for, and if it occur the use of the drug must be discontinued. Exalgin, analgen, and asaprol have been similarly employed; Moncorvo being prominent among the physicians who have advocated the use of these drugs and of antipyrin in chorea. Physostigma has its advocates, and there are those who have had recourse to the use of curare in certain chronic cases; in view, however, of the dangers which attend its use, the latter drug is best avoided. D'Orsay Hecht questions the value of medicinal treatment, and instances the results of Wall, who found that the length of time in which patients got well was much the same when they were treated by salicylate of sodium, aspirin, arsenic, or cod-liver oil, together with general measures to secure mental and physical rest and improved hygiene.

Of the sedative class of drugs chloral has been found the most useful; and good results from its use have been recorded, more especially by Sir W. Gairdner and Dr. Charlton Bastian. The patient is kept continuously under the influence of the remedy, the object being to obtain arrest of the spasmodic movements by prolonged sleep. A careful watch must be kept on such patients, and the drug suspended in the event of serious cardiac depression or gastric irritation. It is only in the severer and less tractable forms of chorea that this plan of treatment is to be tried, for it must be admitted that whilst undoubted good appears to result in some cases, in others the natural course of the disease does not appear to be shortened by it; in others again, though the spasms cease or become greatly reduced during the use of the drug, they return with all their former vigour when the treatment is discontinued. Moreover, such patients may be mentally deranged for a variable length of time after the use of the remedy.

The bromides are of little use except in combination with chloral, when they may avert the headache induced by the latter drug, though good results have been credited to them in chorea gravidarum. Sulphonal, trional, paraldehyde, chloralose, and chloretone (W. E. Wynter) have each been employed in the manner recommended for chloral, but my experience of veronal contraindicates its use in this way. Bourneville and Katz allege a rapid cure with bromide of camphor given in capsules of 20 centigrams. Few are bold enough to adopt Jaccoud's plan of giving large doses of opium to attain the same end in young children; indeed, morphine as a sedative in chorea is less potent than chloral, and may increase any mental excitement which is present. Hyoscyamine has been employed; and subcutaneous injections of the hydrochloride or hydrobromide of hyoscine have been useful in severe cases, and Babinski has found subcutaneous injection of hydrobromide of scopolamine similarly useful. Cimicifuga, cannabis indica, conium, and similar drugs have had their advocates, though most of them are of doubtful value.

Counter-irritations to the spine, freezing the skin, and similar measures are not to be recommended.

Electrical treatment has not been attended with any success in chorea, though it has been tried in all its forms. In the later stages of the paretic forms of the disease, however, some good may result from the use of mild galvanic currents to the affected limbs. Similarly, massage is of value in the later stages of chronic cases, a time when carefully planned exercises are also of great value in re-educating the patient in the performance of voluntary movements with precision, and in the proper power, rate, and adjustment of movements. The patient may be directed to touch a certain point with the finger or toe, to make traction with the limb against various degrees of elastic resistance, or to touch different points with the finger or toe a varying number of times in equal intervals. Besides this the patient may be taught to imitate various simple exercises, and to practise the same before a glass, when defects are more readily detected, and the influence of the will is brought more and more to bear on the movements. In the performance of all these exercises care must be taken not to fatigue the patient. Repose should be encouraged in the intervals between the exercises.

Passive movements also serve a useful purpose; for, in addition to their effect on the muscles, after a movement has been performed in this way several times it can be performed voluntarily with greater vigour and rapidity.

Hydropathic treatment is also of value in chronic cases in which a tonic effect is desired, baths and douches being both useful. Sulphur baths also have yielded good results.

CHRONIC PROGRESSIVE CHOREA

SYNONYMS.—*Chronic Adult Chorea; Hereditary Chorea; Huntington's Chorea; Senile Chorea*

This form of chorea manifests itself in adults after the middle period of life, and has a chronic course which is persistent and progressive. Some authors have regarded "Senile chorea" as distinct from "Hereditary" or "Huntington's chorea," as in the former group of cases there is no hereditary history; others have contended that they are one and the same affection, the negative family history being an insufficient reason for separating maladies which in all other respects are identical. Many of the isolated cases of senile chorea conform so closely in their manifestations to those of the hereditary form, that they cannot reasonably be placed in a different category; but some of them differ from the hereditary form not only in the absence of any family tendency, but also in their benign course, in the absence of any mental affection, and in their course to recovery, all of which points, as we shall presently see, are foreign to hereditary chorea.

HEREDITARY CHOREA.—Though Huntington, in a paper published in 1872, was the first to give a comprehensive description of the affection which bears his name, there is evidence that the affection was known to American physicians at least thirty years before. Moreover, Huntington's father and grandfather had both been familiar with the disease and its characters during a period extending over seventy-eight years. Since Huntington described the affection, instances of its occurrence in this country, in Germany, and in France have been recorded.

Causation.—The most prominent etiological factors are the liability for several members of the same family to become affected, and the frequency of direct hereditary transmission. Remak instances a family in which nineteen members were affected; and Lannois one in which there were seventeen such cases; whilst the disease has been known to be transmitted through as many as four generations. It does not appear to be more commonly transmitted through one sex than through the other; and a marked feature in the transmission is that if one generation escape, subsequent ones remain free from the disease; it does not skip a generation or more and then reappear. In some families the proportion of men affected has been in excess of the women; but, as a rule, the two sexes are equally liable to the disease. The symptoms usually manifest themselves between the ages of thirty and forty; but instances have been recorded in which the disease began before this, in a case recorded by Hoffmann in early adolescence. No immediate cause can, as a rule, be detected, though in some cases mental emotions have appeared to act in this way. Neither rheumatism, the infectious fevers, nor pregnancy play any part in the etiology of the disease.

Morbid Anatomy.—Morbid changes have been found, but nothing that can be said to be pathognomonic. The dura mater has been found in a condition of chronic pachymeningitis, with or without haematoma, and may be unduly adherent to the skull; but in a case examined by Berkley there was atrophy of this membrane. Nearly all the cases have presented evidences of a chronic pia-arachnitis, with a varying degree of adhesion of the pia mater to the surface of the cerebral cortex, and Besta considers the leptomeningitis the primary process to which the changes in the brain are secondary. Schultz, however, regards the leptomeningitis as merely the result of complicating intestinal disease. The most common changes met with in the brain itself have been characteristic either of a primary degeneration of the neurons, or of a chronic encephalitis. Macroscopically, atrophy of the convolutions, widening and deepening of the sulci, and compensatory external hydrocephalus have been found. Increased density of the cerebral substance, with thickening of neuroglia, has been described by some, whilst Collins found a cribriform appearance of the brain in his case. Small foci of softening with vacuolation of nerve-cells has been met with; but the most frequent changes, according to certain observers, consist in disseminated foci of round cells in the cortex and subcortical white matter; a regular diffuse encephalitis, according to Oppenheim and Hoppe. Lannois and Paviot have found similar interstitial infiltration most marked about the large pyramidal cells in the central convolutions; atrophy of the small round cells between the first and second cortical layers was found by Oppenheim and Hoppe, and Menzies found the nerve-cells of all the layers degenerated. In several of the cases examined the walls of the cerebral vessels were thickened. Kronthal and Kalischer concluded that the change in the cerebral cortex consists essentially in disease of the blood-vessels, which not uncommonly leads to proliferation of nuclei, punctate haemorrhages, and hyperplasia of glial and interstitial tissue, while the nerve elements themselves are but little affected. Facklam found recent and old haemorrhagic foci, as did Wiedenhämmer, who concluded that the primary condition is a focal or diffuse haemorrhagic encephalitis. On the other hand, the observations of Dana, Clarke, Collins, and Daddi respectively shew the essential change to be a degeneration of the ganglion-cells of the cerebral cortex. Collins found the large pyramidal and polymorphous cells most affected, and the changes, though widespread, were most pronounced in the Rolandic region. The pericellular and perivascular spaces were enlarged, and consecutive and secondary changes were seen in the interstitial tissue and vascular system. Daddi found the small and middle-sized pyramidal cells of the frontal and psycho-motor areas affected, and although the deeper layers were also involved, the superficial was most altered; whilst Modena, who regards degeneration of the nerve elements as the primary process, asks how it is that choreiform movements are not more often seen in arteriosclerosis and chronic inflammation of the cerebrum if vascular disease and increase of the interstitial tissue are responsible for the clinical picture. Diffuse degeneration of the pyramidal tracts was met with in the pons and medulla by Kronthal and Kalischer, who also found a similar change in the antero-lateral columns of the spinal cord, in which region Oppenheim and Hoppe, and Lannois and Paviot described diffuse sclerosis also. Collins in his patient found slight degeneration of the crossed pyramidal tracts in the spinal cord.

Some of the cases in which morbid changes have been described probably belong to the class of cases of senile chorea in which there is no known heredity; others perhaps should be relegated to the class of athetoid spasms, notably instances in which haematoma of the dura or other gross changes have been found; but many of the cases were genuine instances of hereditary chorea.

Pathology.—The pathology of the disease is obscure. The histological changes met with by certain observers point clearly, as we have seen, to a parenchymatous degeneration, in which the neurons suffer primarily, and in which any interstitial change is secondary; but others have found appearances which suggest that a vascular and interstitial change is the basis of the morbid condition. The former of these views has certainly most to recommend it in the present state of our knowledge of the subject; but none of the changes can be regarded as specific. We know little of the nature of the hereditary influence which is so prominent an element in its etiology, though that it depends on some pre-natal factors there can be little question. We are no more fortunate in our knowledge of what determines the manifestations of the disease at the particular time of life when these most commonly first shew themselves. According to Dana, Lannois and Paviot, the defect is to be found in teratology—an hereditary malformation. The latter observers locate this abnormality in the interstitial tissue, whilst Greppin believes that certain epithelioid cells remain embryonic, and, later in life, form the starting-point of the disease. Dana regards the pyramidal cells themselves as possessed of an hereditary taint, there being less power of resistance to excitations. Collins, on the other hand, considers that the ganglion-cells are genetically wanting in power to survive as long under the same environment as the ordinary ganglion-cell. Some, however, with Steyerthal, regard the symptoms as the outcome of functional disturbance of the cerebellum.

Symptoms.—The onset is as a rule gradual, though instances of sudden onset on emotion have been met with. The motor disturbances are usually the first to manifest themselves, and may consist either in irregular movements, first noticed in the face or arm, or in the unsteady gait. Symptoms indicative of mental derangement have rarely preceded the motor manifestations of the disease.

The spasmodic movements resemble those of ordinary juvenile chorea in being involuntary, irregular, and without rhythm; but they differ in being much slower, and in some cases, according to Professor Osler, inco-ordination may be the chief evidence of derangement of the motor processes. In the earlier stages, the muscular contractions are so slight that there may be little or no resulting movement of the limbs; but when the disease is more advanced the spasmodic movements occur both during rest and during some voluntary act; they are increased by emotion, they are lessened by repose. Sometimes they may be arrested voluntarily; but this only results in the earlier stages, when the will retains a limited and temporary influence. Sometimes the involuntary movement is arrested by the substitution of a voluntary effort. The spasmodic movements cannot be arrested voluntarily when the disease is fully manifested; the parts are then in constant agitation, and the movements are suspended only during sleep.

No part is exempt from the spasmodic movements: the ocular muscles have been implicated, causing rolling movements of the globes; grimaces are caused by slow contractions of the facial muscles; contortions of the tongue occur; and in consequence of the spasms the act of deglutition may be difficult. Speech is affected chiefly because of the disordered movements of the tongue. At first it is slow and halting, later the words are pronounced very indistinctly; moreover, the speech may be interrupted by inspiratory or expiratory sounds, the result of laryngeal spasms. The irregular movements of the upper limbs cause characteristic alterations in the handwriting; and as the disease progresses it becomes impossible for the patient to write at all. The attitude and the swaying of the trunk, especially when the patient is walking, resemble closely those of a drunken person. This is very characteristic when fully manifest; the patient may not only sway from side to side, but may lurch more abruptly in one direction, pulling himself up in time before actually falling. Finally, walking becomes altogether impossible, but this result may be delayed for many years.

Muscular power is not diminished until the disease is well advanced. In exceptional cases the muscles are said to waste; but no qualitative alterations of the electrical reactions have been recorded.

There is no defect of general sensibility, and the special senses also escape. The tendon-jerks are usually increased. There is no affection of the sphincters.

One of the most striking features of the disease is the progressive mental deterioration. At first the patient may be depressed or irritable, memory fails, and the intellectual faculties become blunted. Associated with these manifestations of mental change is a strong tendency to suicide, which, however, ceases in the phase of complete dementia. There may be periods of excitement during the otherwise progressive course of the dementia, and there may be hallucinations at such times.

The symptoms persist until death, an event which may not occur for even thirty years after the first manifestations of the disease; it is preceded by marked cachexia in association with the dementia.

Therapeutical measures are powerless to alter the inevitably fatal course of the disease, so that treatment can only alleviate symptoms.

SENILE CHOREA.—This form of chorea occurs in old age, or earlier in the prematurely degenerate; it is not so rare in this country as is the hereditary form. It differs from juvenile chorea in that it is in no way related to rheumatism and endocarditis, any cardiac lesions met with being purely of a degenerative kind. From Huntington's chorea it is distinguished by the absence of family proclivity, by the frequent absence of mental change, and by the occasional termination in recovery.

Some difference of opinion, however, exists among authors in respect of these distinctions. Charcot, Huet, and Féré have regarded the chronic adult forms of chorea as differing from chorea minor only in their substratum; others again regard the isolated cases of senile chorea as identical with the hereditary form. Prof. Osler, for instance, who is an advocate of the latter view, regards heredity, as in Friedreich's ataxia, as only one and an inconstant feature of the chronic progressive choreas.

Causation.—The manifestations of the disease have been known to begin at all ages between fifty-five and eighty; rarely before this time of life. Men and women are both liable to be attacked, the former in greater proportion than in the juvenile form. A family history of other neurotic affections is sometimes to be obtained. Shock or anxiety has acted as an incidental cause in some cases, but in many no such influence can be traced.

Morbid Anatomy and Pathology.—Such changes as have been met with after death are similar to those described in hereditary chorea. Here, too, some of the changes may possibly be secondary to the chronic overaction of the nerve-elements; indeed, whether primary or secondary, we know nothing of the way in which they are brought about.

Symptoms.—Little need be said of the irregular spasmodic movements which resemble those of other kinds of chorea. The upper limbs are usually more affected than the lower, and articulation may be greatly interfered with by the spasms of the muscles of the face and tongue. A certain degree of muscular enfeeblement may be present. Emotion increases the spasms, sleep generally suspends them; they may continue over long periods of years until the patient's death without appearing to shorten life; in exceptional instances, indeed, patients have recovered after a few months or years.

Treatment.—The few patients that have recovered were treated by the usual drugs of recognised value in juvenile chorea, notably arsenic. Sedatives must be judiciously employed to give temporary rest from the incessant movements, especially in cases in which sleep is prevented by them.

CONGENITAL CHOREA.—There are two different conditions to which the name congenital chorea has been applied: in the one the choreiform movements occur in association with rigidity and exaggeration of the tendon-jerks; in the other there is no such rigidity.

Four cases of the flaccid variety have been collected by Ballet, one of which came under his own observation. Males and females are equally liable to be affected, and no circumstances attending the delivery of such children throw any light on the causation. The spasmodic movements begin a few days after birth. In Ballet's case the face and upper limbs were affected first. The spasms are irregular and arrhythmical, of considerable range, are made worse by voluntary movements or emotion, and are suspended in sleep. No cardiac lesions are present.

Though the subjects of this form of congenital chorea are not idiots, they are slow and backward, their intellectual development being below the normal standard. The course of the affection is chronic, and the movements persist in spite of treatment.

This variety is regarded as belonging to the same category as the hereditary chorea of Huntington.

In the cases in which the choreiform movements were associated with rigidity and an exaggerated state of the tendon-jerks, labour was difficult and premature, and the children were idiots; there seems little doubt, therefore, that these cases belong to the group of cerebral diplegias, and that the involuntary movements should be regarded as a form of athetosis rather than as a variety of chorea (*vide* also p. 456).

ELECTRIC CHOREA.—The name "electric chorea" has been applied to various and widely different conditions, but is best reserved for an affection known also as "Dubini's disease," as this physician first described the condition in 1846.

The other conditions to which the name "electric chorea" has been applied are certain cases of ordinary chorea, in which the spasmodic movements are more sudden than usual; cases of hysterical chorea with shock-like movements; and an affection which resembles hysterical chorea, and in which rhythmical spasms involve the neck muscles more especially. This "electric chorea" of Bergeron is a benign affection which rapidly ends in recovery.

Causation.—"Dubini's disease" occurs most commonly in Lombardy and neighbouring parts of Italy, and appears to be due to an acute infection. Its occurrence in certain localities has suggested that it is related in some way to malaria; but the affection is rare even in those parts, and in many other malarial districts it is unknown. Males and females are both liable to be attacked, and it occurs at all ages.

Morbid Anatomy.—No constant changes have been met with in the central nervous system, but the meninges and spinal cord have frequently been found congested.

Pathology.—There is much in the clinical manifestations of the disease to suggest an acute infection. That the toxic agent first attacks the neurons of the cerebral cortex is suggested by the frequent unilateral commencement of the spasms, and by the occurrence of convulsions; but that the neurons of the anterior horns of the spinal cord eventually become affected seems certain from the muscular atrophy which supervenes; however, we have no anatomical evidence in support of either conjecture.

Symptoms.—The disease is ushered in by pains in the neck and back; the temperature is raised in proportion to the degree of acuteness of the case, and may be raised throughout the course of the affection. Spasmodic movements occur as one of the earliest manifestations; one of the upper limbs is affected first, the movements beginning either in the upper part of the arm or in the fingers. Next, the spasms most commonly affect the muscles of the leg of the same side before becoming generalised. The muscular contractions differ from those in ordinary chorea by being sudden and shock-like, exactly like those evoked by momentary electrical excitations. In addition to these movements epileptiform convulsions occur in a good many of the cases, and are sometimes restricted to one side of the body. Palsies may follow such attacks; but, apart from this, in patients who survive for some months, the limbs become feeble in the order in which they were affected by the shock-like spasms, the paresis in the end becoming general. The muscles waste and no longer respond to faradism.

The disease pursues a progressive course to a fatal termination, which is usually postponed for several months, but may take place after but a few days' illness. Death may follow an epileptiform convulsion, or there may be a gradually deepening coma.

Treatment does not appear to be capable of altering the progressive course of the disease, or of averting death.

J. S. Risien Russell.

REFERENCES

General.—1. Appenzell. *Corr.-Bl. f. schweiz. Ärzte*, Bern, 1898, xxviii. 408, 439.—2. Babington. *Guy's Hosp. Rep.*, Lond., 1841, vi. 411.—3. Baxter. *Brain*, Lond., 1880, ii. 124.—4. Bernt. *Monographia Choreae St. Viti*, Prag, 1810.—5. Bouteille. *Traité de la chorée*, Paris, 1810.—6. Bright. *Reports on Medical Cases*, 1831, vol. ii. part 2, p. 468.—7. Cartier. *Quelques considérations sur la symptomatologie et la nature de la chorée*, Paris, 1876.—8. Féré. *Twentieth Century Practice of Medicine*, Lond., 1897, x. 661.—9. Goodall. *Guy's Hosp. Rep.*, Lond., 1890, xlvii. 35.—10. Gowers. *Diseases of the Nervous System*, Lond., 2nd ed., 1893, vol. ii. 591.—11. Huet. Thèse de Paris, 1889.—12. Hughes. *Guy's Hosp. Rep.*, Lond., 1846, iv. 360.—13. Hughes and Burton Brown. *Ibid.*, 1855, 3. s., i. 217.—14. Krafft-Ebing. *Allg. Wien. med. Ztg.*, 1897, xlii. 25, 37, 47.—15. Mitchell and Rhein. *Phila. Med. Journ.*, 1898, i. 153.—16. Moncorvo. *Bull. Acad. de méd.*, Paris, 1898, xxxix. 574; *Pediatrics*, New York, 1898, vi. 337.—17. Osler. *On Chorea and Choreiform Affections*, Lond., 1894.—18. Pritchard. *Med. News*, N.Y., 1899, lxix. 228.—19. Pye-Smith. *Guy's Hosp. Rep.*, Lond., 1874, xix. 329.—20. Roeser. *Journ. der pract. Heilk.*, Berl., 1828, lxvii. 54.—21. Roger. *Arch. gén. de méd.*, Paris, 1866, 7^e s., viii. 641; *ibid.*, 1867, 7^e s., ix. 54.—22. Russell, J. W. *Lancet*, Lond., 1899, i. 894.—23. Sée. *De la chorée et des affections nerveuses*, Paris, 1851.—24. Sinkler. Dercum's *Nervous Diseases by Amer. Authors*, Edin., 1895, 227.—25. Stewart. *Edin. Med. Journ.*, 1899, N.S. v. 257.—26. Sturges. *On Chorea*, 2nd ed., Lond., 1893.—27. Sydenham's Works, Latham's translation, vol. ii. 198 (Syd. Soc. ed.).—28. Tomkins. *Amer. Journ. Obst.*, N.Y., 1897, xxxv. 335.

Etiology.—29. Brush. *Med. Rec.*, N.Y., 1895, xlvii. 463.—30. Burr. *Arch. Pediat.*, N.Y., 1898, xv. 29.—31. Carslaw. *Glasgow Med. Journ.*, 1891, xxxv. 229.—32. Comby. *Bull. et mém. Soc. méd. des hôp.*, 1891, viii. 261.—33. Fletcher, M. *St. Barth. Hosp. Rep.*, Lond., 1897, xxxii. 383.—34. Ginzzetti. *Riforma med.*, Napoli, 1893, ix. 422, 434, 448.—35. Haushalter. *Rev. méd. de l'est*, Nancy, 1894, xxvi. 233.—36. Legay. Thèse de Paris, 1897.—37. Lewis. *Trans. Assoc. Amer. Phys.*, 1892, vii. 249.—38. Litten. *Charité-Ann.*, Berl., 1884, xi. 265.—39. Mackenzie. *Brit. Med. Journ.*, 1887, i. 425.—40. Maragliano. *Centralbl. f. innere Med.*, Leipz., 1899, xx. 489.—41. Marfan. *Semaine méd.*, Paris, 1897, xvii. 153.—42. Mettler. *Amer. Journ. Med. Sc.*, Phila., 1903, cxxvi. 481.—43. Money. *Brain*, Lond., 1882, v. 513.—44. Owen. *Trans. Internat. Med. Congr.*, Washington, 1887, v. 157.—45. Peiper. *Deutsch. med. Woch.*, Berl., 1885, xi. 116.—46. Poynton and Holmes. *Lancet*, Lond., 1906, ii. 982.—47. Poynton and Paine. *Ibid.*, 1905, ii. 1760.—48. Priestley. *Brit. Med. Journ.*, 1897, ii. 805.—49. Rachford. *Med. News*, Phila., 1893, lxii. 429.—50. Ranney. *Med. Rec.*, N.Y., 1894, xlv. 551, 582.—51. De Schweinitz. *N.Y. Med. Journ.*, 1888, xlvii. 679.—52. Siegert. *Med. Klinik*, Berl., 1908, iv. 1899.—53. Sinkler. Pepper's *Syst. Med.*, 1886, v. 439.—54. Steiner. *Jahrb. f. Kinderheilk.*, Wien, 1870, iii. 291.—55. Stevens. *Trans. N.Y. Acad. Med.*, 1876, ii. 438.—56. Sturges. *Lancet*, 1888, ii. 605.—57. Triboulet. Thèse de Paris, 1893.—58. Straton. *Brit. Med. Journ.*, 1885, ii. 437.—59. Wichmann. *Deutsch. med. Wchnschr.*, Berl., 1890, xvi. 632, 659.

Rheumatism.—60. Batten. *Lancet*, 1898, ii. 1195.—61. Brown. *Journ. Nerv. and Ment. Dis.*, N.Y., 1893, xviii. 513.—62. Duckworth. *Atti d. XI. cong. med. internaz.*, Rome, 1894, iii. Med. int. 354.—63. *Idem. Wien. med. Bl.*, 1894, xvii. 192, 208.—63*a*. Fagge. *Principles and Practice of Medicine*, 1886, i. 641.—64. Garrod. *Med.-Chir. Trans.*, Lond., 1889, lxxii. 145; *Lancet*, 1892, i. 793.—65. Guck. *Am. Med.-Surg. Bull.*, N.Y., 1896, x. 572.—66. Herringham. *Med.-Chir. Trans.*, Lond., 1889, lxxii. 117.—67. Meyer. *Jahrb. f. Kinderh.*, Wien, 1895, xl. 1, 144.—68. Poynton and Holmes. *Lancet*, Lond., 1906, ii. 982.—69. Scheele. *Deutsch. med. Woch.*, Berl., 1885, xi. 702.—70. Syers. *Lancet*, 1889, ii. 1271.—71. Townsend. *Trans. Amer. Pediat. Soc.*, 1892, iv.

Pregnancy.—72. Arai. *Ein Beitrag zur Kenntnis der Chorea Gravidarum*, 1895.—73. Barnes. *Trans. Obstet. Soc.*, Lond., 1868, x. 147.—74. Bué. *Presse méd.*, Par., 1894, 279.—75. Cameron. *Montreal Med. Journ.*, 1896, xxiv. 491.—76. Dakin. *Practitioner*, Lond., 1897, lix. 571.—77. Delage. *Ann. de la policlin. de Par.*, 1898, viii. 121.—78. Dufournier. *Bull. méd.*, Par., 1894, viii. 1031.—79. Festenberg. *Deutsch. med. Wchnschr.*, 1897, xxiii. 196.—80. French and Hicks. *Practitioner*, Lond., 1906, lxxvii. 178.—81. Jones. *Trans. Obst. Soc.*, Lond., 1889, xxxi. 243.—82. Levick. *Amer. Journ. Med. Sc.*, Phila., 1862, xliii. 40.—83. McCann. *Trans. Obst. Soc.*, Lond., 1891, xxxiii. 413, 466.—84. Schwechten. *Über Chorea Gravidarum*, Inaug. Diss., Berlin, 1876.

Hysteria.—85. Albert. *Gaz. des hôp.*, Paris, 1895, lxviii. 262.—86. Auché. *Progr. méd.*, Paris, 1891, xiv. 435.—87. Auché et Carrière. *Arch. clin. de Bordeaux*, 1895, iv. 74.—88. Bouchaud. *Journ. des sc. méd. de Lille*, 1898, i. 551.—89. Comby. *Bull. et mém. Soc. méd. des hôp.*, Paris, 1891, viii. 261.—90. Dettling. Thèse de Lyon, 1892.—91. Duchateau. Thèse de Paris, 1893.—92. Horcholle. Thèse de Paris, 1888.—93. Joffroy. *Bull. et mém. Soc. méd. des hôp.*, Paris, 1891, viii. 143.—94. Laveran. *Ibid.*, 285.—95. Séglas. *Ibid.*, 164.

Mental.—96. Arndt. *Arch. f. Psychiat.*, Berl., 1869, i. 509.—97. Buccelli. *Riv. sper. di freniat.*, Reggio-Emilia, 1902, xxviii. (*Orig.*) 1.—98. Bernstein. *Allg. Ztschr. f. Psychiat.*, Berl., 1897, liii. 538.—99. Burr. *Journ. Nerv. and Ment. Dis.*, N.Y., 1908, xxxv. 353.—100. Dieckerhoff. *Chorea und Geistesstörung*, Bonn, 1894.—101. Diller. *Amer. Journ. Med. Sc.*, Phila., 1890, xcix. 329.—102. Gay. *Brain*, Lond., 1889, xii. 151.—103. Knauer. *Monatsschr. f. Psychiat. u. Neurol.*, Berl., 1897, i. 339.—104. Kurella. *Allg. Ztschr. f. Psychiat.*, Berl., 1895, li. 770.—105. Lewis. *Med. Times and Gaz.*, Lond., 1876, ii. 280.—106. Marcé. *De l'état mental dans la chorée*, Paris, 1860; *Bull. Acad. de méd.*, Paris, 1859-60, xxiv. 741, 1076.—107. Meyer. *Arch. f. Psychiat.*, Berl., 1870, ii. 535.—108. Powell. *Brain*, Lond., 1889, xii. 157.—109. Zinn. *Arch. f. Psychiat.*, Berl., 1896, xxviii. 411.

Reflexes.—109*a*. Bury. *Med. Chronicle*, 1909, li. 141.—109*b*. Gordon. *Brit. Med. Journ.*, 1901, i. 765.—109*c*. Oddo. *Gaz. des hôp. de Paris*, 1900, 1343.—109*d*. Sinkler. *Dercum's Nervous Diseases by American Authors*, Edin., 1895, 240.

Pathology.—110. Apert. *Compt. rend. Soc. de biol.*, Paris, 1898, v. 128.—111. Berkley. *Johns Hopkins Hosp. Rep.*, 1891, ii. 317.—112. Carpenter. *Lancet*, Lond., 1907, ii. 1521.—113. Cesaris-Demel. *Gazz. med. di Torino*, 1897, xlviii. 581, 601.—114. Chéron. *Gaz. des hôp.*, Paris, 1895, lxviii. 1457; *Journ. de méd. de Par.*, 1896, viii. 184, 198, 231.—115. Cramer und Többen. *Monatsschr. f. Psychiat. u. Neurol.*, Berl., 1906, xviii. 509.—116. Dana. *Brain*, Lond., 1890, xiii. 71.—117. *Idem. Amer. Journ. Med. Sc.*, Phila., 1894, cvii. 31.—118. Delcourt et Sand. *Arch. de méd. des enf.*, Paris, 1908, ii. 826.—119. Donkin and Hebb. *Med. Times and Gaz.*, Lond., 1884, ii. 743.—120. Ecomono und Karplus. *Deutsche Ztschr. f. Nervenh.*, Leipz., 1909, xxxvi. 166.—121. Elischer. *Virchows Arch.*, Berl., 1874, lxi. 485; 1875, lxiii. 104.—122. Gowers, Sir W. *Phonogr. Rec. of Clin. Teaching and Med. Sc.*, Lond., 1904, x. 113, 129.—123. von Halban und Infeld. *Arb. aus d. Neurol. Inst. zu Wien*, 1902.—124. Handford. *Brain*, 1889, xii. 129.—125. Jakowenko. *Wiestnik psichiatr. i. nerv. patol.*, 1889.—126. *Idem. Neurol. Centralbl.*, Leipz., 1889, viii. 484.—127. Langmead. *Lancet*, Lond., 1908, i. 154.—128. Lewis. *Med. Times and Gaz.*, Lond., 1876, ii. 280.—129. Leroux. *Presse méd.*, Par., 1896, 141.—130. Maragliano. *Centralbl. f. innere Med.*, Leipz., 1899, xx. 489.—131. Marfan. *Sem. méd.*, Paris, 1897, xvii. 153.—132. Money. *Med.-Chir. Trans.*, Lond., 1885, lxviii. 277.—133. Monakow. *Gehirnpathologie*, Wien, 1897, 1.—134. Naunyn. *Mitt. aus d. med. Klin. zu Königsberg*, 1888, 296.—135. Nauwerck. *Beitr. z. path. Anat. u. Phys.*, Jena, 1886, i. 407.—136. Orzechowski. *Arb. aus d. Neurol. Inst. a. d. Wien. Univ.*, Wien, 1907, xvi., Teil 2, 530.—137. Patella. *Gazz. d. osp.*, 1888, ix. 573.—138. Pianese. *La Natura infettiva della corea del Sydenham.* Napoli, 1893.—139. Richter. *Western Lancet*, xii. 529.—140. Triboulet. Thèse de Paris, 1893.—141. Turner, F. C. *Trans. Path. Soc.*, Lond., 1892, xliii. 8.—142. Wollenberg. *Arch. f. Psychiat.*, Berl., 1892, xxiii. 167.—143. Wood. *Journ. Nerv. and Ment. Dis.*, Chicago, 1893, xx. 241.

Paralytic.—144. Albarel. *Journ. de clin. et de thérap. inf.*, Paris, 1897, v. 803.—145. Ball. *Phila. Med. Journ.* 1898, i. 299.—146. Filatow. *Arch. f. Kinderh.*, Stuttg., 1894-95, xviii. 432.—147. Gingeot. *Gaz. hebd. de méd.*, Paris, 1887, xxiv. 129.—148. Gowers. *Brit. Med. Journ.*, 1881, i. 636.—149. Gompertz. *Allg. med. Centr.-Ztg.*, Berl., 1896, lxv. 85.—150. Ollive. *Des paralysies chez les choréiques.* Thèse de Paris, 1884.—151. Raymond. *Bull. méd.*, Paris, 1896, x. 1203.—152. Solaro.

Pediatria, Napoli, 1896, iv. 169.—153. TODD. *Med. Times and Gaz.*, Lond., 1852, xxvi. 79.

Congenital.—154. BALLET. *Bull. méd.*, Paris, 1896, x. 223.—155. BROWN-SÉQUARD. *Med. Times and Gaz.*, Lond., 1863, ii. 92.—156. COLLINS. *N.Y. Med. Journ.*, 1892, lvi. 159.—157. *Idem. Journ. Nerv. and Ment. Dis.*, Chicago, 1892, xix. 726.—158. DILLER. *Am. Journ. Med. Sc.*, Phila., 1890, xcix. 329.—159. FOX. *Brit. Med. Journ.*, 1873, ii. 9.—160. GRAY. *N.Y. Med. Journ.*, 1892, lvi. 159.—161. *Idem. Journ. Nerv. and Ment. Dis.*, Chicago, 1892, xix. 726.—162. HELLER. *Wien. med. Wchnschr.*, 1876, xxvi. 455.—163. HIRSCH. Inaug.-Diss. Leipz., 1895.—164. JOHNSTON. *Am. Journ. Med. Sc.*, Phila., 1895, cx. 377.—165. OSLER. *Journ. Nerv. and Ment. Dis.*, Chicago, 1893, xx. 97. — 166. PRESTON. *N.Y. Med. Journ.*, 1896, lxiii. 350.—167. RIEDER. *München. med. Wchnschr.*, 1889, xxxv. 623.—168. DE ST. FLORENT. *La Chorée congénitale*, Paris, 1896.—169. SCHLESINGER. *Ztschr. f. klin. Med.*, Berl., 1892, xx. 506.

Chronic Progressive.—170. BERKLEY. *Med. News*, N.Y., 1883, xliii. 200.—171. BESTA. *Riv. sper. di freniat.*, Reggio-Emilia, 1905, xxi. (*Orig.*) 205.—172. BIERNACKI. *Berlin. klin. Wchnschr.*, 1890, xxvii. 485.—173. BIONDI. *Boll. d. clin.*, Milan, 1897, xiv. 385.—174. BONDURANT. *Alienist and Neurol.*, St. Louis, 1896, xvii. 247.—175. *Idem. Journ. Nerv. and Ment. Dis.*, N.Y., 1896, xxiii. 680.—176. BRISSAUD. *Rev. neurol.*, Paris, 1896, iv. 417.—177. CHAUFFARD. *Bull. et mém. Soc. méd. des hôp.*, Paris, 1895, xii. 312.—178. CIRINCIONE e MIRTO. *Psichiatria, la neuropatologia*, etc., 1890, vii. 343 ; viii. 18.—179. CLARKE. *Brain*, Lond., 1897, xx. 22.—180. COLLINS. *Boston Med. and Surg. Journ.*, 1897, cxxxvii. 657.—181. *Idem. Med. News*, N.Y., 1898, lxii. 31.—182. *Idem. Am. Journ. Med. Sc.*, Phila., 1898, cxvi. 275.—183. DADDI. *Riv. di patol. nerv.*, Firenze, 1905, x. 153.—184. DANA. *Journ. Nerv. and Ment. Dis.*, N.Y., 1895, xxii. 565.—185. DILLER. *Am. Journ. Med. Sc.*, 1889, xcviii. 585.—186. DREVES. Inaug.-Diss. Göttingen, 1891.—187. DUGLISON'S *Practice of Medicine*, 1st ed. 1842, vol. ii. p. 312 ; 3rd ed. 1848, ii. p. 216.—188. EWALD. *Ztschr. f. klin. Med.*, Berl., 1884, supplement to Bd. vii. S. 51.—189. FACKLAM. *Arch. f. Psychiat.*, Berl., 1898, xxx. 137.—190. GEDDES and CLINCH. *Journ. Ment. Sc.*, Lond., 1898, xliv. 811.—191. GREPPIN. *Arch. f. Psychiat.*, 1892, xxiv. 155. — 191*a*. HATTIE. *Amer. Journ. Insan.*, Balt., 1909, lxvi. 123. — 192. HERRINGHAM. *Brain*, Lond., 1888, xi. 134.—193. HOFFMANN. *Virchows Arch.*, 1888, cxi. 513.—194. HUBER. *Ibid.*, 1887, cviii. 267.—195. HUET. *De la chorée chronique*, Paris, 1889. — 196. HUNTINGTON. *Med. and Surg. Reporter*, Phila., 1872, xxvi. 317.—197. JOLLY. *Neurol. Centralbl.*, Leipz., 1891, x. 321.—198. JORDEL. *Contribution à l'histoire de la chorée chronique*, Nancy, 1894.—199. KAST. *Deutsche med. Wchnschr.*, Berl., 1895, xxi. 186.—200. KRONTHAL und KALISCHER. *Neurol. Centralbl.*, Leipz., 1892, xi. 592, 631.—201. *Idem. Virchows Arch.*, Berl., 1895, cxxxix. 303.—202. LANNOIS et PAVIOT. *Rev. de méd.*, Paris, 1898, xviii. 207.—203. MASSALONGO. *Policlin.*, Rome, 1895, ii. 481.—204. MENZIES. *Journ. Ment. Sc.* Lond., 1892, xxxviii. 560 ; 1893, xxxix. 50.—205. MODENA. *Ann. del prov. di Ancona*, 1905, iii. 55.—206. MORPURGO. *Riv. sper. di freniat.*, Reggio-Emilia, 1898, xxiv. 498.—207. OPPENHEIM und HOPPE. *Arch. f. Psychiat.*, Berl., 1893, xxv. 617. —208. OSLER. *Journ. Nerv. and Ment. Dis.*, N.Y., 1893, xviii. 97.—209. *Idem. Johns Hopkins Hosp. Bull.*, 1894, v. 119.—210. PATRY. Thèse de Paris, 1897.—211. PERETTI. *Berlin. klin. Wchnschr.*, 1885, xxii. 824.—212. PHELPS. *Journ. Nerv. and Ment. Dis.*, N.Y., 1892, xix. 765.—213. REMAK. *Neurol. Centralbl.*, Leipz., 1891, x. 326.—214. RIESMAN. *Am. Journ. Med. Sc.*, Phila., 1897, cxiv. 180.—215. DE ROSSI. *Clin. med. ital.*, Milan, 1898, xxxvii. 502.—216. RUSK. *Journ. Nerv. and Ment. Dis.*, N.Y., 1903, xxx. 172.—217. SCHILLING. *Deutsch. med. Ztg.*, Berl., 1895, xvi. 559.—218. SCHLESINGER. *Ztschr. f. klin. Med.*, Berl., 1892, xx. 127.—219. SCHMIDT. *Deutsch. med. Wchnschr.*, 1892, xviii. 585.—220. SCHULTZ. *Charité-Ann.*, Berl., 1908, xxxii. 189.—221. SINKLER. *Journ. Nerv. and Ment. Dis.*, N.Y., 1889, xiv. 69.—221*a*. STEVENSON and PURDUM. *Amer. Journ. Insan.*, Balt., 1909, lxvi. 129.—222. STEYERTHAL. *Arch. f. Psychiat.*, Berl., 1908, xliv. 656.—223. VILLANI. *Riforma med.*, Napoli, 1897, xiii. 795, 808, 818, 832.—224. WIEDENHÄMMER. *Neurol. Centralbl.*, Leipz., 1907, xx. 116.

Treatment.—225. BABINSKI. *Rev. neurol.*, Paris, 1907, xv. 86.—226. BASTIAN *Lancet*, 1889, ii. 55.—227. BOURNEVILLE et KATZ. *Progr. méd.*, Paris, 1898, viii. 33 —228. CHÉRON. *Gaz. des hôp.*, Paris, 1895, lxviii. 1457.—229. *Idem. Journ. de méd de Paris*, 1896, viii. 184, 198, 231.—230. CHURTON. *Internat. Clin.*, Phila., 1895, ii.

119.—231. COMBY. *Méd. mod.*, Paris, 1896, vii. 529.—232. COUGNOT. Thèse de Paris, 1895.—233. *Idem. Journ. de méd. et chir. prat.*, Paris, 1895, lxvi. 864.—234. DORLAND and POTTS. *Journ. Am. Med. Assoc.*, Chicago, 1893, xxi. 919.—235. DRESCH. *Bull. gén. de thérap.*, Paris, 1890, cxviii. 337.—236. DUJARDIN-BEAUMETZ. *Ibid.*, 1894, cxxvi. 193.—237. GAIRDNER. *Lancet*, Lond., 1889, ii. 205.—238. HECHT, D'O. *Illinois Med. Journ.*, Nov. 1907.—239. HUBRECHT. Thèse de Paris, 1895.—240. KNAPP. *Boston Med. and Surg. Journ.*, 1895, cxxxii. 204.—241. KOPLIK. *Med. Rec.*, N.Y., 1908, lxxiii. 95.—242. LEES, D. B. *Brit. Journ. Child. Dis.*, Lond., 1909, vi. 97.—243. LOVELAND. *Univ. Med. Mag.*, Phila., 1893-94, vi. 605.—244. METTENHEIMER. *Arch. f. Kinderh.*, Stuttg., 1894, xvii. 417.—245. MONCORVO. *Journ. de clin. et de thérap. inf.*, Paris, 1896, iv. 967 ; 1898, vi. 401, 427, 450.—246. *Idem. Rev. mens. des mal. de l'enf.*, Paris, 1895, xiii. 238.—247. *Idem. Bull. de l'Acad. de méd.*, Paris, 1898, xxxix. 574.—248. OVEREND. *Lancet*, 1897, ii. 248.—249. RANNEY. *Med. Rec.*, N.Y., 1894, xlv. 551, 582.—250. REGNIER. *Arch. de neurol.*, Paris, 1895, xxix. 380.—251. ROCKWELL. *Journ. Am. Med. Assoc.*, Chicago, 1895, xxv. 1092.—252. *Idem. Med. Rec.*, N.Y., 1895, xlviii. 148.—253. SACCA. *Gazz. med. lomb.*, Milano, 1898, lvii. 197.—254. SIEGERT. *Med. Klinik*, Berl., 1908, iv. 1899.—255. WARNER. *Internat. Clin.*, Phila., 1897, i. 208.—256. WEISS. *Allg. Wien. med. Ztg.*, 1894, xxxix. 467.—257. WILLIAMSON. *Lancet*, Lond., 1903, ii. 526.—258. WOOD. *Trans. Assoc. Amer. Phys.*, Phila., 1893, viii. 101.—259. WYNTER (Chloretone), *Brit. Med. Journ.*, 1908, ii. 912.

J. S. R. R.

MIGRAINE

SYNONYMS.—*Sick Headache*; *Bilious Headache*; *Megrim*; *Hemicrania*.

By JAMES MACKENZIE, M.D., LL.D.

Definition. — A functional disorder of the central nervous system, characterised by paroxysmal attacks of headache, usually limited to one side and associated with sickness, mental depression, affections of sight, and other nervous phenomena.

Although headache is the most prominent feature, yet the symptoms appearing in other parts of the system give the characteristics peculiar to this complaint. The accompanying mental lassitude and depression, the tendency to nausea and vomiting, the cold and shivery feelings that pass over the skin, the disagreeable affections of sight and other distressing sensations combine, with the actual pain in the head, to render life, for the time being, miserable to the sufferer, and to stamp the condition as a distinct and readily recognisable symptom-group.

As this complaint is specially frequent amongst the highly-educated and observant, and as many of the victims have been medical men, its symptoms have been carefully noted and its features frequently discussed. I have been a sufferer since my earliest recollections, and have been in the habit of comparing my experiences with those of fellow-sufferers under my care, and with the published descriptions of the disorder. The result of this personal experience is embodied in this article.

Etiology.—In a large number of cases the affection is undoubtedly hereditary. When thus transmitted the features of the complaint may be wonderfully alike in parent and offspring; more frequently, however, each case has distinctive peculiarities of its own. Migraine usually appears in the earlier decades of life, rarely coming on for the first time after the age of thirty. After forty-five years of age, sometimes earlier, the attacks are prone to abate, becoming less frequent and less violent, until with advancing years they may finally disappear, or, what is perhaps more usual, the nature of the attacks alter, so that the headache and gastric symptoms become less severe, whilst other phenomena, such as the vasomotor and ocular, become more pronounced. Migraine may, however, persist with severity till late in life, and sufferers at seventy years of age are not uncommon. It has been suggested that persons of certain constitutions, such as the gouty, the hysterical, and the subjects of other neuroses (asthma, epilepsy, tics) are more liable to migraine than others, and even that migraine is but one of the symptoms of these conditions. But considering the universality of the complaint, and that many sufferers do not shew any evidence whatever of any constitutional dyscrasia, it seems more reasonable to assume that there is no relation of the kind, and that migraine is an independent disorder manifested in a gouty, hysterical, or neurotic subject. Women are undoubtedly more liable to the disorder than men.

One of the most peculiar features of migraine is the periodicity of the attacks. Many patients know when an attack is due, although there may not be any premonitory symptoms. They are also aware that during this period certain contributory causes, such as indiscretions in diet, irregularity in the routine of their lives, disturbing impressions upon their senses, breathing vitiated air, exposure to damp or cold, may suffice to induce an attack. After the attack is over they usually feel extremely well, and can expose themselves to these contributory causes with impunity, being assured that for the time being they are not liable to an attack. But gradually this immunity wears off and the liability returns, with its expected attack, again to be followed by another period of immunity. In many cases in which the conditions of life are detrimental to bodily health, the period of immunity becomes so short that the patient is almost continuously suffering from a dull headache, not necessarily migrainous in origin, but the individual is liable to frequent attacks of true migraine (status hemicranialis).

Another feature bearing on the etiology is the freedom from attacks that may arise in certain circumstances. As already mentioned, the patient after an attack is insusceptible for a period. The onset of other ailments, for example, fevers, may put an end to the attacks; thus, among 2000 cases of enteric fever observed by Dr. F. Hare no attack of migraine ever occurred. This writer ingeniously suggests that the freedom from attacks, which follows the insertion of a seton in the neck, is really due to the absorption of products from an infected wound. The development of other ailments, for example, epilepsy, has been noted to coincide with the disappearance of migrainous attacks.

Pathology.—No demonstrable lesion has been found to account for the symptoms in migraine. The cause of the disorder has therefore been a subject of innumerable speculations. The symptoms are so varied, appear in such different parts of the body, are excited by so many causes, and have affinities with such a number of disorders that plausible hypotheses to account for the attacks are wide in range, diverse, and often contradictory. Dr. E. Liveing sums up the most noteworthy of those prevalent when he wrote his masterly work on this subject (1873). There was the old conception of biliousness, the hypothesis that migraine was a neurosis arising from sympathy or from reflex irritation from other organs, such as the stomach, uterus, or the eyes. There were the vascular explanations, namely, arterial hyperaemia, venous engorgement, and spasm or relaxation of the smaller vessels. Some abnormal stimulation of the sympathetic nervous system was looked upon by many as the essential cause, whilst Dr. Liveing inclined to the hypothesis of nerve-storm. The anatomical seat of the lesion is given by Wollaston as probably in the optic thalamus, a position also favoured by Dr. Liveing. Du Bois Reymond on the other hand assumed the seat to be in the cilio-spinal region.

Since the publication of Dr. Liveing's work in 1873, surmises and speculations have continued to be propounded. Of the more recent hypotheses there is a mechanical one by Spitzer, who suggests that in migrainous subjects there is a congenital narrowing of the foramen of Monro, into which, at times, the choroid plexus slips and blocks up the passage; in consequence of the retention of the fluids in the lateral ventricles, pressure is exerted on the cerebral cortex thus inducing the symptoms of migraine; and that the sudden relief is due to the prolapse of the choroid plexus and sudden escape of the dammed-up fluid. The conception of the ophthalmic origin of migraine has been revived, and some ardent advocates maintain that all migraines are due to eye-strain. The circulation of morbid products in the blood from absorption of toxins from the intestinal canal, or the result of metabolism, is held by many to be the cause of migraine. One of these hypotheses has gained a great vogue chiefly through the writings of Dr. Haig, who attributes the whole mischief of this and many other complaints to excess of uric acid in the blood. It must be admitted, however, that his arguments, when considered dispassionately, do not carry conviction, whilst the chemical analysis on which he bases many of his arguments is not confirmed by other investigators. Dr. Luff indeed states that though Dr. Haig makes "constant references to the amount of uric acid in the blood, varying under different conditions, not once did he give any estimation of the uric acid in the blood nor any account of the extraction of uric acid from the blood." Dr. F. Hare's explanation is diametrically opposed to Dr. Haig's, and is to the effect that migraine and other paroxysmal neuroses "are often due to a gradually increasing accumulation in the blood of unoxidised carbonaceous material derived from the food; that sooner or later, according to circumstances, the load becomes intolerable; and that then there occurs, through the medium of the nervous system, a more or less precipitate and complex conservative reaction on the part of the organism, resulting in some degree of temporary relief; in short, that the paroxysmal neuroses depend upon hyperpyraemia and constitute acarbonising processes." (For Plavec's hypothesis, see p. 611.)

Consideration of all the different views and of the data adduced in their support leads to the conclusion that the symptoms of migraine arise from stimulation of certain areas in the central nervous system, and that this stimulation may be provoked in many ways, as by pressure, by reflex stimulation, or by the circulation in the blood of certain materials, the nature of which has not yet been satisfactorily determined. The region of the central nervous system that is thus susceptible to stimulation is still unknown. It is scarcely correct in the present state of our knowledge to speak of migraine as a "disease" in the sense that there is some definite anatomical lesion. Many parts of the central nervous system may react in a specific way when stimulated, for instance, many people become pallid and vomit when they smoke tobacco. This does not indicate a disease of the nervous system, but a susceptibility to a certain form of poison. The freedom from attacks of migraine, which may last for months or years or become permanent, does not imply a transient or permanent cure of a "disease," but only that this particular region is no longer stimulated in a particular way or that it has lost its sensitiveness or become habituated to the stimulus. The position of this susceptible region in the central nervous system can only be determined when the nature and mechanism of the symptoms produced are understood. When these are fully appreciated, then by pursuing the methods employed for detecting a focal lesion in the brain and spinal cord, the probable region will be discovered. It must, however, be confessed that notwithstanding the ease with which the symptoms are recognised, the mechanism of their production is far from clear. Even such a clamant symptom as pain is one of the least understood phenomena.

In the following remarks brief references will be made to the more prominent symptoms, so as to indicate the line of inquiry that may be followed in seeking a solution of this problem. In the present state of our knowledge a complete solution is out of the question.

Pain.—There are certain fundamental laws in the production of pain which must be understood before the mechanism of any given pain is discovered. When pain is felt, in addition to the sensation of pain there is always the sense of locality—that is, the pain is referred to a region more or less precise. This region is always that of the peripheral distribution of a cerebrospinal sensory nerve. When the stimulus adequate to produce pain arises in any part of the sensory neuron, whether in the central nervous system, in the nerve trunk, or at the peripheral distribution in the external body-wall, the part to which the pain is referred is invariably in the field of the peripheral distribution of the nerve. In all cases of pain the question has to be considered: What part of the sensory neuron is being stimulated? In the absence of some obvious cause it is necessary to inquire if the stimulus has affected any other nerves, which may have produced some characteristic reaction. If there is evidence of this, some place must be sought for where a stimulus could at the same time affect these different nerves, provided we are sufficiently familiar with the relation of these nerves, which, in the case of migraine, we unfortunately are not. Another way of applying this method is to observe how pain spreads. It is surprising that the laws governing the radiation of pain have not received the attention they merit. In many cases the manner in which the pain spreads reveals the part where the stimulus producing the pain reaches the sensory nerve. A stimulus affecting any part of a sensory nerve-fibre may not only give rise to pain in the distribution of the nerve-fibres primarily stimulated, but, by spreading, may give rise to pain in the region of distribution of other nerve-fibres. The region into which the pain spreads is determined by the nerve-cells that lie in contact with the cells at the central end of the neuron stimulated. Thus, in toothache the pain may not only be felt in the tooth and its immediate neighbourhood, but may be felt in the cheek or in the side of the head. Here the stimulus from the tooth, in passing back, spreads to other cells in the centre for the fifth nerve, with the result that pain is also felt in the region of their peripheral distribution, in the cheek or the sides of the head. In migraine, the pain, though appearing in the upper division of the fifth nerve, rarely spreads to the lower division. On the other hand it frequently spreads to the back of the head into the region supplied by the upper cervical nerves. It is manifest that the stimulus producing the pain must arise in some part where the fifth cranial and upper cervical nerves can be affected.

It has been surmised that the pain in migraine may be due to stimulation of the periphery of the nerve by a greater flow of blood to the part, and proof of this is adduced in the relief afforded by compressing the temporal artery, or by the application of cold to the scalp. It may, however, be pointed out that this relief is but temporary, and that pains arising from stimulation of the nerve, in any part of its course from the cortex to the periphery, may be relieved by a stimulus applied to the part in which the pain is felt. There may be great heat in the head from dilatation of the arterioles and no pain be felt. In some instances the pain is markedly increased with each pulsation of the heart, and this rather points to a central stimulation.

Symptoms of Stimulation of the Autonomic System of Nerves.—(Vaso-motor and Pilo-motor Effects.)—The blood-vessels and the pilar muscles are under the control of the autonomic system of nerves (Langley). The evidence of their action is seen in the relaxation and contraction of the peripheral vessels and in the waves of goose-skin which pass over the trunk. There is no constant sequence in the evidences of autonomic stimulation. In some people the pain of migraine may be accompanied by blanching of the face and contraction of the carotid and temporal arteries. In others these arteries are dilated so that the head is hot. In some these changes alternate, and while the head is hot and the temporal artery dilated, the hand or foot on the same side may be pale and cold and the

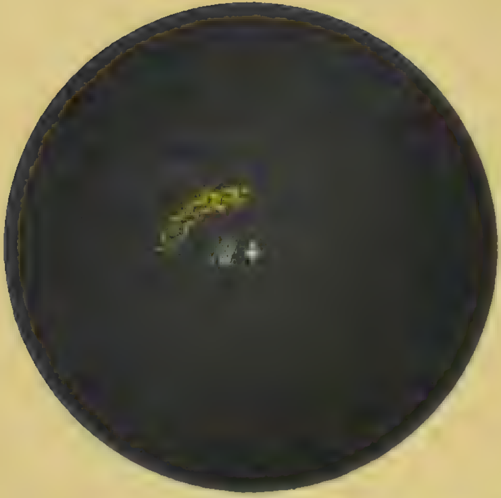

Fig. 1.

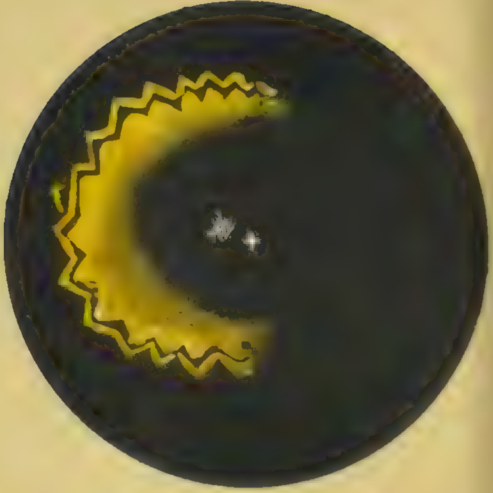

Fig. 2.

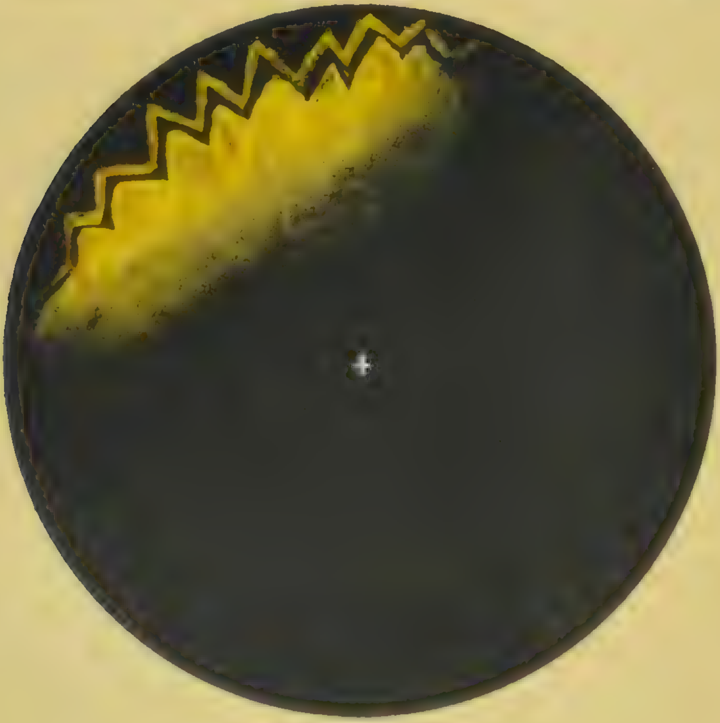

Fig. 3.

PLATE II

Fig. 1.—This and the following two figures represent the ocular phenomena in the field of vision at different stages during an attack of migraine, when the eyes are closed. The central white cross is the point to which the sight was directed; the small shaded area to the left is the position of the blind spot; the zigzag lines and diffused coloration represent the scintillating scotoma. The attack began at 10.45 a.m. with the appearance of the blind spot. A few minutes later the zigzag scintillation appeared as in this figure.

Fig. 2.—Represents the condition at 11 a.m. The blind spot persisted, the zigzag lines had widened out and spread toward the periphery, and there was a diffuse bright coloration within them.

Fig. 3.—Represents the condition at 11.10 a.m. The blind spot had disappeared and the scintillation was limited to the upper and inner part of the field of vision of the right eye. At 11.11 a.m. the scintillation had gone.

Face page 568.

arteries contracted. But it is impossible to decide whether these effects are due to a local stimulation of some high centre or to a spread of the stimulus to different parts of the autonomic system. The slight ptosis and dilatation of the pupil sometimes seen correspond to functional changes in the cilio-spinal region of the cord.

Ocular Phenomena.—The symptoms affecting the sight usually present two very distinct conditions; in one there is a loss of sight limited to a small but well-defined area; in the other the sight is obscured by figures of a bright and scintillating character. In the former there is a loss of sight at or near the centre of vision, so that the object gazed at, as the letters in reading, is not visible. In writing, the letter on which the gaze is fixed may be seen, but the letter immediately before it is invisible. The situation of this scotoma is constant during one attack. The scintillating scotoma on the other hand is ever changing. Beginning some minutes after the appearance of the central scotoma, first as an irregular, narrow, zigzag band of bright light near the centre of vision, it increases in size and in luminosity receding towards the peripheral field of vision. In receding towards the periphery the zigzags become larger and widen out (*vide* Figs. 1, 2, 3, Plate II.). The following account of a typical attack will convey a better impression of this curious visual phenomenon.

While writing at 10.45 a.m. I found that the letters just written became invisible, thus in writing *d* of the word *and*, the letters *an* could no longer be seen. A few minutes after, a slight scintillation appeared to the left of the point of sight. In Fig. 1, Plate II., the white cross in the centre represents the object looked at, the shaded spot represents the blind area, and the coloured zigzag lines the position of the scintillations at their first appearance. By 11 a.m. the circle of scintillating light had become much wider, and the blind area still persisted as in Fig. 2, Plate II. On looking at the face of a person, if the sight were directed to his left eye, his nose or his *right* eye could not be seen. The scintillating light surrounded his head but did not follow the outlines accurately. At five minutes past eleven the circle had extended outwards till it appeared only at the outer part of the field of vision. The blind spot disappeared and the whole of the words that were being written could be seen. At 11.10 a.m. the scintillating light was only seen at a small portion of the periphery of the field of vision at the upper and inner part of the right-eye field, as in Fig. 3. At 11.11 a.m. the scintillations had all gone. At 11.20 a.m. the left ear became warm. At 11.25 a.m. slight headache appeared, referred to the right temporal region. At 12 noon the headache increased and the right side of head became hot, and the right hand and foot cold. At 12.15 p.m. the headache was severe, with a sense of nausea, the right hand warm, the right foot cold. At 12.30 p.m. the headache was slight and the other symptoms had gone. During their presence the scintillations were continually vibrating with very small excursions. The impression during the whole time was that the scintillations and the blind spot affected the right eye only, though it was impossible to see any better when the right eye was covered. During a similar attack in which a large quantity of amyl nitrite was inhaled, I felt very faint and became almost unconscious, but the scintillations persisted. The scintillations came on once when I was about to operate on a case of appendicitis, but in the concentrated attention given to the operation they disappeared, only to return about a quarter of an hour after the completion of the operation.

Coloured drawings of his own spectra in migraine were communicated by Airy to the Royal Society, and were copied in the works of Drs. Liveing and P. W. Latham. Sir W. Gowers also reproduces them with a number of additional drawings (14), and gives a critical analysis of the ocular phenomena in migraine (13, 14). In my own case the hues are not so strikingly iridescent as in the spectra given by Airy, the colours being mostly of a brilliant luminous yellow.

Vertigo.—Giddiness may be the first sign of an attack, or it may occur during an attack. Sometimes while sitting quite still with the head steady the patient may lose the sensation, but on the slightest movement of the head, turning it slightly to one side or the other, the giddiness occurs. Oppenheim attributes the giddiness to cerebellar stimulation, and speaks of "a cerebellar migraine," but this explanation is inadequate, inasmuch as vertigo may result "from affection either of the higher cerebral centres, or of the co-ordinating cerebellar centres, or from affection of any of the afferent paths as from the semicircular canals" (Purves Stewart).

Visceral Reflexes.—Vomiting in migraine is evidently due to cerebral irritation through the centre in the medulla, and being a late symptom in an attack it is probably a secondary result. Sometimes after an attack of migraine patients pass large quantities of pale urine of a low specific gravity. This symptom indicates a stimulation of the floor of the fourth ventricle. The heart's action may be easily affected, the slightest movement of the body increasing the force of the beats and accelerating the rate.

Anaesthesia may be an early symptom and appear in some distant part as in the hand. I have not obtained any records of the exact field of anaesthesia, but in patients who describe it, it does not seem to follow the areas characteristic of a spinal-root affection, but represents more the area of a cortical affection (glove-shaped).

Paralysis.—In rare cases after an attack of migraine there may be left a paralysis of some of the ocular muscles as the internal rectus and superior oblique of one side, giving rise to confused or to double vision. There may even be a slight hemiplegia after an attack of migraine, or transient aphasia if the left cortex be the side stimulated (*vide* p. 605).

Symptoms.—The attacks of migraine vary much in severity not only in individuals, but also in the same individual at different times.

In discussing the pathology the symptoms present in some cases were alluded to. Many patients have no well-defined sequence of symptoms. There is often some premonition; the patient may feel quite well, but become suddenly aware of a blind spot in the field of vision or of peculiar flickering lights of various forms, and in a few hours the headache invariably follows; or the patient wakes in the morning feeling dull or apathetic with a foreboding that an attack is impending. Sometimes this feeling may last a whole day and pass off without further developments. Usually, however, a headache, slight at first but gradually increasing in severity, supervenes and persists with varying severity till the end of the attack. This is sometimes accompanied by a feeling of heat, affecting first, it may be, the ear of the side on which pain is felt, but gradually extending to the side of the head. The temporal artery of the side thus affected may be larger than its fellow on the other side. While the head feels painful and hot, some other parts of the body, as hands or feet, feel cold, and chilly sensations with goose-skin creep over the surface of the trunk. On the other hand the carotid and temporal arteries on the side on which the pain is felt may become small and contracted. Certain areas of skin, as on the hand, may become anaesthetic, so that anything held in the hand may be dropped if the patient's attention be distracted from it. Tenderness of the eyes to bright light, or on continued use, as in reading, is frequent. More rarely there is insensibility of certain areas of the field of vision, with or without the scintillating phenomena already described. Very rarely there is double vision. Gastric disorders are seldom absent; nausea is the most common of them, often going on to violent retching. Not infrequently after one or two violent attacks of vomiting the patient drops off to sleep, and a few hours later wakes well and refreshed; on the other hand, the vomiting may persist at intervals for hours until the patient is quite exhausted and drops into a disturbed, uneasy slumber, awakened at intervals by the bouts of vomiting. Finally he falls asleep, and the attack passes off leaving him prostrate.

This is but a bare outline of the features of an attack of migraine, the symptoms of which vary much in intensity and sequence. They may be so slight as to exhibit little save the premonitory symptoms of mental lethargy. Some cases of hereditary migraine seem never to advance beyond this stage. Again, patients subject to migraine may have temporary attacks of central scotoma, lasting a few hours, or of disturbed accommodation, or of hemianopsia, or of vertigo, without any other symptoms, or accompanied only by a little nausea. In the same way various other phenomena may be present in this minor degree. On the other hand, the attacks may progress with extreme severity; the headache may be so severe as to make the sufferer roll about on the floor, scarcely conscious of what he is doing; drowsiness, amounting almost to stupor, may gradually supervene, until he drops into a fitful sleep, disturbed by paroxysms of pain; complete unconsciousness rarely occurs. There is almost always a certain degree of mental lethargy, and this at times becomes so severe that ideas become confused, and the patient cannot express himself coherently. It would seem that occasionally a patient may become aphasic from affection of the cortical speech-centres; more rarely still there may be a slight and transient hemiparesis. Temporary paralysis of certain of the muscles of the eyeball has occasionally been noted (*vide* p. 605).

Differential Diagnosis.—There is little difficulty in recognising the more characteristic forms of this complaint, as it stands apart with a distinctive individuality. Gross lesions of the brain rarely imitate it, though they may present some phenomena simulating those characteristic of migraine. The occurrence of symptoms for the first time, resembling migraine, in individuals over twenty years of age, should arouse the suspicion of some other cerebral affection. If the symptoms appear in middle age for the first time, however closely they may resemble the symptoms of migraine, they will ultimately be found to be due to some other affection of the brain.

When the symptoms constituting an attack of migraine are imperfectly developed, there may be great difficulty in determining the real nature of the complaint. There is every reason to believe that the attacks can be so modified that the sole manifestation may be some slight transient symptom, such as a central or scintillating scotoma. When, however, in a migrainous subject a headache is present without any of the other characteristic symptoms, there is no reason to consider the headache as an incomplete migrainous attack. Some migrainous subjects are liable to headache without the more characteristic symptoms; and comparison of the conditions in cases in which headache is combined with other symptoms, for instance, ocular phenomena, with those in which ocular symptoms are absent, shews that there is a well-marked distinction between the two groups. There is not usually the same mental depression or apathy, nor the same nausea or objection to food. But most marked of all there is a great difference in the response to treatment. The majority of sufferers from migraine know that when once a real attack starts, with the regular sequence of symptoms, no remedy has any power to stop its onward progress. On the other hand, many of the headaches occurring between the attacks yield to treatment, sometimes very readily. I frequently suffer from headache, and can often cure it by taking 5 or 10 grains of antifebrin. But antifebrin and many other drugs have often been taken to check the attack which starts with ocular symptoms, and invariably without effect. Failure to recognise the distinction between the headache of migraine and other headaches in migrainous subjects evidently explains why the advocates of some special hypothesis find support for their views in the results of treatment. Thus, the reason why G. M. Gould cures 90 per cent of 1000 cases of "migraine" by correcting some error of refraction, and why Drs. Haig and Hare are equally successful by modifying diets in opposite directions, appears to be that they have included under the heading of migraine a number of cases with headache due to other causes.

Treatment.—The frequency and severity with which migraine affects many individuals pursuing sedentary or indoor occupations is in strong contrast to the rarity of the complaint in out-of-door workers, or if a migrainous subject, pursuing a sedentary occupation and suffering from repeated attacks, changes his occupation to one of an active life in the open air, his attacks diminish in number or may cease altogether. Taking these observations as a guide to treatment, the first essential is to get the patient to take active and congenial exercise in the open air. When out-of-door pastimes are impossible, exercise in the open air should be insisted upon as far as the patient's circumstances will permit. Unfortunately the opportunities for these exercises are rarely available for the greatest sufferers from this complaint, such as housewives, dressmakers, or domestic servants; and too often the atmosphere in which they work and sleep is vitiated. It is difficult for them to accumulate that measure of bodily vigour which is necessary to combat the recurring attacks of migraine. They should adopt dietetic and medicinal rules which may favour the development of more robust health. Their diet should be carefully regulated, so as to avoid the errors of feeding to which they are so commonly addicted, and should consist of nutritious and easily assimilated food; this will do much to lessen the frequency of the attacks. They should be impressed with the importance of taking their food in small mouthfuls, which must be thoroughly masticated. Slops, gravies, too much butter and jam should be avoided, and the food should be as dry as possible so as to ensure that the bolus is well moistened by the patient's saliva. These directions alone will often suffice to relieve the gastric disturbance which is so often associated with, and often the cause of, headache. Drugs, such as quinine, iron, nux vomica, will be found beneficial, mainly for their tonic properties. Any tendency to constipation should be corrected.

In migrainous subjects headache is often severe and frequent, without the peculiar and distinctive features of an attack of migraine. In addition to the directions just given, relief is often found from the use of numerous drugs, such as those of the coal-tar series, guarana, cannabis indica, belladonna, the bromides. Dr. G. W. Ross has described a class of patients, with much headache and puffiness under the eyes, who are greatly benefited by calcium salts. For patients in whom the attacks of migraine recur with great frequency, and for those in whom the "status hemicranialis" is established, it is often necessary to employ remedies to give some immediate relief. For this purpose nervine sedatives judiciously administered are useful; of these I have found the bromides, particularly the bromide of ammonium, most beneficial; and in extreme cases the drug should be pushed until slight persistent drowsiness is produced. The patient not only sleeps better, but the attacks often disappear under its use. Then, after a week or two, the employment of tonic medicines and hygienic regulations lead usually to satisfactory results. When the attack recurs with each menstrual period, the administration of the bromide for a week before the anticipated attack often suppresses it. Mr. Whitehead revived the old treatment of so-called counter-irritation by means of a seton put through the skin at the back of the neck. He and others have found this treatment successful.

Dr. F. Hare's suggestion that the success of this method probably depends on the production of an infectious wound has already been mentioned. Many remedies have been suggested to relieve the attack when once it has begun. When the more severe attacks are impending, no remedies appear to be of any avail, either in stopping the attack or in lessening its severity. Sometimes, however, the attacks may be modified by this remedy or that, and the most efficacious of these is found in the coal-tar series of drugs. The effect of these drugs is greatly enhanced if the patient will lie down immediately after taking one of them, especially if a short sleep can be obtained.

Of local applications, cold to the temples while the body is kept warm has sometimes a very soothing if only temporary effect. Pressure applied over the dilated temporal artery sometimes affords relief. Soothing liniments, as of belladonna and aconite, occasionally give ease. The electric current, constant or interrupted, has been found useful by some sufferers. Finally, when the pain is so severe as to be almost unbearable, relief can only be obtained by the judicious use of such exceptional and occasional means as inhalation of chloroform vapour, or the subcutaneous administration of morphine.

JAMES MACKENZIE.

REFERENCES

A full bibliography up to 1873, with a careful record of Symptoms and their Analyses, will be found in Dr. E. Liveing's book *On Megrim and Sick Headache.*

1. Airy, Hubert. "On a Distinct Form of Transient Hemiopia," *Phil. Trans.*, 1870, clx. 247.—2. Allbutt, T. C. "Treatment of Sick Headache," *Brit. Med. Journ.*, 1872, i. 47.—3. Benedikt, M. "Ueber Kopfschmerzen," *Wiener Klinik*, 1898, iii. Heft, p. 1.—4. Brunton, L. *Therapeutics of the Circulation*, 1908, 116.—5. Campbell, H. *Headache and other Morbid Cephalic Sensations*, 1894.—6. Charcot. *Clinique des maladies du système nerveux ; sur un cas de migraine ophthalmoplégique*, 1892, 70.—7. Dobson, T. H. B. "On Migraine," *Brit. Med. Journ.*, 1908, i. 314.—8. Du Bois-Reymond. "Zur Kenntniss der Hemikrania," *Arch. f. Anat. u. Physiol.*, 1860, 461.—9. Eulenburg. Article on "Hemicrania," in von Ziemssen's *Cyclopaedia of Pract. Medicine*, xiv.—10. Franz, S. I. "Physiological Study of a Case of Migraine," *Amer. Journ. Physiol.*, 1907, xix. 14.—11. Gould, G. M. *The Ocular Origin of Migraine*, 1905, xlv. 1296.—12. Gowers, W. R. *The Borderland of Epilepsy*, 1907.—13. *Idem. Lectures on Diseases of the Nervous System* (second series), 1904.—14. *Idem.* "Subjective Visual Sensations," *Trans. Ophth. Soc.*, U.K. (Bowman Lecture), 1895, xv. 1.—15. Haig, A. *Uric Acid as a Factor in the Causation of Disease*, 1908.—16. *Idem. Uric Acid: An Epitome of the Subject*, 1906.—17. Hallion, L. Article "Migraine," *Manuel de médecine*, 1894, tome iv. p. 477.—18. Hare, F. *The Food Factor in Disease*, 1905.—19. Hirtz. Article on "Migraine" in the *Nouveau dictionnaire de médecine*, Paris, 1876.—20. Jones, C. "Migraine and Arthritis," *Lancet*, London, 1898, i. 326.—21. Klein, C. "Bemerkungen zum Wesen der Hemicränie," *Wien. med. Presse*, 1898, xxxix. 412.—22. Langley, J. N. "The Autonomic Nervous System," *Brain*, London, 1903, xxvi. 1.—23. Latham, P. W. *On Nervous or Sick Headache*, 1873.—24. Liveing, E. *On Megrim and Sick Headache*, 1873.—25. Luff, A. P. *Proc. Roy. Soc. Med.*, London, 1909, ii. (Therap. Sect.), 66.—26. Mackenzie, J. *Symptoms and their Interpretation*, 1909.—27. Mendel, E. "Die Migräne," *Deutsche med. Wchnschr.*, 1906, xxxii. 785.—28. Möllendorf. "Ueber Hemikränie," *Virchows Arch.*, 1867, xli. 385.—29. Oppenheim, H. *Diseases of the Nervous System*, translated by E. Mayer, 1904.—30. Ormerod and Spicer. "Recurrent Paralysis of Third Nerve with Migraine," *Brit. Med. Journ.*, 1895, ii. 1561.—31. Putzel, L. Article "Migraine," *Reference Handbook of the Medical Sciences*, iv. 813.—32. Ross, G. W. "On the Relief of Certain Headaches by the Administration of one of the Salts of Calcium,"

Lancet, London, 1906, i. 143.—33. Russell, A. E. "The Pathology and Treatment of Headache," *Clin. Journ.*, London, 1908, xxxii. 136.—34. Sherrington, C. S. Article "Cutaneous Sensations," Schäfer's *Textbook of Physiology*, ii. 920.—35. Snell, S. "Recurrent Third Nerve Paralyses associated with Migraine," *Brit. Med. Journ.*, 1893, ii. 119.—36. Spitzer. *Neurol. Centralbl.*, Leipz., 1901, xx. 735.—37. Starr, M. Allen. *Organic and Functional Nervous Diseases*, 2nd ed., "Migraine," 794.—38. Stekel. "Die moderne Pathologie und Therapie der Migräne," *Wien. med. Wchnschr.*, 1897, xlvii. 46.—39. Stephenson, S. "A Note upon some unusual Forms of Migraine in Children," *Med. Press and Circ.*, 1906, lxxxi. 419.—40. Stewart, Purves. "Migraine," *Hospital*, 1907, xlii. 551; also *Diagnosis of Nervous Diseases*, 2nd edition, 1908.—41. Whitehead, W. "The Surgical Treatment of Migraine," *Brit. Med. Journ.*, 1901, i. 335. 42. Wilks, S. *Lectures on Diseases of the Nervous System*, 1878.

J. M.

IMPEDIMENTS OF SPEECH

By W. S. Colman, M.D., F.R.C.P.

In this country the word "stammering" is used loosely for all forms of speech-defect, but especially as synonymous with stuttering. In German literature, however, "stammering" (Stammeln) is never employed in the sense of stuttering (Stottern), but always to indicate other forms of impediment of speech. In order to avoid confusion, therefore, the word "stammering" will not be used in this article.

I. Stuttering.—**Definition.**—A spasmodic affection of the muscles concerned in articulation, occasioned by erroneous nervous control, leading to a sudden check in the utterance of words, or to a rapid repetition of the literal sound in connexion with which the difficulty arises.

History.—It is only during the last sixty years that stuttering has been looked on as a complaint worthy of the attention of the physician. The first scientific treatment of the subject was contributed by Neil Arnott, in 1829, in an article on "Voice and Speech" in his *Elements of Physics*. In France, about the same time, Columbat recognised the nature of the complaint, and devised appropriate exercises to overcome it; almost simultaneously an accurate account of the subject was published by Schulthess in Zurich. These papers, unfortunately, had but little influence, and the subsequent literature on the subject is full of the most grotesque notions of the pathology and treatment of the disorder. In 1840, under the influence of Dieffenbach, a brief period of surgical enthusiasm occurred, and extensive divisions of the lingual muscles were practised by many surgeons. The enthusiasm subsided in a few years owing to the number of fatal results, and this irrational procedure was entirely abandoned. Now, after many years, investigators are returning to the views and methods of Arnott, Columbat, and Schulthess, and elaborating them. The work of Kussmaul, Gutzmann, and Wyllie has placed the knowledge of the subject on a sound scientific basis.

Mechanism of Normal Articulation.—The mechanism of articulation requires particular consideration as familiarity with it is absolutely necessary for the intelligent treatment of these disorders of speech. The production of articulate speech requires the orderly co-operation of three muscular mechanisms: (i.) the respiratory apparatus for supplying a blast of air; (ii.) the larynx for transforming the blast of air into voice; and (iii.) the musculature of the lips, tongue, and palate, for altering the shape of the passages so as to modify the voice and differentiate sounds into words. Not only must the component muscles of each of these delicate mechanisms act in perfect concert, but there must be absolute co-ordination of each of these mechanisms with one another. It is the difficulty of performing the necessary movements of the tongue and lips which usually obtrudes itself on a stutterer's attention, but, as we shall see, the essential difficulty consists in securing proper co-ordination between the vocal and articulatory mechanisms.

The Physiological Alphabet.—The English alphabet, being both defective and redundant, requires to be reconstructed on a phonetic basis, the various literal sounds being grouped according to the position in the mouth at which the mouth is modified to produce them. Several consonantal sounds (for example, *th*, *ph*, and *sh*) have to be added, and those which are really consonantal diphthongs, such as *q* (*kw*) and *j* (*dzh*), have to be omitted. The most convenient and scientific grouping of the consonants is that devised by Prof. John Wyllie of Edinburgh, which is here reproduced. The section on stuttering in his work on *Disorders of Speech* contains the most thoughtful and suggestive consideration of the subject yet published.

CONSONANTS

(From Wyllie's *Disorders of Speech*)

	Voiceless Oral Consonants.	Voiced Oral Consonants.	Voiced Nasal Resonants.
Labials. (1*st Stop Position.*)	*P* (W)[1]	*B* W	M
Labio-Dentals.	F	V	
Linguo-Dentals.	Th S	Th Z	
Anterior Linguo-Palatals. (2*nd Stop Position.*)	Sh *T* (L)[1]	Zh *D* L R	N
Posterior Linguo-Palatals. (3*rd Stop Position.*)	*K* H or Ch	*G* Y (R)[1]	Ng

---

[1] The voiceless *W* and the voiceless *L* have been given above within brackets, the former being now almost confined to Scotland, and the latter being peculiar to Wales. The burring or uvular *R* is also given within brackets.

The *vowel sounds* are grouped most conveniently according to their phonetic value rather than according to the letters; for in English each letter is pronounced in several different ways. The different sounds are best remembered by the mnemonic given by Pitman:—

Long.—Half pay she thought so poor.
Corresponding Short.—That pen is not one foot.

I is omitted from this list, not being a simple vowel sound, but a diphthong and equivalent to *ah-ee*. *Y* as a vowel is closely allied to *i*, and *w* to *u*. It is a matter of dispute whether *h* should be regarded as a special consonant or as an unmodified expiration. Wyllie's view that it is a guttural allied to the Scottish *ch* is probably correct. It is an occasional cause of stuttering.

For the study of the various forms of speech-defect and for the intelligent direction of treatment it is essential that the medical attendant shall make himself perfectly familiar with the exact position of the lips and tongue requisite for the production of the various vowels and consonants. This can readily be acquired by observing the movements and position of his own tongue, aided by the description in standard text-books of physiology.

The part played by the laryngeal and buccal musculatures respectively in producing the various sounds must not be overlooked. The *vowels* are mainly laryngeal sounds, modification of the sound for each vowel being effected by altering the shape and volume of the buccal cavity as a resonating chamber.

The *consonants* are mainly buccal sounds, but in many there is an important laryngeal element. It will be noticed that in Wyllie's physiological alphabet the consonantal sounds are grouped into "voiced" and "voiceless," a distinction of practical importance in treatment. In the voiceless consonants—such as *p*, *t*, and *k*—there is no vocal element, but a nearly noiseless blast of air. In the case of the voiced consonants —such as *b*, *d*, *g*, and *m*—sound is produced in the larynx each time that the consonant is uttered. This can be easily realised by saying aloud *b-b-b-b*, without employing any vowel, when it will be found impossible to pronounce the consonant without using the voice, and the result may be contrasted with the almost silent production of the corresponding voiceless consonant *p-p-p-p*. This grouping corresponds to the division of the letters by the grammarians into "sharp" and "flat"; the voiceless group containing the sharp consonants, whilst the voiced group includes the flat consonants, the nasals, and of course all the vowels. The distinction between the voiced and voiceless consonants may also be demonstrated by the following plan recommended by Wyllie. Pronounce the word "wonder," in which all the consonants are voiced, in a low monotone. The vocal element produces a continuous murmur, which can be kept on indefinitely by repeating the word. If now the syllable "ful" containing the voiceless letter *f* be added, and the word "wonderful" be pronounced in a similar way, a break will at once occur in the vocalisation of the word at the letter *f*.

Causation.—Little is known with certainty of the causes of this disorder. Direct *hereditary transmission* is only occasionally responsible, but a neurotic family history is usually to be obtained, and, apart from the speech disturbance, the patient often betrays a neurotic tendency. *Sex* appears to have an important effect, the large majority of cases occurring in boys. *Imitation*, as from a stuttering nurse or companion has been traced in some instances. *Adenoid vegetations* are often present in stutterers, and aggravate the trouble by interfering with the proper expansion of the chest.

The affection is rarely congenital, but comes on either in early childhood or at some epoch in a child's life, such as going to school, the period of second dentition, or of puberty. A stutterer who has recovered may relapse in similar circumstances; such as going to a boarding school, or to live abroad, where he has to speak an unfamiliar language. It occasionally first shews itself during convalescence from some acute disease, particularly from diphtheria and measles.

Pathology.—The complaint must be regarded as a functional nervous disorder, without any structural changes, leading to erroneous action of the muscles concerned in speech. Although local conditions of the throat increase the tendency to stutter, they are but contingent direct causes, as is clearly proved by the facility with which almost all stutterers can sing and intone.

Symptoms.—The most characteristic symptom in stuttering is an intermittent inability to emit certain sounds, the disability being noticed only when the patient speaks in a conversational voice, and being rarely present when he sings or intones. The difficulty in articulating is mainly experienced when the difficult consonant occurs as an initial letter, no hesitation being noticed when the same letter occurs in the middle of a word. The actual phenomena observed vary greatly in different individuals. Most frequently the patient has a habit of speaking in a monotonous and rapid fashion, with his chest almost empty. As he is speaking, the flow of words is suddenly interrupted, and he hesitates, with every appearance of violent effort, before pronouncing the difficult consonant. Suddenly the stumbling-block is removed, and the word is pronounced with an explosive jerk, and the subsequent words tumble out rapidly, in a succession of jerks, "like water being poured out of a bottle," until checked by a fresh stutter. This jerky mode of speech often persists in adult life long after all traces of the original stutter have disappeared. The impediment is notoriously worse when the stutterer is nervous or hurried, as when getting a railway ticket; it is also worse when he is in bad health. It is a characteristic of stutterers that they will always persist in their efforts to pronounce the difficult letter, and will never substitute a more easily pronounced synonym, even if it be suggested by a bystander.

Stuttering is very rare with vowels, and is most common with the explosive consonants, *p*, *b*, *t*, *d*, *g*, *k*. Occasionally the stutter at the commencement of a word is induced by nervousness due to the word including some consonant which the patient knows he pronounces with a lisp. For example, several patients stuttered on the initial letter if the word subsequently contained an *r*, which they mispronounced as *w*; and the stutter quite disappeared when, by diligent practice, they had learned to pronounce the *r* with facility. A clergyman became incapacitated for work because he stuttered at the beginning of any word, such as *behold* or *Jehovah*, which contained an *h*, owing to nervousness lest he should omit the aspirate.

In the majority of cases the patient remains silent during his attempt to speak; but occasionally he manages to pronounce the letter, and continues to repeat it, speaking, for instance, of a *b-b-b-ath*, *b-b-b-un*. This, however, is more common in anecdote than in real life. Occasionally the impediment is aggravated by the occurrence of *associated sounds* with the stutter, the patient emitting unpleasant little whoops, grunts, or whimpering sounds during his efforts to speak. Not infrequently these noises are "tricks" that were used, in the first instance, to correct a stutter, and then became grafted upon it. A patient finds, for instance, that by drawing his breath and starting again he can avoid stuttering on the difficult consonant; but in doing this there is great danger of vocalising with the short inspiration, and giving rise to a little whoop or crow (the "drawback phonation" of Wyllie), which is interpolated in his conversation, and may persist long after the stutter has been conquered. In some published cases a meaningless syllable or word, such as "hedera," "nana," etc., is interpolated in the same way, and is very difficult to get rid of.

In most cases the larynx is kept closed while the muscles are acting powerfully, the result being that the patient stands speechless and becomes very red in the face. In other cases the vocal cords are properly adjusted for the production of voice, but the expiratory muscles are not properly brought into play at the proper moment. In a third group vocalisation by the larynx occurs normally, but, owing to spasm of the lingual or labial muscles, articulation is checked. It is important to remember that in the majority of cases the laryngeal muscles are at fault.

In other cases there are *associated movements* which are even more trying to bystanders than the sounds, but are fortunately infrequent. They are usually confined to facial contortions, or to a sudden jerk of the head; but occasionally there are violent gestures of the arms. In one case the whole body became rigid, and was slowly rotated, with the face upturned, the spasm suddenly ceasing when the word was ejaculated. The movements in this case, and in others which have been reported, bore a superficial resemblance to the beginning of an epileptic fit, and confusion is specially apt to arise when the face and neck of the stutterer become congested during his violent expiratory attempts.

Method of Investigation of a Case.—The chest of the patient should be uncovered, and any defect in shape or expansion, such as might occur from adenoids or enlarged tonsils, should be looked for. He should then be made to read aloud, with the chest still uncovered, from some unfamiliar prose book. As he reads, any faults of the respiratory movements should be noted, and any associated movements of the face or limbs observed. Attention should be closely given to the character of the stutter, and a mark should be made under each word at which he hesitates. It will be found that, as a rule, a few initial letters only give trouble, the rest being articulated without effort. The difficult words should be compared, to see if they have some common character, such as the inclusion of some letter which the patient habitually pronounces with a lisp (*vide* p. 573).

Prognosis.—In most of the cases there is a tendency to spontaneous cure. The prognosis is worst in persons of a nervous and sensitive temperament. Recovery is hastened in all cases by systematic intelligent exercise.

Treatment.—There is no royal road to recovery for stutterers. Although the plan of treatment has to be varied for each individual case, the common feature of all is intelligent, regular, long-continued practice. The various mechanical contrivances which have been devised are seldom of the slightest value. These have been largely inspired by the tradition that Demosthenes was a stutterer, and cured himself by shouting with pebbles in his mouth. He certainly had some impediment in his speech, but most probably it was rather of the nature of a lisp. The other means adopted by Demosthenes, however, are better worthy of recall. He retired from society until by patient practice he should have conquered the defect, and he shaved half of his head in order to prevent his return to his wonted haunts until he had succeeded. Surgical operations on the organs of speech have proved useless, and occasionally disastrous, as might have been expected.

The principles underlying treatment are (1) To secure as good chest capacity as possible ; (*a*) by removing any obstruction such as enlarged tonsils or adenoids, should they be present ; (*b*) by suitable breathing exercises and gymnastics. (2) To instruct the patient in the proper use of his voice by regular reading or elocution lessons. When practicable short lessons several times a day are preferable to longer ones less frequently. The best results are naturally to be obtained by a competent scientific teacher (a description which by no means applies to all who profess to cure stuttering). Often, however, this is not feasible, and home tuition under the supervision of the family attendant has to be relied on. It is important that the same person should always give the exercises and lessons after having been instructed by the medical attendant in the principles on which they are based.

In the case of children under eight nothing ambitious or difficult should be attempted. Breathing exercises and simple gymnastics for chest development should be given regularly, and the child should be encouraged at all times of the day to speak slowly and with a full voice. In most cases if this is persevered with for three months it will be found to be sufficient. To get young children to use a full voice without shouting is often difficult. The mother of one of my patients kept a dummy telephone instrument in the nursery and encouraged the child to talk through it to imaginary friends at a distance, and I have found this successful in several instances since.

For older children, in addition to physical exercises, daily reading lessons are essential, and, at any rate at first, should be supervised by the medical attendant. (i.) The chest must be kept well filled with air. (ii.) The patient must speak slowly, with a full resonant voice enunciating all the consonants distinctly. Both these habits are difficult to acquire and much encouragement is necessary. Training is facilitated by having the reading lessons out of doors, or in a large hall, or barn where the voice has to "carry" to reach the instructor, who should stand at some little distance. Even in an ordinary house this "carrying" voice may be obtained by having the boy in a neighbouring room with the doors open, and making him raise his voice, without shouting, so that every syllable can be heard distinctly by the teacher. He must avoid the "sing-song" style into which stutterers often fall, and must try to acquire a deliberate utterance in ordinary conversation. (iii.) When he comes to a word on which he tends to stutter, he must remember that in order to check it he must direct his energies, not to articulation, as he instinctively tries to, but to vocalisation. He should try to raise his voice as if he wanted it to carry farther. If he stutter on one of the "voiced" consonants such as *b*, he must produce the vocal element of the letter. If the consonant is "voiceless," as *p* in *pat*, he must attempt to vocalise the *at*, and he will find little difficulty in prefixing the *p* as the *at* is uttered.

Singing exercises are particularly useful, they give confidence to the patient, and teach him how to vocalise more effectually. Associated movements are best checked by making the patient practise his reading exercises before a mirror, so that he may be aware of them himself, and endeavour to suppress them.

But to obtain very definite improvement considerable time is required. Even after a month of diligent practice the patient may have made little advance, and be inclined to relinquish the struggle. But, as with learning to swim, when once confidence is restored the rest quickly follows. It is therefore a matter of great importance that a boy should be encouraged, and protected from the scoffs of his fellows while under treatment. It is often of great advantage if he can be taken into the house of a medical man or suitable instructor for some weeks.

The general health may require attention, but no drugs have any effect in curing this troublesome complaint.

II. Lisping.—A defect in articulation caused by the indistinct enunciation of certain consonants, or by the substitution of consonants other than the proper one. These defects may be due to deformities in the conformation of the mouth, or to clumsiness in the use of the tongue and lips. It is seen as a normal condition in infants who are learning to speak, but as a rule the difficulty is soon overcome. The most common substitutions in young children are *t* for *k*, *th* for *s*, *f* for *th*, and *r* for *w*. In a few persons the defect persists to adult life and forms a personal characteristic. The most common defects in adults are inability to roll the *r* sound, with a tendency to substitute *w*, and the substitution of *sh* or *th* for *s*. Lisping is often acquired by affected adults, and paraded as evidence of superior breeding. Some years ago *w* was substituted in this way for *r*, and at the present day many persons affect an inability to pronounce the final *ng*, and substitute *n*.

The difficulties met with in infancy are again encountered in learning a foreign language. For a long time certain consonants present very great difficulty, and may never be spoken with facility. The difficulty of the ordinary Frenchman with the English *th*, and of the Englishman with the German gutturals, are cases in point. Not infrequently the difficulty is insuperable, and the peculiarity of pronunciation becomes a national characteristic. During the "Bread and Cheese Riots" in London, in the reign of Richard II., the mob attempted to exterminate the colony of immigrant traders and skilled workmen from the Hanse Towns, by surrounding the streets in which they lived, and putting to death all who pronounced the words "bread and cheese" with an accent. The use of the test-word "Shibboleth" during a struggle between some Jewish tribes affords another example.

Diligent practice alone can overcome the difficulties in the pronunciation of the consonants in our own or in a foreign language; but this may be facilitated by a knowledge of the correct positions of the tongue or lips. The teacher must be familiar with them himself, must shew them to the patient, and then get him to imitate him.

III. IDIOGLOSSIA.—This name was given by Dr. Hale White and Mr. Golding-Bird to a very severe form of lisping, in which the patient's speech is so altered that he seems to be speaking in a language of his own. On carefully analysing the words, however, it is found that many of the letters are not pronounced by the patient, who substitutes for them others, most usually *t*, *d*, or *n*.

A good illustration is the example of a boy under my own care who thus repeated the Lord's Prayer: "Ouë Tahde na ah in edde, anno de Di na, I tidde tah, I du de di on eet a te e edde, te ut te da ouë dade ded, e didde ouë tetedde a ne ahdin to te tetedde adase ut, ne no te tetate, ninne utte enu, to I ah te nini, pouë e dordy, to edde e edde, Amé." Strange as this looks and sounds, it will be found that there is method in it. The patient was unable to pronounce many of the consonants, and substituted for them *t*, *d*, or *n*.

As with stuttering, about two-thirds of the patients are males. They are often bright and quick, and, as they have great facility in communicating their ideas and wishes by facial expression and pantomime, they take little pains themselves to overcome the defect. The peculiarity of speech is usually present from the time when the child begins to speak, and it tends to disappear after the age of eight, if the child realises the inconvenience of the defect, and takes pains to overcome it. In all the cases seen by myself there was a well-marked neurotic family history. One patient had had an attack of left hemiplegia in infancy from which he had completely recovered; another was suffering from valvular disease of the heart. None, except the first mentioned, shewed any indications of organic disease of the brain.

The auditory word-centre appears to be perfectly normal, since the children learn quickly to understand what is said. There seems to be some imperfection in the training of the motor centre in the frontal lobe, which, as Dr. Bastian has shewn, is trained through the medium of the auditory centre; but of the exact nature of the irregularity we are at present ignorant.

On carefully testing the patients with the sounds given in the physiological alphabet (p. 570), the exact sounds which are defective and those which are substituted for them can be demonstrated. The posterior linguo-palatals *k*, *g*, and next to them *f*, *v*, and *r*, are most frequently affected. The labials and linguo-dentals nearly always escape. The average number of consonants which twelve patients failed to pronounce was eight; and several others, which could be pronounced as initials, presented an insuperable difficulty when they occurred as final letters. In rapid automatic speaking they almost all substituted *t* for *d*; the former for voiceless, and the latter for voiced consonants: when speaking slowly and with care, the substituted sounds are more varied. The vowels have not been affected in any of the recorded cases.

Treatment.—It must be remembered that when once the child has become accustomed to express his thoughts by these erroneous sounds, it is as difficult for him to express them in our way as to acquire a foreign language. And just as it is important on going abroad to learn a foreign tongue to keep from the society of one's fellow-countrymen, and to avoid speaking English, so it is most desirable that the patient should be isolated from those who have learned to translate his jargon. He should be sent away from home if possible, and his training undertaken by a relative or some skilled teacher—preferably some one accustomed to instruct deaf-mutes in speaking; or he may be sent to a school for deaf-mutes. The teacher must shew him the exact position of the tongue in the pronunciation of the various consonant sounds, and get him to imitate it. At first only the consonant sounds themselves should be practised, then syllables; but the patient should not try to pronounce words and sentences until considerable facility in the enunciation of the simple sounds has been acquired. In severe cases this takes several months; in slighter cases much less. The progress depends largely on the earnestness with which the patient enters into the exercises, and also, of course, on the patience and capacity of the teacher; but the ultimate results are usually most satisfactory.

W. S. Colman.

REFERENCES

1. Arnott, Neil. *Elements of Physics*, 4th ed., London, 1829. — 2. Barth. "Neuere Ansichte über Stottern, etc.," *Wien. klin. Rundschau*, 1904, xviii. 697, 724. — 3. Becquerel. *Traité du Bégaiement*, Paris, 1843. — 4. Bell, C. *Phil. Trans.*, 1832.—5. Coën. "Das Stotterübel," Stuttgart, 1889.—6. *Idem.* "Neueste Erfahrung über Sprachstörungen," *Wiener Klinik*, 1901, xxvii. 201.—7. Colman, W. S. *Lancet*, London, 1895, i., and 1906, ii. 70.—8. Columbat. *Du Bégaiement*, Paris, 1830.—9. Decroly et Rouma. *Policlin.*, Brux., 1903, xii. 197, 272.—10. Denhardt. *Deutsch. Med.-Ztg.*, 1908, xxix. 93.—11. Dieffenbach. *Die Heilung des Stotterns durch eine neue chirurgische Operation*, Berlin, 1841. — 12. Guillaume. Art. "Bégaiement," *Dict. encycl. des sciences méd.*, Paris, 1868.—13. Gutzmann, H. *Störungen der Sprache, und ihre Heilung*, Berlin, 1893.—14. *Idem.* *Ztschr. f. klin. Med.*, Berlin, 1905, lvii. 385.—15. *Idem.* *Deutsch. med. Wchnschr.*, 1908, xxxii. 612.—16. Hadden. *Journ. Ment. Sc.*, London, 1891, xxxvii. 96.—17. Hunt. *Stuttering and Stammering*, 6th ed., London, 1865.—18. Knopf. *München. med. Wchnschr.*, 1908, lv. 2669. — 19. Kobrak, F. *Med. Klinik*, Berlin, 1908. — 20. Kussmaul. "Disturbances of Speech," von Ziemssen's *Cyclopaedia of Med.* (English Transl.), vol. xiv.—21. Langwill, H. G. *Rev. Neurol. and Psychiat.*, Edin., 1907, v. 259.—22. Makuen. *New York Med. Journ.*, 1906, lxxxiv. 1261.—23. Merkel. *Physiologie der menschl. Sprache*, Leipzig, 1866.—24. Scripture, E. W. *Med. Rec.*, New York, 1907, lxxi. 771, and 1908, lxxii. 480.—25. Steinhardt. *Aerztl. Rundschau*, München, 1907, xvii. 181.—26. Taylor, F. *Med.-Chir. Trans.*, London, 1891, lxxiv. 191. — 27. White, Hale, and Golding-Bird. *Ibid.*, 1891, lxxiv. 181. — 28. Worthington, R. A. *Lancet*, London, 1906, ii. 789.—29. Wyllie. *The Disorders of Speech*, Edinburgh, 1894.

(A full bibliography of the earlier works will be found appended to Gutzmann's Störungen der Sprache.)

W. S. C.

TETANY

Synonyms.—*Heat rheumatism ; Intermittent tetanus ; Essential contracture ; Nurse's contracture ; Epidemic muscular contractions ; Idiopathic contracture ; Intermittent rheumatic contracture; Tetanilla ; Remittent tetanus ; Rheumatic tetanus ; Arthro-gryposis ; Pseudo-tetanus ; Tonic occupation spasm ; Shoemaker's spasm.*

By J. S. Risien Russell, M.D., F.R.C.P.

Tetany is an affection characterised by tonic muscular spasms involving especially the distal portions of the limbs ; it is usually bilateral, and either paroxysmal or continuous. In severe cases the muscles of the trunk, neck, face, eyes, and larynx may become involved in the spasm. The affection occurs in a number of circumstances ; more than one toxic agent appears capable of generating it, and there are many exciting causes which evoke paroxysms of spasm in those so afflicted.

Some writers limit the name "tetany" to cases in which there is marked intermission of the spasm, and apply the name *Arthro-gryposis* to those in which the spasm is more continuous. The carpo-pedal contractions met with in rickety children have been regarded by some as quite distinct from tetany; others look on this as a minor form of tetany. Henoch draws a sharp line of distinction between the disease as met with in children and that which occurs in adults; and Strümpell denies its existence in early childhood. Schlesinger and others, chiefly on etiological considerations, distinguish "true tetany" from "pseudo-tetany." Increased mechanical and electrical excitability of the nerves and muscles are looked on by some as the distinguishing characteristics of the affection, whilst others do not regard these as essential. The truth is, there are different gradations of one and the same affection, from the mild "carpo-pedal contractions" associated with rickets to severe general spasms, which may even simulate tetanus. The absence of certain cardinal symptoms does not necessarily negative the diagnosis.

Spasmodic ergotism is regarded by some as a variety of tetany, others look on it as quite distinct. The degenerative changes met with in the nervous system in chronic ergotism supply the most cogent argument against the identity of the two affections; but the changes may be the secondary results of the prolonged action on the nerve structures of toxic agents such as those responsible for the phenomena of tetany. The affection is included in the present account of tetany.

To Steinheim or Dance is usually given the credit of having first published cases of this affection; but Clarke, in his *Commentaries on some of the most important Diseases of Children*, published in 1815, described cases of spasm of the glottis complicated with rigidity of the extremities, and thus clearly recognised the association of laryngismus stridulus and tetany in children. Moreover, there is evidence in the writings of Hippocrates and of subsequent ancient authorities that the affection was recognised even by them.

The name "tetany," by which the affection is now universally known, originated with Lucien Corvisart in 1852.

Erb, Frankl-Hochwart, and Hoffmann have more especially studied the electrical changes in the nerves, and attention has been directed to their altered mechanical irritability by Chvostek, N. Weiss, and Schultze.

The experimental investigations of Schiff, Horsley, von Eiselsberg, and others, and the surgical procedures of Wölfler, Billroth, and Kocher, revealed an apparent affinity of disease of the thyroid gland to tetany. N. Weiss, from Billroth's clinic, first published the remarkable fact that total extirpation of the thyroid results in tetany. Our knowledge of the pathology of the affection has been still further advanced by chemical investigations. Gley, Vassale, and many others who have adduced striking evidence that tetany is in reality due to removal of the parathyroid bodies, first described in man by Sandström in 1880 (*vide* Vol. IV. Part I., p. 323). MacCallum and Voegtlin have carried us a step farther by shewing that this effect is probably brought about through interference with calcium metabolism (p. 587), and their results have been more or less confirmed by the observations of Parhon and Uréches. Experimental work such as that of Bouveret and Devic has proved the existence of a poison in the contents of the stomach in cases of tetany associated with gastric dilatation.

Etiology.—Tetany is met with at all periods of life, but occurs most frequently in infants and young adults. It is a little more frequent in males than in females; but this is not the case at all ages, for in early childhood the proportion of cases in males is greatly in excess of those in females; after the age of twenty years this state of things is reversed, females being affected more frequently than males.

A peculiar liability to the affection appears to exist in some families, and is supposed to be due to parathyroid insufficiency. The condition occurs in a number of circumstances, of which the following are the chief:—

A. *In Adults.*—(1) Tetany may occur with chronic diarrhoea or obstinate constipation, intestinal worms, peritonitis, appendicitis, and acute dyspepsia. Those conditions are all of minor importance when compared with dilatation of the stomach (*vide* Vol. III. p. 538). An attack is especially apt to occur after the stomach has been washed out; and percussion over the epigastrium, a severe attack of vomiting, or the administration of an enema, may similarly induce the spasm. The gastric dilatation is, as a rule, due to contraction of a chronic ulcer in the pyloric region or duodenum with cicatrisation or carcinoma.

(2) Pregnancy, Lactation, and the Puerperal State.—A considerable proportion of cases of this affection occur in women during lactation, pregnancy, the puerperal state, and also in connexion with menstruation and morbid conditions of the uterus and ovaries. It was in a series of cases in suckling women that Trousseau first described the condition, and this led him to name the affection "*Nurse's contracture.*" When the spasms occur during pregnancy the affection usually shews itself during the latter half. It may occur in several pregnancies of the same person, not necessarily successive; in a case of Maccall's it occurred during the suckling of no fewer than seven children. The spasms are rare during labour, though they sometimes occur then for the first time; and the same may be said of their occurrence during the puerperium. When menstruation becomes re-established tetany is prone to recur at each period, especially during cold weather. Not only is it not common to meet with the affection early in pregnancy, but the onset of pregnancy has resulted in the disappearance of tetany when it had existed previously; in such circumstances it has reappeared at the fourth or fifth month.

In some years many such cases occur, whilst in other years they are much fewer. Frankl-Hochwart found that, out of 52 cases of this form of the affection, 39 occurred from January to April; whilst during the other eight months of the year only 13 were met with.

Tonnellé noticed that young girls with tetany were cured at the onset of the first menses. Some cases of the affection have occurred at the climacterium, and endometritis and carcinoma of the uterus have also been associated with its development.

(3) Thyroidectomy.—Tetany was supposed to follow removal of the thyroid gland in man and in animals, but the weight of evidence is in support of the view that the condition is in reality due to removal of the parathyroid bodies with the thyroid. The time of the year when the operation is performed appears to make no difference, but it is significant that the affection only occurs when the whole gland is removed, for it is in these circumstances that the parathyroid bodies would be likely to be removed as well. Some, however, still contend that tetany may result from extirpation of the thyroid, and the affection has also been met with in association with atrophy of the thyroid and myxoedema. An interesting case of absence of the thyroid in which there was a mixture of myxoedema and tetany has been recorded (Stewart).

(4) Acute Fevers.—The affection is sometimes a sequel of certain acute fevers, such as small-pox, cholera, enteric, scarlet, and rheumatic fevers, measles, acute tonsillitis, diphtheria, influenza, pneumonia, or malaria. Peculiar epidemics of tetany after enteric fever have been met with. The alleged association with rheumatism is open to question, as swelling in the neighbourhood of joints occurs in tetany, quite apart from rheumatism. As regards malaria, also, it is well to remember that in some people tetany spasms only occur at certain times in the day (for example, morning or afternoon); and that rise of temperature and enlargement of the spleen occur in so-called "idiopathic or occupation tetany."

(5) Toxic Conditions. — Jaccoud, Kussmaul, Hoffmann, Imbert-Gourbeyre, and Wick have recorded instances of the association with albuminuria and nephritis. Alcohol has been regarded as a cause, as have chloroform, morphine, and lead. It was the resemblance of the spasm seen in ergotism to that of other forms of tetany which first suggested the possibility that a toxic agent is responsible for such phenomena. Schlesinger reports a case in which, after ergotin injections, tetanoid spasms resulted, and ceased when the ergotin treatment was discontinued. Trousseau's and Chvostek's phenomena were both present.

(6) Epidemics of tetany have been described on the continent of Europe and in America. Some of these have but simulated tetany; notably that recorded by Simon and Regnard in a girls' school at Gentilly in France; one or two indeed were real cases of tetany, the rest were simulations. In the epidemics in certain prisons in Belgium, in 1844, the malady was epidemic cerebrospinal meningitis. Other groups of concurrent cases have been traced to conditions known to be favourable to tetany, such as epidemics of diarrhoea or of some acute fever, or to bad hygienic conditions.

But after the various possible sources of error have been excluded, certain outbreaks remain which have suggested a common source of infection. The epidemics described by N. Weiss and by von Jaksch in young men of the working-classes, sometimes attended with fever, and running a course of two or three weeks, belong to this category. Moreover, the affection is peculiarly apt to occur during certain months, and to vary widely in frequency year by year.

This "idiopathic tetany" is peculiarly frequent in Vienna and Heidelberg; very few women are attacked, and people engaged in certain occupations are peculiarly liable to it. Thus, of 399 cases collected by Frankl-Hochwart, 174 occurred in shoemakers ("shoemaker's spasm"), and 95 in tailors. It is especially prone to occur between the ages of sixteen and twenty-five years, 83 per cent of such cases being met with at this time of life; in Vienna the epidemics occurred chiefly during the months of March and April.

(7) The association of Tetany with other Diseases of the Nervous System.—Epilepsy and tetany may occur together, as may hysteria and tetany. Exophthalmic goitre, syringomyelia, and a unilateral lesion of the spinal cord secondary to influenza, have been found in association with tetany, but such occurrences are exceptional, and perhaps mere coincidences. Osteomalacia has been similarly associated, which is interesting, as a hyperplasia of the parathyroid bodies has been met with in both affections.

(8) Occasional Causes.—Sexual excess, masturbation, fatigue from prolonged muscular effort, trauma, anaemia, emotion, have all been regarded as occasional, but probably only contingent, causes of tetany.

Some of the cases regarded as depending on emotion have been associated with exposure to cold and wet; but emotion commonly evokes a paroxysm of spasm in patients known to be the subjects of tetany. Indeed, when tetany has once occurred in any person it may be subsequently induced by conditions other than that responsible for the primary attack.

Prolonged muscular effort and consequent fatigue may result in the generation of a toxic product of metabolism capable of inducing the manifestations of tetany.

(9) Geographical Distribution. Season and climate.—The malady is very frequent in Vienna and Heidelberg. In the neurological clinics of the Austrian capital about seven-tenths per cent of the cases dealt with are recorded as instances of tetany, whereas in Berlin only one-tenth per cent of such cases are recognised. It has not always been as common in Vienna; and the form of tetany associated with lactation and pregnancy, which was formerly so common in Paris, is now rarely met with in this city. In Moscow, again, where cases occurred rarely before and since, there was an epidemic in one year. In America tetany is comparatively rare; in 1894 Griffith was able to collect only seventy-two records in that country.

It is more common in winter and spring than in summer and autumn; and a curve prepared by Frankl-Hockwart, based on 368 cases of the disease met with in the hospitals of Vienna during a period of fifteen years, shews that the maximum number of cases occur in that city in March and April; and the minimum in July, August, September, and October. Exposure to cold is commonly regarded as a cause of the affection; but it is probable that cold only determines the outbreak of the latent disease.

B. *In Children.*—As in adults, tetany in children is not equally distributed over Europe. In London it is much more common than the adult form; in Leipzig both are rare; in Marseilles, Oddo was able to find only 4 out of 3500 cases seen at the Dispensary for Diseases of Children in three years; and Boral found 24 out of 5443 cases. It is not always equally prevalent year by year; it may spring up almost in epidemic form one year in some place where before it was comparatively rare, as in New York, in 1889, and in Prague in 1890. Of 150 cases in children Frankl-Hochwart found that the majority occurred in February, March, and April. It is almost always children of the lower classes that are affected; a large number of them are usually debilitated, and evidences of rickets are rarely absent. The affection is especially apt to be associated with gastro-intestinal disturbances, either following prolonged exhausting diarrhoea, obstinate constipation, or dilatation of the stomach consequent on errors of feeding, as Comby has insisted; whilst Gaujoux believes that gastro-intestinal disturbances act by producing intoxication; heredity, debility, bad social conditions, and the like, he regards as only disposing factors. Tetany may occur in generalised tuberculosis (Winternitz); and relapsing tetany has been seen in idiopathic dilatation of the colon or Hirschsprung's disease (Langmead). Intestinal entozoa are also met with in some cases. Escherich, who believes that various forms of tetany in children are due to degeneration of the parathyroid bodies, found that of 328 patients examined in the first six months of life the percentage rose from 2 in the first month to 56·2 in the sixth. All that can be said of dentition is that it may favour the occurrence of tetany by augmenting the excitability of the nervous system.

A minor degree of the same affection is met with in rickety children under the title of "carpo-pedal contractions," a condition which we regard as identical with tetany. There is a close relation between tetany and laryngismus stridulus in young children, as was first noted by Clarke and by Kellie in the earliest records we have of tetany in children. Both conditions are closely associated with rickets.

Morbid Anatomy.—The number of fatal cases of tetany is small, and of necropsies still smaller. In the majority of these no changes have been found.

Apart from the sclerosis of the posterior columns of the spinal cord met with in spasmodic ergotism, the changes that have been found are hyperaemia of the dura and pia mater at the base of the brain, subarachnoid effusions and a softened or oedematous condition of the cerebral tissue, and hyperaemia of the pons, medulla, upper cervical cord, and of those peripheral nerves which had seemed more especially affected during life; but the nerves and muscles have been found normal. Capillary haemorrhages, collections of lymphoid cells surrounding the blood-vessels, and small foci of myelitis have all been met with in rare instances. The ganglion-cells of the spinal cord have been found shrunken in appearance and vacuolated; and Bonome and Cervesato found atrophy and rarefaction of the ganglion-cells, atrophy and varicosity of the fibres, augmentation of the neuroglial cells, and formation of spaces resembling the appearance seen in syringomyelia. These changes were most pronounced in the cells of the anterior horns, especially the internal group, which explains the absence of true paralysis and atrophy.

Koster examined a case microscopically in which nothing abnormal was found in the central nervous system, peripheral nerves, or muscles; but the cells of the spinal cord were not examined by the Nissl method.

Since attention has been directed to the parathyroid bodies in tetany, disturbances of circulation, haemorrhages, or simple hyperplasia, have been described in these glands.

Pick, who has found calcification of the small vessels of the grey matter of the cerebellum and cerebrum, notably in the nucleus dentatus and basal ganglia, considers that this condition is to be regarded as the actual cause of the tetany in these cases, and not merely an attendant trophic change.

Pathogeny.—The seat of the morbid process which gives rise to the phenomena of tetany has been the subject of no small amount of speculation. De la Berge believed that the muscles were at fault; Delpech, Hasse, and others have looked to the peripheral nerves for an explanation; Kussmaul, Erb, and Berger to the spinal cord, and other pathologists to the anterior-horn cells: Sir W. Gowers infers that the nerve-cells of the spinal cord and medulla are primarily deranged. Weiss and Bonome and Cervesato also regard the affection as a form of anterior poliomyelitis.

There seems little doubt that the poison responsible for tetany acts on the neurons of the anterior horns of the spinal cord; but, among other considerations, the clinical aspect and issue negative grave organic alteration of the neurons as a rule, though changes, such as have been exceptionally met with, may follow the prolonged action of some toxic substance.

Weiss has suspected derangement of the sympathetic system; and Dr. Hughlings Jackson of the cerebellum.

That the nutrition of the peripheral nerves is altered in tetany is placed beyond question by their behaviour to mechanical and electrical stimuli; it is, however, equally certain that a derangement of the anterior-horn cells of the spinal cord and of the cells of the medulla must coexist therewith.

Evidence of the possible implication of the cerebrum is to be found in the occasional association of hysteria, epilepsy, and insanity with tetany, and by the interosseal flexion of the fingers—as in some forms of cerebral spasm; but this peculiar position of the fingers can be explained equally well by irritation of a given group of nerve-cells in the ventral horn of the cervical cord. There is, however, nothing to support the view of certain older authors, that tetany is purely of cerebral origin.

Numerous hypotheses have from time to time been advanced in explanation of the phenomena. That of its rheumatic origin is no longer tenable; there seems to be no kinship between tetany and rheumatism.

Again, there is a pseudo-tetany of hysterical origin; but it is no less certain that true tetany is quite independent of hysteria.

The dehydration hypothesis of Kussmaul, in view of the tetany of gastric dilatation, has been since discarded even by its originator.

There is much to suggest that tetany is reflex in origin, a view that has been supported by G. Sée, Müller, Berlizheimer, and others; but a careful examination of the facts of tetany, and of toxic processes, makes it probable that reflex causes are only responsible for evoking and not for generating paroxysms of the malady.

Hyperchlorhydria was supposed by Bouveret and Devic to explain the association of gastric ulcer and tetany, and such excess of hydrochloric acid in gastric tetany has been found by Gumprecht and Thiroloix and Du Pasquin; but this cannot be the explanation for all cases, for Blazicek, and others, have recorded cases in which free hydrochloric acid was absent from the stomach contents.

Auto-intoxication by the pent-up, decomposed, and fermenting contents of the dilated stomach is a hypothesis supported by important experimental and other evidence. This hypothesis has been upheld by Bouchard, Gerhardt, Baginsky, and others. Galliard, by an emetic, arrested attacks of tetany in a patient with dilatation of the stomach, and so concluded that tetany is toxic in nature, not reflex. Müller, von Jaksch, and Berlizheimer and Blazicek, and Landsteiner, have all failed to obtain any positive results from experimental inoculation of the gastric juice. Kulnew, Bouveret and Devic, Cassäet and Ferré, Fleiner, Ewald and Jacobson, and Gumprecht, have all isolated toxic products from the stomach contents, and Albu obtained an alkaloid from the urine of Ewald's patient.

Closely linked to these effects in gastric dilatation are the toxic effects of constipation and retention of faeces, which have been studied by Reuss. Albu has also attempted to shew that intestinal parasites produce their effects in association with tetany by the excretion of a substance that has a toxic action on the organism—a view also discussed by Peiper.

There is no proof that the poisons which cause uraemia can produce tetany, but if tetany be due to a poison, the elimination of the toxic body by the kidneys may be fraught with danger to the renal epithelium; if, on the other hand, renal disease be present, failure of the kidneys to eliminate in sufficient quantity any poison which is capable of inducing tetany may possibly determine the attack.

That some toxic product is primarily responsible for the altered conditions of the neurons in what has been called tetanilla strumipriva was first suggested by Sir Victor Horsley. Two views have been held concerning the thyroid in this supposed form of tetany. One is that the gland is capable of neutralising certain toxic products of general metabolism, either by altering their composition or by counteracting their influence by some internal secretion which acts either directly on the neurons so as to immunise them, or indirectly by providing an antitoxin to neutralise the toxin in the blood. The other view is that the thyroid secretes some substance necessary for the nutrition of the organism. Notkin isolated a substance from the thyroid (thyreo-protein) which, when injected into the blood-stream, subcutaneously, or into the peritoneal cavity, produced in animals phenomena like tetany. Fränkel succeeded in separating a basic product from the thyroid (thyreo-antitoxin) which, when administered to animals deprived of their thyroids, can prevent the spasms, but is powerless to avert death. Baumann isolated another substance (thyreo-iodin), but, according to Notkin, this, while capable of neutralising the poisons causing myxoedema and goitre, does not neutralise the poison of tetany; for tremors and convulsions resulted in thyroidectomised dogs in spite of its administration.

Gley and Masoin have found increased toxicity of the urine after thyroidectomy, which toxicity is said to increase considerably at the moment of the epileptiform attack; these observations have, however, been called in question by Godart and Slosse.

There can, however, be no doubt that what was previously ascribed to the thyroid is in reality associated with the glands described by Sandström as parathyroids, and by von Köhn as epithelial bodies, which in man are situated on the posterior surface and lower half of the lateral lobes of the thyroid (*vide* Vol. IV. Part i. p. 323). According to Bielchowsky these glands appear to be essential to life, and the symptom-group of tetany is connected with their disease. It has been definitely proved that results hitherto ascribed to extirpation of the thyroid are in reality due to removal of the parathyroid bodies as well, for when these bodies are removed with as little interference with the thyroid as possible tetany and death of the animals have resulted, whereas when the thyroid is removed without the parathyroids tetany does not result. When only partial removal of the parathyroids has been practised, the resulting tetanic spasms have only been slight and of brief duration. Some have opposed this view, and Caro has attempted to prove that removal of the parathyroids in dogs does not cause tetany. Competent authorities have, however, proved that tetany results in these circumstances in dogs as well as in a number of other animals; and Gley, Pineles, Erdheim, and many other writers, regard the dependence of tetany on operative interference with the parathyroid bodies as proved. In adducing clinical proof in support of this view Glaserfeld points with reason to the striking fact that Kocher only met with tetany in 0·5 per cent of all his cases of thyroidectomy in which the operation was performed by the method of Mikulicz and himself, which avoids injury to the parathyroid bodies.

Vassale is of opinion that the chief function of the parathyroid bodies is the neutralisation of poisons which would otherwise injure the nervous system, so that the tetanic spasms are due to functional insufficiency of the parathyroids, which allows of the action of this supposed toxin. According to Saiz it is now generally accepted that the cause of tetany is insufficiency of the parathyroid bodies, together with other predisposing causes. Deficiency of these bodies may be congenital, familial, or hereditary; they may be deficient in number, altered in structure, or diminished in resistance, although apparently normal in anatomical structure. The epidemic occurrence of tetany is explained by the fact that an unknown toxin, which appears to be connected with special places and seasons, injures the function of the parathyroid bodies. Saiz insists on the importance of distinguishing between the congenital and acquired forms of parathyroid insufficiency, and considers that to the former group belong not only the sporadic cases, but also many instances of family tetany which occur in localities where tetany is endemic. He does not agree with Vassale that the majority of cases of pregnancy tetany are due to congenital deficiency of the parathyroids, for many cases occur without any familial relation, and merely because a specific toxin has injured the parathyroid bodies. W. G. MacCallum and Voegtlin have shewn that there are intimate relations between the metabolism of calcium and the parathyroids, that the excretion of calcium is increased and the calcium content in the blood and brain diminished in experimental tetany, and that the symptoms of tetany can be removed by injections of calcium. They accordingly suppose that parathyroid secretion prevents the formation of certain substances which are capable of entering into combination with calcium, and which thus abstract it from the tissues and cause its excretion. As increased excitability of the nervous system has been shewn to result from deficiency in calcium (Gregor and Sabbatini), they regard the interference with calcium metabolism as the way in which tetany results from removal of the parathyroids.

Von Jaksch's inoculation of rabbits with the blood of tetany gave negative results, as did bacteriological researches. That we have to do with an infectious disease is suggested by its epidemic and endemic character; that fever is often present; that hallucinations occur; that several cases occur simultaneously in the same family or quarter; that it may remain absent from a place for years, and then suddenly spring up again; that in some years several cases occur, in others a few only; and that it occurs most frequently during certain months.

That toxic substances are capable of producing tetany we have proof in its occasional occurrence in ergot, chloroform, morphine, alcohol, carbonic oxide, lead, and spermine poisoning; but as cases of the kind are relatively so rare, it seems that some other etiological factor must also be present. Similarly, in the tetany of acute infectious diseases we cannot tell whether the spasmodic affection depends upon the poison of the primary disease, or upon some other factors.

Halsted found that partial removal of the thyroid in dogs did not result in tetany unless the dogs were pregnant; in which case tetany appeared one or two days before delivery. Again, several cases of tetany in pregnancy have occurred in women whose thyroid glands were atrophied. On the other hand, there are some points of similarity between this and the epidemic or idiopathic form; for instance, it is most common in certain towns, where it occurs most frequently during certain months of the year.

Fatigue, which appears to play so prominent a part in the etiology of some cases of tetany, has been found by Monari to be attended with the presence of highly toxic substances in the urine—an observation which Stolthagen was, however, unable to confirm. In view of the increased metabolism in these circumstances, it is not improbable that such toxic products may appear in the urine, and, moreover, that their accumulation in the system may set up tetany—more especially when the system is further depressed by the cold and wet which so commonly accompany fatigue.

There are those, like Kassowitz and Escherich, who regard infantile tetany as derived directly from rickets, as the rickets and tetany curves rise together, and as phosphorus has a beneficial action in both affections; but probably the true relation between rickets and tetany is that similar conditions favour the occurrence of both (Comby). Tetany is only met with in certain places, whilst rickets is met with all over the world; and cases of tetany have been recorded in which no sign of rickets could be found. Nevertheless, in the light of MacCallum and Voegtlin's observations it is noteworthy that deficiency in calcium is common to both affections.

The balance of evidence thus favours the toxic or chemical origin of tetany, but it would seem that more than one substance may act in this fashion. However this may be, most modern writers, while recognising that the symptom-group of tetany may result from acute infections, pregnancy, and various other causes, consider that insufficiency of the parathyroid bodies, congenital or acquired, is the essential underlying condition that permits of the symptoms; but whether they can do so by a direct action on the neurons, or whether they can only act indirectly by damaging the parathyroids, must be left for future research to determine.

Symptoms.—*Muscular Spasm.*—The attack of muscular spasm is preceded, as a rule, by subjective sensory sensations, such as "tingling," "numbness," "burning," which may persist for a few hours or days before the objective motor phenomena shew themselves. In rare cases headache, malaise, vomiting, and pain in the back precede the spasms; and in others there are no premonitory symptoms. The spasms are usually symmetrical, and begin, as a rule, in both hands; or, if in one, the other becomes involved before any other part of the body. The feet are next affected, or, in rarer instances, the spasm begins in the feet, and is either limited to them, or spreads later to the upper limbs. In some instances the two limbs on the same side of the body are affected, the opposite limbs being free; in other cases one upper or one lower limb is affected alone; on still rarer occasions there is an alternation, the upper limb being involved on one side and the lower on the other. Rarely the spasm begins in the trunk muscles; still more rarely it begins in the face. The part affected becomes stiff and cramped, and so rigidly fixed that voluntary movement of it becomes impossible.

Most commonly the thumb is adducted and in close contact with the index finger, or flexed into the palm beneath the index and other fingers, which are usually flexed at the metacarpo-phalangeal joints, and extended at the phalangeal articulations; the outer and inner borders of the hand are approximated by spasm of the thenar and hypothenar muscles, so that the palm is hollowed and the fingers are drawn together, mainly by spasm of the interossei; thus, from the cone-like shape, it has been called "the accoucheur's hand." Occasionally the fingers are flexed at all their joints, by spasm of the long flexors, so that the fist is tightly closed; and so powerful is the spasm sometimes that the nails may be driven into the skin, and sloughs produced. The conical hand and the fist are both met with at all ages, and indeed both may be met with in the same individual at the same time. Again, if the chief spasm is in the interossei and lumbricales, the hand may assume the bird-claw position ("main en griffe"). In very rare instances the fingers have been extended at all their joints, and widely separated from each other. The wrist is as a rule slightly flexed, and the hand may be inclined to the ulnar side; but in rare instances the wrist has been extended. The elbow, if affected, is usually flexed, the forearm strongly pronated and folded across the chest, and the upper arm closely adducted to the side of the body.

The toes are powerfully flexed and adducted, the great toe being in some cases forcibly adducted below the others. The feet are arched so that the dorsum is convex while the sole is concave; and the foot is extended at the ankle and inverted, assuming the position of talipes equino-varus, the result of spasm of the sural muscles. Occasionally the foot has been dorsi-flexed at the ankle instead of being extended, a position brought about by spasm of the anterior tibial group of muscles. Sometimes the foot is rendered immobile in the normal position.

The knees are usually extended, but in rare instances have been flexed; and the thighs are sometimes adducted so that the limbs come together and even cross each other: in rare cases they are flexed on the abdomen.

When the trunk muscles are included in the spasm, those on the ventral aspect of the body are usually affected; the head is bent forward by spasm of the anterior neck muscles, the sterno-mastoids stand out, and the chin may touch the sternum. Very rarely one or other sterno-mastoid is affected without its fellow, in which case the attitude is that of torticollis. The pectoralis major is especially prominent on both sides. The abdominal muscles become rigid, and the recti stand out under the skin like two cords. Spasmodic retention of urine occurs in some cases. Trousseau supposed that spasm of the sphincter of the bladder is the cause of it. Oddo observed a case in which this symptom was met with at the outset.

When in addition to the neck, thoracic, and abdominal muscles, the diaphragm also is involved in spasm, respiration becomes gravely affected.

Less commonly the bulk of the spasm is in the dorsal muscles, the head is drawn back and the back arched in opisthotonos. The absence of this symptom in tetany was at one time regarded as a valuable diagnostic sign between this condition and tetanus, but that it is sometimes met with in the former condition is placed beyond question by the number of instances of the disease, otherwise typical, which have presented this symptom. Like spasm of the masseters, it has been regarded as one of the points in favour of trismus neonatorum being a form of tetanus rather than a form of tetany. But many cases of undoubted tetany have presented this symptom as a late phenomenon. When trismus occurs it is usually in cases of widespread spasm. Facial spasm produces a peculiar ghastly grin, the eyes being half closed and the angles of the mouth drawn outward. The spasm does not confine itself to the territory of the motor portion of the fifth and the seventh cranial nerves, for the ocular muscles may be involved, with the results of convergent or divergent strabismus, conjugate deviation, and even immobility of the pupils.

The tongue may participate in the spasm, and the consequent stiffness may cause difficulty of articulation. Swallowing may be difficult, owing to spasm of the muscles of the pharynx, and articulation from spasm of the laryngeal muscles. Noisy breathing and stridor naturally accompany this condition, and laryngoscopic examination during the spasm reveals that on attempts at phonation the vocal cords do not approximate, nor do they separate on deep inspiration.

The degree of spasm and the extent of its distribution vary considerably; it may be but slight, involving a single muscle or group of muscles, or it may be widespread. The hands may be affected alone, or the hands and feet; in other cases the whole of the limbs are involved; in others again the trunk and neck muscles are invaded by the spasm. Usually when there is further spread to the muscles of the face the spasms are severe, but, on the other hand, facial spasm has been met with in mild attacks. In cases of still more widespread spasm, when the masseters, the muscles of the tongue, pharynx, and larynx become involved, and the breathing may become exceedingly difficult, the patient becomes cyanosed, and for a short time consciousness may be lost. These conditions may indeed result from fixation of the thorax alone, the muscles of the larynx being free. Participation of the muscles of the trunk and face in cases of less intensity is much more common in children than in adults.

The spasms are usually paroxysmal. Each paroxysm lasts from a few minutes to a few days; the muscular contracture then slowly passes off, but in a few hours or days recurs. In some cases the spasm never wholly passes off in the intervals, but persists in lessened degree in the arms, and less frequently in the legs. Even in cases in which the spasm appears to pass off, a feeling of stiffness remains in the muscles concerned. The paroxysmal character of the affection is less evident in cases of moderate but persistent, or but slightly remittent, spasm. If we may make three classes of intermittent, remittent, and continuous spasm, yet all sorts of intermediate grades are seen. The spasm is never so severe in the continuous form as in the paroxysmal. All these forms are met with at the various periods of life, though, as a rule, the remittent and intermittent forms are more common in adults, and the continuous in children.

The contracted muscles are very hard and firm, and considerable force has often to be employed in order to overcome the spasm; a procedure which, as a rule, causes the patient pain, but is sometimes attended with relief. As soon as the force is withdrawn the muscles generally become shortened again in spasm; so that if, for instance, the fingers and thumb are straightened, they return to their former condition of flexion as soon as they are released. Further, if in the intervals between paroxysms, when the spasm of the muscles is moderate, this be forcibly overcome, a much more powerful after-spasm results, which may become widespread and paroxysmal. Fibrillar contractions are sometimes seen in the muscles; and in rare instances clonic spasm has been the initial phenomenon of an attack.

The spasm of tetany may persist during sleep, though, as a rule, it lessens; attacks may occur for the first time during sleep. Not uncommonly minor degrees of tetany occur during sleep, especially in weakly women. The condition is bilateral, and the fingers are either in the characteristic position, or in the less usual one in which there is flexion at all the joints. The spasm is never severe, and is preceded by tingling which persists after the spasm has passed off. The affected parts feel stiff, and sometimes arrest of movement may last for a few minutes only, or may continue for an hour or two. Some patients of this kind have also slight attacks of tetany during the day.

Movement is hampered in proportion to the severity of the paroxysm; thus, in severe spasm all voluntary movement may be impossible, or it may cause only a certain amount of difficulty and awkwardness. A feeling of stiffness, or a bruised feeling with tenderness on pressure, often persists after all obvious spasm of the muscles has passed off. Sometimes a certain amount of muscular paresis follows the paroxysm of spasm, or alternates with it.

During the intervals muscular effort may evoke an attack; lavage of a dilated stomach often has a similar result; sometimes the first spasm follows this procedure. Percussion of the abdomen may induce an attack; and Sir W. Gowers mentions a case of perforative peritonitis attended with a paroxysm of tetany. The phenomenon known as "Trousseau's symptom" is dependent on this power of evoking a paroxysm at will. Compression of the affected limb so as to impede the venous or arterial circulation in it, or pressure on the chief nerve-trunks of the limb, suffice to bring on the spasm in a large proportion of cases of tetany. Kussmaul found that pressure on the artery rather than on the nerve produces the phenomenon; but, as Sir W. Gowers shews, the close neighbourhood of the nerves and the vessels makes it improbable that one can be effectually compressed without the other; slight pressure on the nerve, such as might well result when the vessel is being compressed, is more effective than severer pressure. It is, however, quite clear from a large amount of evidence that pressure on either the artery or nerve will evoke the spasm in a few minutes. When present the phenomenon is a valuable diagnostic sign, but it may be absent in some otherwise typical cases of tetany. The phenomenon has generally been regarded as a direct effect on the muscles, but Schlesinger considers it a reflex, and has recently called attention to a new sign in tetany, his interpretation of which gives support to this hypothesis. Schlesinger finds that if the lower limb be kept extended at the knee and then strongly flexed at the hips, in a very short time a spasm results in the extensors of the knee. As the sciatic and not the anterior crural nerve is stretched and thus stimulated by the procedure, and as there is no interference with the circulation as proved by the persistence of pulsation in the vessels at the ankle, he concludes that the phenomenon is reflexly induced.

Mechanical Irritability of the Nerves and Muscles.—There is a remarkable increase in the mechanical excitability of the motor nerves and muscles of the affected parts, commonly known as "Chvostek's symptom." The phenomenon can be demonstrated in the intervals between the paroxysms when percussion of the motor nerve to a muscle especially, or of the muscle itself, results in a well-marked contraction of the latter. This exalted excitability of motor nerves and muscles may be seen in any part of the body, but has been most frequently met with in the face where a single tap on the trunk of the facial nerve in the region of the stylo-mastoid foramen, or on the pes anserinus, often causes a momentary contraction of all the muscles supplied by the nerve. This so-called "facial phenomenon" may also be evoked by a tap on the cheek just external to the angle of the mouth, or, in fact, on any of the branches of the nerve; and Schultze shewed that the contraction could be elicited by passing the finger-nail rapidly down the side of the face from the temple to the cheek and jaw. The importance of this phenomenon in tetany was clearly recognised by Sir T. Barlow, on whose suggestion Dr. Abercrombie undertook an investigation which resulted in his finding the condition invariably present in tetany of young children, in whom he could find no similar increased excitability to mechanical stimulation in the nerves of the limbs.

Mechanical excitability of muscles is less easy to demonstrate, but is characterised by idio-muscular contraction following percussion, pinching or traction of the muscle-fibres.

Electrical Irritability of Nerves and Muscles.—That a similar greatly increased excitability of the nerves exists to both the interrupted and constant electrical currents has been pointed out by Erb, who also called attention to the peculiar altered method of response to the constant current met with in tetany. It consists in a more ready response with the positive pole (anode) when the current is closed or opened, than with the negative pole (cathode) when the current is closed. The contraction which results is not a momentary one, as in health, but is prolonged, so that tetanus results; and in no other condition has anodal opening tetanus been met with in man. The order of response to galvanism is therefore:—

(i.) A.C.C., (ii.) A.O.C., (iii.) K.C.C. ; or (i.) A.O.C., (ii.) A.C.C., (iii.) K.C.C., A.O.T. This is not always the case, however, for, though the excitability to galvanism is increased, the cathode may still elicit the first response. Again, Thomas has shewn that with increased excitability to faradism and galvanism, 2 milliamperes through the nerve, with the cathode as the stimulating pole, tetanus resulted and did not subside until the current was broken, when it ceased instantly. When the anode was substituted for the cathode no such tetanus resulted, even with 7 ma. ; and when fibrillar twitchings existed before excitation they became less marked as the current was increased. Peters finds that minimal galvanic currents of 3 or 4 milliamperes or less are capable of causing the arms and legs to jump each time the current is closed, if the cathode be placed on the cervical or thoracic spine and the anode on the sternum. The phenomenon is absent in mild cases, and when present is abolished by lumbar puncture. Increased excitability to galvanism was met with almost constantly by Frankl-Hochwart, who has never met with an acute case in which this was not evident; but increased excitability to faradism was much less frequently observed. All nerves are not equally prone to this exalted state of irritability as was pointed out by Erb; the ulnar is peculiarly liable to shew it, and electrical excitation of the ulnar nerve in a healthy person exactly reproduces the most common position of the hands and forearms in the spasm of tetany. The facial nerve so rarely presents any increased electrical excitability, that some writers have stated that it forms an exception to the rule and is never affected; in some cases, however, though rarely, it presents this exaltation of excitability. It is not a little curious that a nerve which so commonly shews such a remarkable increase of irritability to mechanical stimulation should so rarely shew an increase of electrical excitability.

Sensory Phenomena.—Pain is a prominent symptom in severe cases; the amount of suffering is sometimes intense, whether in children or adults. The pain is cramp-like in character and extends up the limbs in the course of the chief nerves. Even when the spasm of the muscles appears to be continuous the pain may be paroxysmal. In slight cases pain may be absent, or moderate in degree; it may be deep-seated or superficial, the former being either spontaneous or provoked by movement. The spontaneous, deep-seated pain is cramp-like, and occurs at the moment the paroxysms of contracture come on; this is the most characteristic pain, and only occurs when the contractures are sufficiently intense. Pains in the joints are not constant; they may accompany the swelling, heat, and redness of the periarticular tissues. The pain may result from active or passive movements of the parts in spasm: the pains may occur in the joints also. Direct pressure on muscles, nerves, or joints of affected parts often produces pain, but less intense than that produced by movement. The superficial pains are almost exclusively limited to the neighbourhood of the fingers and dorsum of the hand, or, more rarely, under the arch of the foot. They consist in formications, burnings, and so forth. Subjective sensations, such as heat and cold, pins and needles, and similar paraesthesias, resembling those which may be the forerunners of the muscular spasm, may persist during the intervals, and may be attended by a moderate amount of blunting of sensibility to light touches, to painful stimuli, or to both.

Hoffmann has described a greatly increased irritability to mechanical stimulation in the sensory nerves, similar to that met with in the motor; the slightest pressure on them resulting in corresponding paraesthesias. The same observer has shewn in them an increased excitability to mechanical and electrical stimulation; and that the anode more readily produces a sensation than does the cathode. The increased irritability of motor and sensory nerves to mechanical and electrical exaltation is greatest when the affection is at its height; but usually persists for some weeks after the muscular spasm, and when "Trousseau's phenomenon" can no longer be elicited. Indeed it tells us whether the attack of tetany is really at an end or not.

Muscular sense is said by some writers not to be impaired; others have found it affected. In some cases the size and consistence of objects cannot be determined; and in walking the feet may have a false sensation of treading on a soft carpet.

Little that calls for special comment has been noted in connexion with the special senses.

Reflexes.—The knee-jerk is normal in some cases, exaggerated in others; in some again it is greatly diminished, or unobtainable. The last condition is probably often due to muscular spasm rather than to a central or peripheral defect in the reflex arc. In some chronic cases of spasmodic ergotism, however, in which the knee-jerks were absent, the posterior columns of the spinal cord proved to be sclerosed.

Vasomotor and Trophic Phenomena.—Oedema of the dorsum of the hands and feet is the most characteristic of the vasomotor and trophic disturbances in tetany, and is due to an infiltration of the cellular tissue. It is a solid oedema, and is often accompanied by diffuse redness and heat of the skin over the region. The oedema persists between the paroxysms, and disappears with the attack of tetany. It is nearly always bilateral, but it often predominates on the side of the severer contractures. Sometimes the joints are swollen, or the sheaths of the extensor tendons. This oedema is much more frequent and intense in children than in adults. General anasarca, with or without ascites, has been met with, and that without albuminuria.

Other vasomotor phenomena consist in flushing of the face, injection of the eyes, and redness of the ears. Evidence of similar vaso-dilatation within the cranial cavity is found in the giddiness, loss of consciousness, subjective sensations in the ears, and fleeting affections of vision. Vaso-dilatation also occurs in the extremities, redness of the hands being especially frequent, accompanied by elevated temperature, and sometimes by abundant sweating. Sweating sometimes terminates an attack of spasm so grave that the patient's life seems in danger. In other cases the skin is unnaturally dry. These phenomena disappear with the paroxysm. Cyanosis of the extremities occurs sometimes and likewise disappears with the paroxysm of contraction. Gangrene has been met with in cachectic children. Westphal has recorded two interesting cases of hysterical pseudo-tetany in which there were vasomotor disturbances and arterial phenomena identical with those which occur in intermittent claudication, although the vessel-walls were healthy.

Eruptions of toxic characters, erythematous, scarlatiniform, morbilliform, and urticarial, may occur.

Mental Symptoms.—The mental faculties remain undisturbed as a rule, but both Frankl-Hochwart and Kraepelin have called attention to the occasional occurrence of mental symptoms in association with tetany; these have included varying degrees of disturbance from transitory delirium with confusion up to dementia.

General Symptoms.—The pulse is accelerated, breathing may be rapid, and the temperature, though usually normal, may be raised in severe and acute cases, but rarely over 101° F.

The urine is normal in the majority of cases, but polyuria has been met with by Mader, Hoffmann, Neusser, and Frankl-Hochwart; Oddo and Sarles have demonstrated a remarkable increase of phosphates in the urine of children suffering from tetany. Albuminuria is rarely met with, but more often in adults than in children; tetany, as a rule, is much more severe in adults, and elimination of large quantities of its toxic substances may injure the renal epithelium. Imbert-Gourbeyre, Delpech, Rabond, Löb, Dreifus-Brisac, Laprevotte, Bouveret and Devic, and Miller, among others, have met with this sequence; and Bouveret and Cervesato found albumin in the urine in two cases of infantile tetany with fever, but without renal lesion. Loos found only a trace present in two cases out of seventy-two children affected with tetany. In some cases the renal affection had existed before, whilst in others it shewed itself for the first time during the attack. In experimental tetany the amount of calcium excreted has been found to be increased (MacCallum and Voegtlin). Mucinuria is met with in the variety of tetany which results from ablation of the thyroid. Glycosuria has also been observed, but acetonuria and diaceturia are met with more frequently than glycosuria. Indicanuria is much more frequent and intense in children than in adults. So common is it in children, that it may be regarded as one of the symptoms of infantile tetany, which is intimately associated with gastro-intestinal disturbance. Certain toxic bodies, of the nature of pepto-toxins, have been found in the urine by Ewald.

Diagnosis.—Characteristic cases rarely present any difficulties in diagnosis; the character of the spasms, their onset in the extremities of the limbs, their restriction to certain groups of muscles, their symmetry, their intermission and reproduction at will, by pressure on the main nerve-trunks or vessels of an affected limb, form an unmistakable clinical picture. If, moreover, mechanical and electrical excitability of motor and sensory nerves be increased with the peculiar altered electrical response to galvanism, it is difficult to see how a mistake in diagnosis could be made.

It is otherwise, however, with many of the unusual or aberrant cases of the affection, which may be either so slight or so severe as to make a diagnosis difficult. In the slighter varieties, or earlier stages, the sensory phenomena (such as tingling and numbness), may be so prominent as to attract the patient's attention almost exclusively; and a history of any muscular spasm can only be elicited on careful inquiry. Further, these are the very cases in which the additional aids to diagnosis, such as the Trousseau phenomenon, the increased mechanical and electrical irritability of nerves, and the altered response to galvanism, may be absent. According to Chvostek, "Trousseau's phenomenon" is the least reliable, for it is frequently absent, disappears more rapidly than the others, and is, furthermore, not pathognomonic.

It is in such slight or incipient cases that valuable assistance in diagnosis may be obtained by a careful search into the causes, such as lactation, pregnancy, dilatation of the stomach, diarrhoea, or fatigue.

Neuritis.—When there are marked sensory symptoms with cutaneous hyperaesthesia and the reflexes are absent, diagnosis may be difficult unless the special indications of tetany be well marked.

Tetanus.—In unusually severe attacks in which, in addition to the muscles of the extremities, those of the trunk and face, including the masseters, are in a state of spasm, the diagnosis sometimes requires great care. These cases have been mistaken for tetanus, and conversely tetanus for tetany. In tetany the fingers and hands are the parts usually earliest and most affected, whereas in tetanus they escape; spasm of the masseters, if present in tetany, is, as a rule, a late phenomenon, whilst it is the earliest manifestation of tetanus; in tetany the spasm intermits, and the usual posture of the hands or feet is characteristic. The flexors, pronators, adductors, and internal rotators are in action in tetany; in tetanus the extensors, supinators, abductors, and external rotators.

Tetanus neonatorum occurs during the first days of life. There is a definite source of infection in the umbilical wound, and trismus is a prominent feature of the disease, the limbs only becoming affected when the spasm becomes generalised, and after the muscles of the neck and pharynx have been involved; the inverse of what obtains in tetany.

In *spinal meningitis* fever is more commonly present, and severe pain in the back; pain, if present in the extremities, radiates along the course of the sensory nerves, and is not, like the pain of tetany, dependent on the muscular spasms for its production, or limited to the muscles actually in spasm; the muscular spasms do not remit. "Trousseau's phenomenon" cannot be obtained, and the nerves shew no increased mechanical and electrical excitability. The tingling and slighter pains of tetany, which may be present apart from any obvious muscular spasm, closely resemble the milder subjective sensations associated with spinal meningitis. In meningitis the parts affected by spasm are prone to become paralysed; and among other evidences of spread of the inflammation to the spinal cord are the affection of the sphincters and the liability to the formation of bed-sores. Moreover, in lumbar puncture we have a ready means of distinguishing between tetany and all forms of meningitis, including the tuberculous variety, which has next to be considered.

Tuberculous Meningitis.—Tetany occurs in children chiefly between the ages of one and three years; tuberculous meningitis chiefly between three and five years. The long premonitory stage of tuberculous meningitis is wanting in tetany. Spasm, if present, is rarely prominent in meningitis, does not long resemble that of tetany, and is not repeated with the same constancy or in the same order. Headache and vomiting are much more intense, and pain is much more in the skin than in the muscles. Paralysis of cranial nerves, inequality of pupils, or the presence of optic neuritis, may leave no room for doubt. The pulse is quick in tetany, and presents neither the slowness nor the irregularity met with in tuberculous meningitis.

Cerebral Congestion.—Though accompanied by spasm, which may be transitory and repeated, cerebral congestion is preceded by much more pronounced premonitory cerebral symptoms than occur in tetany, among which may be mentioned headache, vertigo, and vomiting. The symptoms which accompany cerebral congestion do not present the localisation or evolution characteristic of tetany, nor is the causation the same.

Epilepsy, in which the spasm is tonic and bilateral, may resemble tetany; but consciousness is lost, the paroxysms are exceedingly short, and there is complete relaxation of the spasm in the intervals. Jacksonian epilepsy has been mistaken for tetany, but the conditions are widely different; in the former, tonic and clonic convulsions begin in one upper or lower extremity, or on one side of the face, and not on both sides at the same time; if they become general, consciousness is lost. When the spasm passes off, the part that was affected is left more or less enfeebled for a variable time. Headache, vomiting, and optic neuritis would be conclusive. It must not be forgotten, however, that epilepsy and tetany may be associated in the same individual, and it is said that in the absence of other manifestations of tetany the facial symptom has been met with in epilepsy.

Contracture, the result of *organic brain disease*, is often unilateral, and usually permanent; there is paralysis as well as spasm, and usually other evidences of cerebral defects.

Little's Disease and Infantile Cerebral Diplegias.—Tetany limited to the lower limbs in children may simulate these spastic paraplegias; in the upper limbs the latter may be confounded with generalised tetany. The great point of diagnosis is that the contraction in these conditions is permanent and continuous; in tetany it is essentially paroxysmal. Evidence of mental defect and the existence of ankle-clonus and the extensor type of plantar reflex may further establish the diagnosis of cerebral diplegia.

Hysterical contracture sometimes resembles the spasm of tetany very closely, and may be difficult to distinguish. It is generally unilateral, whilst tetany is but rarely so; yet certain cases of bilateral spasm have been designated "hysterical tetany." Other indications of the existence of hysteria are usually to be found, including the readiness with which the patient responds to suggestion, whilst "Trousseau's phenomenon" and increased electrical irritability of nerves are absent; the latter, according to Schlesinger and Curschmann, being the most important point in the diagnosis, for Erb's electrical test is never positive in hysterical pseudo-tetany, whereas both Chvostek's and Trousseau's phenomena may be. Chvostek (18) does not, however, admit that even Erb's sign is to be relied on, as excessive electrical excitability may be met with in hysteria.

The chances of confusing an attack of tetany with one of *acute rheumatism* must be very slight. Swelling and redness of the hands or feet in tetany may suggest rheumatism, but the peculiar position of the hands or feet, and the other characteristic signs of tetany, render the diagnosis of this condition clear, whilst their absence renders the alternative diagnosis of rheumatism equally certain.

Prognosis.—The prognosis in tetany is as a rule regarded as favourable, except in thyroidectomy or dilatation of the stomach. Frankl-Hochwart, however, points out that this view has been arrived at without taking relapses into consideration, and when so doing he found that four-fifths of the cases with which he was able to keep in touch proved to be permanent sufferers, so that the prognosis as regards recovery is considerably worse than is usually supposed. Out of 40 cases of gastric tetany 31, or 77·5 per cent, proved fatal (Albu). On the other hand, Mr. Moynihan and others have shewn that gastric tetany is neither the rare nor the fatal affection it has been supposed to be, and that cures can be effected by gastro-enterostomy.

Of other forms of tetany, that met with in very young children, whose power of resistance is small, must be regarded as serious, especially when spasm of the glottis or general convulsions supervene. As a rule, these patients have been much exhausted by constant diarrhoea, which often persists, and is directly responsible for a fatal issue. Similarly, adults who are cachectic, or whose strength has been much reduced, may give cause for apprehension, death from exhaustion having resulted from the violence of the muscular spasm.

As a rule, adults succumb to the progressive character of the disease and the intensity of the poison; children more commonly die of asphyxia consequent on spasm of the glottis or respiratory muscles. Pneumonia may be the cause of death in some cases owing to interference with respiration resulting in congestion of the lungs.

It is by no means easy to forecast the duration of an attack of tetany. Mild attacks of continuous tetany are usually of short duration, and even where the affection is more severe; but if the attack is prolonged over several days the duration of the disease is likely to be comparatively short. When tetany occurs during pregnancy, it may usually be predicted, without fear of error, that the spasms will continue until delivery. If the patient has had tetany in the course of any previous pregnancy, its behaviour then is a fairly good guide in subsequent attacks. Those who have been attacked during pregnancy are liable to future attacks in subsequent pregnancies. Similarly, in other varieties of tetany, future attacks are prone to appear if the patient be exposed to one or other of the influences known to be intimately associated with the affection. It may safely be said that so long as the increased irritability of the nerves is present, the paroxysms of spasm may recur.

Treatment.—The only rational treatment of an affection of the kind is that which attempts to influence the fundamental condition which underlies the symptoms, so that it is to organo-therapy that we must look if the prevalent view of the pathogeny of the affection is correct. With few exceptions, however, no good has resulted from the administration of parathyroid, an experience that Pineles has also had with experimental tetany, and von Eiselsberg and others by transplantation of the glands in post-operative tetany. Success has been claimed for treatment by the administration of thyroid in cases in which tetany has resulted after the removal of the gland and in pregnancy, but Chvostek doubts whether the cases have been kept under observation long enough to be sure that they have not relapsed. Good results have also been claimed for the simultaneous administration of both glands, in view of the fact that they are both involved in some cases of tetany. Alt and Scott found that injection of pituitary extract stopped the symptoms of tetany that had been induced in cats by removal of their parathyroid bodies. According to W. G. MacCallum, injection of a soluble salt of calcium will stop the spasms of tetany in dogs from whom the parathyroids have been removed, and calcium given by the mouth has had a like effect in man. It accordingly follows that articles of food rich in calcium, such as milk and eggs, should be given in liberal quantity.

Among prophylactic measures that have been recommended are attention to hygienic conditions of life and the avoidance of debilitating influences; Chvostek thinks it well for the patient to avoid a sedentary life that entails much bending forward of the head.

Meanwhile we must direct our treatment to palliate spasm, to eliminate toxins, and to modify the conditions favourable to their generation. Gastro-enterostomy has been performed with complete success in the gastric variety of the affection by Mayo Robson, Moynihan, and others, and has altered the prognosis in what was hitherto regarded as a particularly fatal form of the disease. According to Fleiner, increasing loss of strength, diminished diuresis, and mechanical over-excitability are indications for operation, but Chvostek is unable to assent to this, as many cases recover without operation, and from a prognostic standpoint it is impossible to separate these from those which run a fatal course. He has found lacto-phenin useful in many of his cases.

In the subjects of dilated stomach, whether adults or children, an emetic should be administered at the outset, and should be supplemented in the former by washing out the organ with warm water, to which some antiseptic agent such as resorcin or naphthol may be added; whilst in the

KING'S COLLEGE HOSPITAL
MEDICAL SCHOOL

latter the emetic, if it achieve nothing, may be repeated in forty-eight hours. No treatment of the kind must, however, be allowed to delay the operation when the indications for gastro-enterostomy are clear. It is important, more especially in cases with gastro-intestinal disturbance in children, or those attended with constipation, that a purgative should be administered every second day, and, as a derivative and antiseptic, calomel answers best. Enemas of boiled water, to which naphthol or some such antiseptic may be added, are especially useful in children, flushing out the lower bowel and at the same time having an antiseptic action. Anthelmintic remedies should be administered at the outset if there be reason to suspect this source of reflex irritation.

Every care should be taken to promote elimination by the kidneys and skin; Turkish baths, the wet pack, or some similar means of free diaphoresis are of especial value in cases apparently due to exposure to cold and wet.

The second great object is to correct the dietary so as to lessen the production of toxic gastro-intestinal substances: in adults the diet should be liberal, nutritious, and at the same time easily digestible; and in children the most stringent rules should be laid down as regards breast or artificial feeding, regular intervals being strictly enjoined in both cases; and in the latter the amount should be carefully regulated in proportion to the age of the patient; the milk should be sterilised, and substituted for any solid food given prematurely.

An attempt should be made to rectify the gastric chemistry by the administration of acids or alkalis, according as one or the other appears indicated. Lime water or Vichy water is of especial value in hyperacidity in children.

In gastric dilatation of the adult, the stomach should be washed out systematically; and in the gastro-intestinal trouble of children the lower bowel should be regularly evacuated by means of enemas. Intestinal antiseptics, such as calomel, benzo-naphthol, or subnitrate of bismuth, must also be administered.

The patient should be placed in the best possible hygienic circumstances, and all debilitating influences removed. Persistent diarrhoea must be checked by such astringents as opium and aromatic sulphuric acid.

Suckling must be stopped. Rickets must be combated by hygienic measures, suitable feeding with milk, cream, or raw meat juice, careful regulation of the bowels, cod-liver oil, lacto-phosphate of lime or of lime and iron, and so forth.

In both adults and children the patient's strength must be built up, the general nutrition improved, and the nutrition of the nervous system above all, by the administration of such remedies as cod-liver oil, malt, arsenic, phosphorus, and iron.

Every possible measure must be adopted to avoid the attacks of spasm. The room should be kept at an equable temperature, and the practice of bringing children the subjects of tetany to the out-patient departments of hospitals on cold days should be strongly condemned. Changes of clothes, whether of the body or bed, should be carefully warmed. Tepid water only should be used in washing, and all movements should be as gentle as possible; in feeding, especially in children, great care and precaution are necessary.

Of external applications for the relief of the contracture in tetany stimulating liniments are useful, and compresses of chloroform applied to the rigid muscles. Cold applications are sometimes successful, and Trousseau applied ice to the spine with advantage in some cases. For the relief of laryngismus stridulus, on the other hand, Trousseau preferred a hot sponge applied to the skin over the larynx.

The patient should be placed in a tepid bath as soon as possible after the spasm has begun; and, to diminish the number of paroxysms, baths should be given in the intervals three or four times a day, at a temperature of about 30° C., the patient being immersed for about ten or fifteen minutes at a time. Baths are especially useful in acute attacks of tetany with frequent paroxysms and some elevation of temperature; they are of no use in the chronic forms of the affection.

Electrical treatment of the nerves and muscles is useless. Faradism is never appropriate; but feeble descending galvanic currents with a large anode on the vertebral column, and the cathode to the nerve-trunks, are not without benefit in diminishing the irritability of the spinal cord; the method is indicated rather in tetany which is tending to become chronic.

Of drugs administered internally the bromides, chloral, and urethane are better than morphine, which, though useful in some cases, tends to constipation and cerebral congestion. Pain may, however, compel its use. Great caution is necessary in administering opium to young children. Chloral, if given by enema to children, causes no increase of the spasm, such as results when a child is forced to take medicine by the mouth. In one case in which both bromide and chloral failed, Cheadle found Calabar bean successful; but the patient was under treatment with cod-liver oil and iron at the same time. Belladonna is disappointing. Valerian and allied drugs may be given in conjunction with one or other of the sedatives. Tonnellé in some cases used enemas of asafetida with advantage. In tetany complicating tuberculosis there is a disturbance in the calcium metabolism with an excessive excretion of this mineral, and calcium salts should therefore be given by the mouth (Winternitz).

If death appear imminent from asphyxia, owing to spasm of the larynx, or of the respiratory muscles, or of both, inhalations of chloroform must be employed. The greatest possible care is necessary in the administration of chloroform, as it may increase the spasm at first; but relaxation is usually obtained on complete narcosis. Unfortunately this mode of treatment does not prevent recurrence of the spasm; but Hauber, in two cases in which other methods of treatment failed, succeeded with inhalations of chloroform combined with energetic massage during the narcosis.

Rapid relaxation of the spasm of tetany may be brought about by the administration of a subcutaneous injection of pilocarpine; it sets in as diaphoresis is induced. Chvostek, whilst considering diaphoresis useful, is opposed to the use of pilocarpine.

For nocturnal tetany Sir William Gowers finds nothing so good as a dose of digitalis given at bed-time. Bromide is, however, sometimes successful; and, whatever measure of this kind be adopted, a tonic line of treatment should be pursued during the day.

J. S. Risien Russell.

REFERENCES

In selecting papers for this bibliography from the extensive literature that has grown up around the subject of tetany, some have been chosen for their importance as general treatises on the subject; others because they deal specially with some part of it; many again have been selected, in part at least, because they contain more or less complete lists of the writings on tetany previous to the publication of the particular paper included in the present bibliography. The publications have been arranged in groups, as far as possible, according to the chief topics dealt with, in the hope that this may facilitate reference.

General Treatises: 1. Althaus. *Boston Med. and Surg. Journ.*, 1886, cxv. 389, 417.—2. Bechterew. *Deutsche Ztschr. f. Nervenh.*, Leipz., 1894-5, vi. 457.—3. Bruns. *Neurol. Centralbl.*, Leipz., 1896, xv. 668.—4. Cassel. *Berlin. klin. Wchnschr.*, 1896, xxxiii. 547.—5. Corvisart. "De la contracture des extrémités en tétanie chez l'adulte," *Thèse*, Paris, 1852.—6. Ewald. *Verhandl. d. XII. Congr. f. innere Med.*, Wiesbaden, 1893, xii. 298; *Wien. med. Presse*, 1893, xxxiv. 858.—7. Frankl-Hochwart. Nothnagel's *Spec. Path. u. Therap.*, Bd. xi. T. ii. Abt. 4, 1897, p. 81; *Die Tetanie*, Berlin, 1891.—8. Gowers. *A Manual of Dis. of the Nerv. Syst.*, Lond., 2nd ed. 1893, vol. ii. p. 698.—9. Griffith. *Amer. Journ. Med. Sc.*, Phila., 1895, N.S., cix. 158; *Trans. Ass. Am. Physic.*, Phila., 1894, ix. 257.—10. Hoffmann. *Deut. Arch. f. klin. Med.*, Leipz., 1888, xliii. 53; *Deutsche Ztschr. f. Nervenh.*, Leipz., 1896, ix. 278.—11. Howard. *Amer. Journ. Med. Sc.*, Phila., 1906, cxxi. 301.—12. Krafft-Ebing. *Allg. Wien. med. Ztg.*, 1894, xxxix. 361, 370; *Med. Press and Circ.*, London, 1894, N.S. lviii. 279; also *Lehrbuch der Psychiatrie*, 1894.—13. Lothrop. *Boston Med. and Surg. Journ.*, 1896, cxxxv. 513, 529.—14. Parsons. *Dublin Journ. Med. Sc.*, 1894, xcviii. 201.—15. Remond. *Gaz. des hôp. de Paris*, 1891, lxiv. 1225.—16. Riegel. *Deutsche Arch. f. klin. Med.*, Leipz., 1874, xii. 399.—17. Trousseau. *Cliniques de l'Hôtel-Dieu*, ii. 207; *Gaz. des hôp. de Paris*, 1851, 3^e s., iii. 513; *ibid.*, 1860, xxxiii. 173. **Diagnosis and Etiology**: 18. Chvostek. *Deutsche med. Wchnschr.*, 1909, xxxv. 825, 873.—19. Curschmann. *Deutsche Ztschr. f. Nervenh.*, Leipz., 1904, xxvii. 239. **Infantile Tetany**: 20. Abercrombie. "On Tetany in Young Children," *Thesis*, Cambridge, 1880.—21. Baginsky. *Arch. f. Kinderheilk.*, Stuttg., 1885-86, vii. 321.—22. Bonome e Cervesato. *Pediatria*, Firenze, 1895, iii. 144, 171.—23. Boral. *Beiträge zur Kinderheilkunde*, (Kassowitz), Wien, 1893, 1.—24. Bouchut. *Gaz. des hôp. de Paris*, 1875, xlviii. 458; 1876, xlix. 1145.—25. Cheadle. *Lancet*, 1887, i. 919, 967; *Med. Times and Gaz.*, London, 1880, i. 289, 317.—26. Comby. *Atti dell' XI. congresso med. internaz.*, Roma, 1894, iii. 34.—27. Escherich. *Wien. klin. Wchnschr.*, 1890, 769.—28. Ganghofner. *Ztschr. f. Heilk.*, Prag, 1891, xii. 447.—29. Henoch. *Die Tetanie d. Kind.*, Berlin.—30. Kassowitz. *Wien. med. Wchnschr.*, 1893, lxiii. 545, 647, 828, 910.—31. Langmead. *Trans. Clin. Soc.*, Lond., 1906, xxxix. 48.—32. Lewi. *Arch. Pediat.*, New York, 1895, xii. 602.—33. Oddo. *Rev. de méd.*, Paris, 1896, xvi. 458, 573, 667, 749; *Méd. infant.*, Paris, 1894, i. 483.—34. Tonnellé. *Gaz. méd. de Paris*, 1832, iii. 1. **Gastric Dilatation**: 35. Bamberger. *Bericht der Krankenanstalt Rudolfstiftung in Wien*, 1891 (1892), 290.—36. Bouveret et Devic. *Rev. de méd.*, Paris, 1892, xii. 48, 97.—37. Cassaet et Benech. *Compt. rend. Soc. de biol.*, Paris, 1894, x. 1, 532.—38. Dreifus-Brissac. *Gaz. hebd. de méd.*, Paris, 1885, xxii. 439.—39. Dickson, Carnegie. *Practitioner*, London, 1903, lxx. 44.—40. Dujardin-Beaumetz et Oettinger. *Union méd.*, Paris, 1884, xxxvii. 169; *Société méd.*, 1884, 169, 209.—41. Ewald. *Neurol.*

Centralbl., Leipz., 1893, 326.—42. Fenwick. *Trans. Clin. Soc.*, London, 1895, xxxviii. 13.—43. Fleiner.—*Arch. f. Verdauungskr.*, Berlin, 1895, i. 243.—44. Galliard. *Compt. rend. Assoc. franç. pour l'avance. de sc., c.-r.*, Paris (1884), 1883, xii. 715.—45. Gassner. *Inaug. Diss.*, Strassburg, 1878.—46. Kussmaul. *Berlin. klin. Wchnschr.*, 1872, ix. 441 ; *Deutsche Arch. f. klin. Med.*, Leipz., 1869, vi. 481.—47. Laprevotte. "Des accidents tétaniformes dans la dilatation de l'estomac," *Thèse*, Paris, 1884.—48. Leven. *Maladies de l'estomac*, Paris, 1879.—49. Luna. *Marseille méd.*, 1895, xxxii. 232.—50. Lopez. "Contribution à l'étude de la tétanie dans la dilatation de l'estomac," *Thèse*, Paris, 1892.—51. Martin. *Lancet*, 1887, i. 74.—52. Moorhead. *Practitioner*, London, 1904, lxxiii. 60.—53. Moynihan. *Ibid.*, 1903, lxx. 354.—54. Müller. *Deutsche militärärzt. Ztschr.*, Berl., 1884, 439 ; *Charité-Ann.*, Berl., 1888, xiii. 273.—55. Neumann. *Deutsche Klinik*, Berl., 1861, xii. 26.—56. Oddo et Luna. *Bull. et mém. Soc. méd. des hôp.*, 1896, xiii. 372, 423.—57. Palliard. *Rev. de méd.*, Paris, 1888, viii. 406.—58. Richartz. *Über Tetanie und sonstige Krampfzustände bei Hypersekretion des Magens*, Giessen, 1893.—59. Schlesinger. *Wien. klin. Wchnschr.*, 1894, vii. 165. **Tetanilla Strumipriva**: 60. Blazicek. *Ibid.*, 1894, vii. 826, 869.—61. Bramwell, Byrom. *Brit. Med. Journ.*, 1895, i. 1196.—62. Drechsel. *Centralbl. f. Physiol.*, Leipz., 1895-6, ix. 705.—63. Eiselsberg. *Über Tetanie in Anschlusse an Kropfoperationen*, Wien, 1890 ; *Beiträge zur Chirurgie*, Festschrift gewidmet Th. Billroth, Stuttg., 1892, 371.—64. Fuhr. *Arch. f. Path. u. Pharmakol.*, Leipz., 1886, xxi. 387.—65. Gottstein. *Deutsche Ztschr. f. Nervenh.*, Leipz., 1894-5, vi. 177.—66. Hoffmann. *Deutsche Ztschr. f. Nervenh.*, 1896, ix. 272.—67. Horsley. *Proc. Roy. Soc.*, Lond., 1884-5, xxxviii. 5 ; 1886, xl. 6 ; *Brit. Med. Journ.*, 1885, i. 111 ; 1890, i. 287 ; ii. 201.—68. Kocher. *Arch. f. klin. Chir.*, Berl., 1883, xxix. 302.—69. Lépine. *Semaine méd.*, Paris, 1896, xvi. 57, 333.—70. Levy-Dorn. *Therap. Monatsh.*, Berlin, 1896, x. 63.—71. Masoin. *Arch. de physiol. norm. et path.*, Paris, 1895, 5. s., vii. 368.—72. Mikulicz. *Wien. med. Wchnschr.*, 1886, xxxvi. 1, 40, 70, 97.—73. Notkin. *Ibid.* 1895, xlv. 824, 872.—74. Schiff. *Arch. f. Path. u. Pharmakol.*, Leipz., 1884, xviii. 25.—75. Szuman. *Centralbl. f. Chir.*, Leipz., 1884, xi. 29.—76. Wagner. *Wien. med. Bl.*, 1884, vii. 771, 931.—77. Weiss, N. *Samml. klin. Vortr.*, Leipz., 1880, Nr. 63 (M. Nr. 189), *Anz. der K.K. Gesellsch. d. Ärzte in Wien*, 1883, No. 31, 209.—78. Wölfler. *Verhandl. der deutschen Gesellsch. f. Chir.*, 1883. **Pathogenesis**: 79.—Caro. *Mitt. a. d. Grenz. d. Med. u. Chir.*, Jena, 1907, xvii. 447.—80. Chvostek. *Wien. klin. Wchnschr.*, 1905, xviii. 969 ; *ibid.*, 1907, xx. 487.—81. Erdheim. *Mitt. a. d. Grenz. d. Med. u. Chir.*, Jena, 1906, xvi. 632.—82. Escherich. *München. med. Wchnschr.*, 1907, liv. 2073.—83. Glaserfeld. *Berlin. klin. Wchnschr.*, 1909, xlvi. 112.—84. MacCallum and Voegtlin. *Journ. Exper. Med.*, N.Y., 1909, xi. 118.—85. Marinesco. *Semaine méd.*, Paris, 1905, xxv. 289.—86. Pick, *Neurol. Centralbl.*, Leipz., 1902, xxi. 578.—87. *Ibid.*, 1903, xxii. 754.—88. Pineles. *Deutsches Arch. f. klin. Med.*, Leipz., 1905, lxxxv. 49.—89. Rudinger. *Ztschr. f. exper. Path. u. Therap.*, Berlin, 1909, v. 205.—90. Saiz. *Wien. klin. Wchnschr.*, 1908, xxi. 1322.—91. Vassale. *Riv. sper. di freniat.*, Reggio-Emilia, 1897, xxiii. 915.—92. Winternitz. *Bull. Johns Hopkins Hosp.*, Balt., 1909, xx. 269. **Pregnancy, etc.**: 93. Burresi. *Gaz. méd. de Paris*, 1856, xi. 176.—94. Frankl-Hochwart. *Die Tetanie*, Berlin, 1891.—95. Gottstein. *Deutsche Ztschr. f. Nervenh.*, Leipz., 1895, vi. 177.—96. Hoffmann. *Deutsches Arch. f. klin. Med.*, Leipz., 1888, xliii. 53.—97. Meinert. *Arch. f. Gynäkol.*, Berlin, 1887, xxx. 444.—98. Neumann. *Ibid.*, 1895, xlviii. 49 : *Centralbl. f. Gynäkol.*, Leipz., 1894, xviii. 499.—99. Thomas. *Johns Hopkins Hosp. Bull.*, Balt., 1895, vi. 85.—100. Trousseau. *Gaz. des hôp.*, Paris, 1851, 3^{e} s., iii. 513 ; *ibid.*, 1856, xxix. 285 ; *ibid.*, 1860, xxxiii. 173. **Epidemics**: 101. Bernhardt. *Berlin. klin. Wchnschr.*, 1891, xxviii. 633.—102. Dupuy. *Gaz. obst. de Paris*, 1877, vi. 7.—103. Hillairet. *Gaz. des hôp.*, Paris, 1876, xlviii. 1124.—104. Jaksch. *Ztschr. f. klin. Med.*, Berlin, 1890, xvii. 144.—105. Magnan. *Gaz. des hôp.*, 1876, 1100.—106. Mareska. *Ann. et bull. de la Soc. de méd. de Gand*, 1846.—107. Regnard. *Gaz. des hôp.*, Paris, 1876, 1077.—108. Simon. *Progrès méd.*, Paris, 1876, iv. 856.—109. Simon and Rigard. *Med. Examiner*, 1877, ii. No. 3.—110. Schultze. *Méd. mod.*, Paris, 1895, vi. 211. **Infectious Diseases**: 111. Aran. *Union méd.*, Paris, 1855, ix. 341 ; *Bull. Soc. méd. des hôp. de Paris*, 1855.—112. Blazicek. *Semaine méd.*, Paris, 1896, xvi. 95.—113. Bouchut. *Gaz. des hôp.*, Paris, 1876, xlix. 1145.—114. Bucquay. *France méd.*, Paris, 1876.—115. Leger. *Ibid.*—116. Löb. *Arch. f. Kinderheilk.*, Stuttg., 1889,

x. 212.—117. Moutard-Martin. *Soc. méd. des hôp.*, 1856.—118. Oddo. *Thèse.* Paris, 1886.—119. Schotten. *Berlin. klin. Wchnschr.*, 1888, xxv., 273. **Hysteria**: 120. Blazicek. *Wien. klin. Wchnschr.*, 1896, ix. 373.—121. Gilles de la Tourette et Bolognesi. *Nouv. icon. de la Salpêtrière*, Paris, 1895, 277.—122. Murray. *Med. Rec.*, N.Y., 1896, xlix. 400.—123. Nikolajenic. *Wien. klin. Wchnschr.*, 1893, vi. 526.—124. Raymond. *Bull. méd.*, Paris, 1888, ii. 599.—125. Schlesinger. *Neurol. Centralbl.*, Leipz., 1893, xii. 459.—126. Zaldivar. "De la nature hystérique de la tétanie essentielle." *Thèse.* Paris, 1888. **Mechanical and Electrical Condition of Nerves and Muscles**: 127. Althaus. *Boston Med. and Surg. Journ.*, 1886, cxv. 389, 417.—128. Benedikt. *Elektrotherapie*, Aufl. 2, 1886, 612.—129. Burkhardt. *Corr.-Bl. f. schweiz. Ärzte*, Bern, 1893, xxiii. 17.—130. Chvostek. *Ztschr. f. klin. Med.*, Berlin, 1891, xix. 489.—131. v. Ziemssen's *Handbuch*, Leipzig, Vogel, 1878; *Arch. f. Psychiat.*, Berlin, 1874, iv. 271; *Electrotherapie*, Aufl. 2, 1886.—132. Frankl-Hochwart. *Centralbl. f. klin. Med.*, Leipz., 1887, viii. 377; *Deutsches Arch. f. klin. Med.*, Leipz., 1888, xliii. 21; xliv. 429.—133. Hayem. *De l'excitabilité électrique et mécanique des muscles et des nerfs dans la tétanie*, Paris, 1887.—134. Hoffmann. *Arch. f. klin. Med.*, Leipz., 1888, xliii. 53.—135. Loos. *Wien. klin. Wchnschr.*, 1891, iv. 915.—136. Onimus. *Gaz. des hôp.*, Paris, 1876, xlviii. 246.—136*a*. Peters. *Deutsche Arch. f. klin. Med.*, Leipz., 1903, lxxvi. 69.—137. Schlesinger, *Allg. Wien. med. Ztg.*, 1890, xxxv. 351, 363, 380; *Neurol. Centralbl.*, Leipz., 1892, xi. 66.—137*a*. *Idem.* "Über ein bisher unbekanntes Symptom bei Tetanie (Beinphänomenon)." *Wien. klin. Wchnschr.*, 1910, xxiii. 315.—138. Schultze. *Deutsche med. Wchnschr.*, Berlin, 1882, viii. 276, 292. **Prognosis**: 139. Frankl-Hochwart. *Neurol. Centralbl.*, Leipz., 1906, xxv. 642, 694.—140. Moynihan. *Practitioner*, London, 1903, lxx. 354. **Treatment**: 141. Chvostek. *Deutsche med. Wchnschr.*, 1909, xxxv. 825, 873.—142. MacCallum. *Bull. Johns Hopkins Hosp.*, Balt., 1908, xix. 244.—143. Moynihan. *Practitioner*, London, 1903, lxx. 354; and *Boston Med. and Surg. Journ.*, 1903, cxlviii. 591.—144. Ott and Scott. *New York Med. Journ.*, 1908, lxxxviii. 1180.—145. Winternitz. *Bull. Johns Hopkins Hosp.*, Balt., 1909, xx. 269.

J. S. R. R.

RECURRENT PARALYSIS

By Prof. J. Michell Clarke, M.D., F.R.C.P.

Certain affections of the nervous system are characterised by recurring attacks of temporary palsy, which in some forms constitute the whole disease, and in others after a variable period end in permanent paralysis. In other instances the recurrent paralysis is merely a phase of a more widespread disorder. These affections are of varied character and the pathological conditions underlying them are of different kinds. The nature of these and the manner in which the transient paralysis is brought about are in most instances obscure and puzzling. For convenience of clinical description such recurrent paralyses may be provisionally grouped together, and it is possible that by thus associating the various forms the elucidation of the mechanism by which they are directly produced may be aided.

Attention has already been drawn (Vol. VII. p. 69) to cases allied to family periodic paralysis in the periodicity of attacks, but differing from it in important particulars. Further observations are necessary to elucidate the relations between them. To the instances there given may be added

Bornstein's case, a boy, aged fourteen, in whom periodic paralysis was associated with the reaction of degeneration in many muscles during the paralytic seizures, and who suffered from attacks of *petit mal* between them. Also the record given by Rich, as existing in his own family for five generations, of temporary motor paralysis due to cold, affecting chiefly the facial muscles; one patient was completely paralysed, except the tongue, as the result of sleeping in damp underclothing. Sensation was unaffected. Paralysis of the tongue could always be brought about by holding snow in the mouth. Whilst in family periodic paralysis the muscles supplied by the cranial nerves usually escape, some of the best examples of recurring paralysis apart from this disease are those of the third nerve, the so-called "ophthalmic migraine," and of the seventh nerve.

The name **ophthalmic migraine** arose from the fact that the recurrent oculomotor paralysis is ushered in by attacks of severe headache or hemicrania with vomiting. In this connexion it is interesting to note the close relation of hemicrania or sick headache to periodic paralysis in the family reported by Holtzapple. In four generations eighteen persons suffered from severe attacks of migraine, seventeen from periodic paralysis, of whom five were subject to both affections, twelve to paralysis only, and thirteen to headache only. All the patients were descended from an ancestress who suffered from sick headache only, and in the branch of the family most severely affected with paralysis—three of whom died in attacks—the mother had migraine but no paralysis. It is remarkable that none of the patients with sick headache are stated to have had oculomotor paralysis after it.

Further, although in attacks of migraine concomitant or consecutive motor disturbance is very uncommon, the nervous phenomena being as a rule entirely on the sensory side, yet some degree of motor paralysis occasionally occurs. This generally consists of a transient loss of power in the arm, which in severe attacks is not infrequently the seat of sensory disturbance in the form of tingling or numbness, and lasts from ten to thirty minutes. Sometimes the leg on the same side is similarly affected, as in one of my patients, who said that in consequence she could not walk for a few minutes. Dr. Liveing in his classical work mentions a few instances of this kind; a case is recorded by Féré in which paralysis of the right arm came on in the course of the attacks, lasted about one and a half hours, to disappear with the scotoma; and Spitzer mentions as an occasional incident of migraine a transitory monoplegia or hemiplegia.

Transitory aphasia is not uncommon as part of a severe attack of migraine, in most instances it is described as of the sensory variety, but in some seems to be due to defect of articulation, the patients stating after the attack that "they could not form their words" during it, although retaining full comprehension of what they wished to say and of spoken speech. Hemiopia is another well-known recurring and transient disability in the attack. Hemiopia of this kind occasionally persists for some time after the headache has ceased, in one case it lasted for one month, and there are several instances on record in which it was at first transient and confined to the duration of the attacks, but ultimately became permanent. In two cases in which this resulted, thrombosis of the central artery of the retina was the cause.

I have recently had under my observation a unique example of the occasional association of motor paralysis with attacks of migraine in a family in which several members suffer from typical and severe accesses of this disorder accompanied by transient but complete hemiplegia. So far as could be ascertained, the earliest member of the family to be affected in this way was the grandfather of my first patient, the latter was a boy aged nine. Three generations are thus involved. Out of eighteen members of the family six have been affected, and possibly two more. The age of onset has varied from seven to twenty-eight years; as some of the unaffected members now living are under nine years of age, the proportion of sufferers is likely to be increased. Males and females have suffered indifferently. The attacks begin with hemiopia, then hemiplegia occurs, occasionally so suddenly that if standing or walking the patient stumbles or even falls, and is followed by intense localised headache or hemicrania; the hemiplegia is complete, is accompanied by loss of sensation on the same side, varying in degree in different cases, and by loss of speech. Vomiting supervenes after about two hours or longer, and the hemiplegia then passes off, or lasts some hours longer. In some of the patients recovery is complete almost at once, in others there is weakness on the hemiplegic side for some days. In the attack witnessed, the headache was in the left temporal region; there was right hemiopia, right hemiplegia affecting the face, chiefly in the lower part, the arm, and the leg; the paralysis was absolute, the muscles flaccid, the knee-jerk was doubtfully absent on admission but all reflexes were present next day. Right partial hemianaesthesia (touch and pin-prick), loss of speech, and difficulty in swallowing were present. This attack of hemiplegia was two to three days in passing off; the arm had then recovered perfectly, but slight awkwardness on movement persisted in the leg for a week. In two other patients seen in the intervals there was no trace of paralysis of any kind, and the reflexes were normal. One boy, aged nine years, was stated to have had one very severe attack in which paralysis affected first the right side of the face, the right arm and leg, and was followed, twenty minutes later, by similar paralysis of the left side; the muscles of the neck were also affected, so that he could not hold his head up. Recovery took place in a few hours. In this attack, besides other essential differences from those of family periodic paralysis, the boy complained that "it had suddenly become dark," and that he could not see his mother; presumably right hemiopia accompanied the right hemiplegia at the onset, and when the paralysis spread to the left side left hemiopia also followed. It should be added that none of the attacks was attended by any kind of muscular spasm or fits, by loss of consciousness, by disturbance of the bladder or rectum, and that the patients, when speech was lost, could always understand what was said to them.

Periodical or recurring palsy of the third nerve often extends over many years, is generally one-sided, and usually begins with severe pain in or over the eyes with headache and vomiting: this part of the attack may last two or three days or longer. It resembles migraine but may differ from it in its longer duration and some other points. The paralysis follows the headache but sometimes appears before the latter has passed off. The attacks may begin in early childhood and last up to middle life. In some cases the attacks of oculomotor paralysis only appear after the patient has suffered from migraine for a period of from two to four years or longer. Women are more often affected than men. Ptosis is generally present, and there may be paresis or complete paralysis of the third nerve with affection of the internal ocular muscles. The interval between the attacks varies from weeks or months to years, and the duration of the attacks from a few days to several weeks or even months. According to Sir W. Gowers, when the attacks occur frequently they are usually brief (days). In the intervals there may either be complete recovery, or some paresis, either of the whole nerve or one of its branches, remains. In one case attacks of "migraine" without paralysis alternated with attacks followed by, in order, ptosis, dilatation of pupils, total paralysis of the third nerve. The fourth and the sixth nerves may be simultaneously affected. Occasionally amblyopia has been noted, and also hyperaesthesia and pains in the distribution of the branches of the fifth nerve. The seventh nerve is exceptionally involved. In some of the cases paresis persists during the intervals, and eventually results in partial or total permanent paralysis.

There is considerable difference of opinion as to the nature of these cases, and they undoubtedly depend on different causes. Some authorities deny their connexion with true migraine, because of the absence of an aura, of evidence of hereditary transmission, and because of the length of the attacks, and of the frequent persistence of some degree of paralysis in the intervals. They thus prefer to speak of these cases as hemicrania with recurrent third nerve paralysis. Mr. Holmes Spicer and Dr. Ormerod, from a study of 36 cases, concluded that although the headaches were one-sided, they had not the characteristics of migraine. On the other hand, there are undoubtedly cases which satisfy all the above clinical criterions for true migraine, in addition begin, like it, in early life, and which shew complete recovery from paralysis in the intervals between the attacks. Further, cases have been described in which attacks of migraine in the simple form have preceded for years a subsequent association with recurrent oculomotor palsy. Probably there are two distinct groups (i.) those in which periodic oculomotor paralysis accompanies or follows attacks clinically identical with ordinary migraine, in which the paralysis does not persist during the intervals, and rarely, if ever, becomes permanent; and (ii.) those in which there are attacks of hemicrania rather than of true migraine, in which more or less oculomotor paralysis is present between the periodic exacerbations, and eventually becomes complete and permanent. It may be said further that in the first group there is probably no organic lesion recognisable, and that the second group depends on a basal lesion, for example, tumour, syphilitic and tuberculous changes, and in these the paralysis, at first periodic, becomes permanent. Thus, although records of pathological investigations are scanty, in two cases (Karplus, Thomsen), a tumour, in each apparently of congenital origin, involved the third nerve at the base of the brain, whilst in another case a plastic exudation, and in two others syphilitic lesions, were found in the same situation. Möbius marks off a group with the following diagnostic signs: (*a*) youth of patients—under twenty-five years of age; (*b*) onset with symptoms of migraine, but without trigeminal neuralgia; (*c*) paralysis, one-sided and total; (*d*) between the attacks, some degree of paralysis persists and with time increases; (*e*) hyperaesthesia in the distribution of the first division of the fifth nerve. He attributes this group to a peripheral basal lesion (new growth).

Sometimes periodic palsy of the third nerve occurs without symptoms of headache and vomiting, and it has been observed in connexion with the Argyll Robertson phenomenon and early signs of tabes dorsalis.

There is undoubted evidence of the existence of a **recurrent facial palsy**, but it is doubtful if it is of the same nature as the oculomotor form, and unlike this, it is only rarely associated with attacks of "migraine." According to Bernhardt the recurrent form occurs in about 7 per cent of all cases of facial paralysis, and affects men somewhat more often than women, the age of onset is generally between twenty and fifty years; the first relapse may occur some weeks or many years after the first attack—the second generally from the fifth to seventh year after it; more than two recurrences are very rare; the recurrence does not implicate the side of the face first affected more often than the opposite side; the relapse, as judged by the electrical reactions, is as severe as the first attack. Of recurrent facial paralyses, about 66 per cent are without definite cause or belong to the so-called "rheumatic" group: these would come under the category of the recurrent paralyses that we have hitherto been considering; of the remainder, 10 per cent depend on disease of the middle ear, 6 per cent occur in syphilitic, and 5 per cent in diabetic patients. In several instances recurrent facial palsy has affected more than one member of a family. In any case, care must be taken not to overlook a recurrent otitis media. In one instance mercurial poisoning, induced by the patient's occupation as a hat-maker, was a possible cause (Abrahamson). The occasional association with other neuroses is exemplified by a case in which attacks of hemicrania were followed from time to time by facial paralysis, and two other members of the family were affected with sleep-ptosis.

In a sense, the paresis of which patients occasionally complain on waking from sleep is a form of recurrent paralysis. It is associated with numbness, tingling, or slight loss of sensation; the paresis may be monoplegic or hemiplegic. It generally disappears in a few minutes.

Weir Mitchell states that the loss of power varies greatly from paresis up to a brief simulation of paralysis. The attacks may recur every night or at intervals, and "may even deepen in intensity after the patient is fully awake." Such attacks occur in neurasthenia, hysteria, gout, dyspepsia, and from abuse of tobacco. Sleep-ptosis is a similar affection: the patient is unable to raise the eyelids for some minutes or longer after waking from sleep.

In *Raynaud's disease*, recurrent attacks of transient hemiplegia have been observed. Thus, Prof. Osler records a case of this disease in a woman, aged thirty-eight, who had two attacks of aphasia, accompanied, in the first, by loss of power in the right hand and paresis in the right foot; and, in the second, by spasm of the right hand. Three years afterwards she had an attack of headache in which slight paralysis of the left arm and leg accompanied local asphyxia of the right hand, and later still she had a further attack of aphasia and right hemiplegia. He also refers to a case reported by Weiss in which there was recurrent temporary aphasia. There are several instances of recurrent attacks of temporary amblyopia in Raynaud's disease, and in one of them permanent loss of vision in the upper half of the visual field of the right eye resulted (*vide* also Vol. VII. p. 134).

In the course of *uraemia* a hemiplegia or a monoplegia is not infrequent as a transitory phenomenon, which more or less rapidly clears up. Dr. A. E. Russell thinks that such paralyses may be due to localised cerebral oedema, and refers to observations on cases of cerebral decompression by Cushing and Bordley, who concluded that local and transient hemiplegia, aphasia, amaurosis, and convulsions were due to localised oedema of the brain. In support of this view Dr. Russell adduces a case of Prof. Osler, and as it is an unusual form of recurrent paralysis, I quote his account of it:—

> The patient, a man aged twenty-nine, at the age of twelve had an attack of right hemiplegia and aphasia, which lasted about a week. Within the year he had five or six further attacks of hemiplegia, each successive one less severe and none of them accompanied by aphasia. From the age of twenty-six he became subject to severe attacks of migraine. At twenty-eight he had a sudden attack of pain and swelling in the feet. He had frequent attacks of angioneurotic oedema of the upper lip, and outbreaks of urticaria associated with severe pains in the legs. He had one attack of abdominal colic accompanied by purpura and urticaria; later he suffered from haematuria and albuminuria. Prof. Osler considered that the recurring attacks of hemiplegia were probably associated with changes in the brain of essentially the same nature as those which occurred in the lips and skin.

There is a well-marked form of recurrent or paroxysmal attacks of paresis or paralysis which occurs in the early stages of diseases of the arteries from syphilis, arteriosclerosis, or atheroma. As in these cases the underlying cause is narrowing or partial obstruction of an artery, the nutrition of the nervous tissues supplied will be damaged as a result of the lessened blood-supply; further, the supply will be liable to vary with changes in the blood-pressure, and variations (depression) in function will therefore ensue. If, as is often the case in such patients, the heart is also damaged and its action weak, the circulation through the diseased vessel may the more easily fail from the strain of unusual exertion or of an attack of some debilitating disease such as diarrhoea. Recurrent attacks of hemiparesis, of aphasia, and of bulbar paralysis may thus occur in the early stage of disease of intracranial vessels, often preceding a permanent paralysis in the distribution of these initial symptoms. The nocturnal palsies above described may be precursory signs of the onset of such permanent paralysis. In some of these states the condition, both in recurrent paresis of the limbs and of the muscular mechanisms supplied by the bulbar nerves, may give rise to symptoms closely resembling those of the "myasthenic reaction" of myasthenia gravis (*vide* Vol. VII. p. 58).

Another explanation has been offered for this form of recurrent paresis, aphasia, or hemiplegia. Dr. W. Russell considers that they are due to a local spasm or contraction of the muscular coat of a cerebral artery, varying in degree and extent, producing either partial or total occlusion of the vessel, and intermittent. The results of such spasm would be (1) to impair the function of the area supplied; (2) to favour the occurrence of thrombosis, if the heart were feeble; or (3) to lead to haemorrhage proximal to the site of contraction if the heart were acting strongly and the vessels not sound. Although this explanation is at first sight a tempting one, positive proof is lacking. In its support the observed spasmodic contraction of retinal arteries with temporary amblyopia in Raynaud's disease, and of branches of the temporal artery in migraine has been adduced. On the other hand, the musculature of the cerebral arteries is feeble and not comparable to that of the systemic; whilst the power of strong contraction of the muscular coat of the latter is undoubted, the evidence of an analogous contractile power in the cerebral arteries is not generally accepted, and it is not certain that the conditions of circulation in the retinal are the same as in the cerebral arteries.

A few words may be added in this connexion on the obscure and difficult subject of the mode of causation of the recurrent paralyses of migraine and of that form of periodical oculomotor paralyses in which there is no recognisable organic disease, because of all the hypotheses that have been advanced in explanation that of localised vasomotor spasm of cerebral arteries is perhaps the one most generally accepted. In support of this hypothesis an analogy with Raynaud's disease is sometimes drawn. We have seen that transient attacks of paralysis are occasionally observed in this disease, and that they were present in the remarkable case of angioneurotic oedema quoted on p. 609, in which attacks of migraine also occurred. In Raynaud's disease contraction of the retinal vessels during attacks of amblyopia has been actually witnessed. Moreover, in this disease permanent amblyopia has been the result of preceding temporary attacks, and similarly in migraine a recurrent hemiopia has become permanent, though very rarely. The pathology of Raynaud's disease is still obscure, and a permanent paralysis of any kind is so rare in migraine in relation to the numberless cases of the disorder, and so apart from its usual course as to make it probable that when it occurs some additional and unusual factor is present. In favour of the origin of the attack of migraine in spasm of cerebral arteries the contraction of the temporal or retinal arteries or of those of the skin, which has been observed in many cases before or during the attack, has been brought forward. Even, however, if the argument from this to a similar state in cerebral arteries is valid, of which there is no proof, such contraction of the superficial and retinal arteries is not a constant feature of the attack, for the face has often been described as flushed and the retinal arteries have been considered to be dilated.

A full discussion of the various theories of migraine would be out of place here, and the reader may be referred to the references given below; the most recent hypothesis is that of Plavec, who attributes ordinary migraine to periodical swelling (hyperaemia) of the hypophysis cerebri, and accounts for the ophthalmoplegic variety by an additional condition of venous stasis together with lateral displacement of the hypophysis, so that it presses upon the third nerve.

The aged sometimes have recurrent difficulty of articulation and of swallowing which may last for days or weeks, and then disappear, to return after a varying interval.

"Intermittent claudication," originally described by Charcot, "intermittent lameness," "myasthenia paroxysmalis angiopathica" (Goldflam) is an affection due to disease of the arteries of the lower extremities (*vide* Vol. VI. p. 611). This disease is generally due to atheroma, and patches of calcification have been seen in the vessels with the *x*-rays; less often it is syphilitic; recent writers lay special stress upon abuse of tobacco as an etiological factor; elderly glycosurics may suffer from it. Men are chiefly affected. After walking a short distance the patient has to stop from muscular weakness, pain in the muscles, and numbness and paraesthesia in one leg. If he rest for a few minutes, he is able to go on until a recurrence of the symptoms shortly compels him to stop again. There is often no pulsation in the dorsalis pedis, and vasomotor changes are generally conspicuous in the limb. Gangrene frequently supervenes. Symptoms like those of "intermittent claudication" affecting the arm may according to Prof. Osler, be met with in cases of cervical ribs (32). He reports a case in which the patient suffered from sensations of numbness or pins and needles, when using the arm. If she continued to work, the skin became red and the limb felt swollen and hot. In a short time she was unable to use the arm and had even dropped things out of her hand. During rest the limb felt natural. Prof. Osler thinks there is probably a special group of these cases, and attributes the symptoms to interference with the blood-supply to the arm from compression of the subclavian in the angle between the cervical rib and the scalenus anticus, so that the demand made for increased blood-supply during muscular exertion cannot be met.

Dejerine describes a group of symptoms which he attributes to disease of the arteries of the spinal cord. In addition to symptoms in the legs similar to those of "intermittent claudication" the patients are liable to imperative calls to micturate; and there may be disturbance of the sexual functions. Further points of distinction are the absence of vasomotor and sensory disorders, presence of pulsation in the art. dorsalis pedis; increase of the deep reflexes in the intervals between the attacks, and during them a further increase of knee-jerks, ankle-clonus, and Babinski's sign. Similar symptoms have been described as affecting the arms. Syphilitic affection of the arteries is the important cause.

A recurring form of multiple neuritis has been described. In one instance the attacks recurred five times—the legs were chiefly involved, the upper extremities being severely affected in only one attack.

In disseminated sclerosis a remarkable remission of the loss of power present sometimes occurs, but this hardly comes under the term recurrent paralysis (*vide* Vol. VII. p. 832).

In Jacksonian epilepsy the localised fits are often followed by a temporary, and in this sense recurrent, paralysis in the same distribution; in intracranial aneurysms recurrent palsies are for a time occasionally observed, and also infrequently in cases of cerebral tumour.

Under the title recurrent pseudotumor cerebri Higier describes a case in which the patient at the age of six years suffered from an illness of twelve months' duration with the following symptoms:—paroxysmal headache, vomiting, attacks of Jacksonian epilepsy, dysphasia, right-sided hemiparesis, paralysis of the left sixth nerve, and double optic neuritis. Gradual recovery took place, and he remained well for thirteen years, when the above menacing symptoms returned. After nine months they slowly disappeared again, leaving the patient in sound health. The remarkable feature in this case is, of course, the recurrence of the same symptoms after so long an interval. The term pseudotumor cerebri is used as a concise designation for cases in which undoubted signs of an intracranial tumour are present, but subside.

The **treatment** of these various forms of recurrent paralysis in any particular case, is that of the cause or of the underlying condition. It is unnecessary, therefore, to consider it at any length in this place. The treatment of the first group of "ophthalmic migraine" is that of migraine. With regard to recurrent paresis or hemiplegia of cerebral origin it should never be forgotten that previous infection with syphilis may be the cause, and in such a case active antisyphilitic treatment should be instituted without delay. When these affections depend on arteriosclerosis, careful regulation of the diet and mode of life, restriction in the use of alcohol and tobacco, the administration of an occasional mild mercurial or other purgative, of small or moderate doses of the iodide of potassium or sodium over long periods, and of the nitrites are the most important points. Many authors lay stress on the abuse of tobacco in the causation of "intermittent claudication."

J. Michell Clarke.

REFERENCES

1. Abrahamson. "Case of Recurrent Peripheral Facial Paralysis," *Journ. Nerv. and Ment. Dis.*, N.Y., 1904, xxxi. 545.—2. Bernhardt. "Über die sogen. recidivierenden Facialislähmung," *Neurol. Centralbl.*, 1899, xviii. 98, 196.—3. Bornstein. "Ein Fall von periodischer Lähmung," abstr. *Jahresb. ü. Neurol. u. Psychiat.*, 1905, viii. 378.—4. *Idem.* "Migraine ophtalmique," *Gaz. lek.*, Warszawa, 1907, xxvii. 75, abstr. *Jahresb. ü. Neurol. u. Psychiat.*, 1908, xi. 844.—5. Dejerine. "Sur la claudication intermittente de la moelle épinière," *Rev. neurol.*, 1906, xiv. 341.—6. Determann. "Intermittierendes Hinken," *Deutsche Ztschr. f. Nervenh.*, Leipz., 1905, xxix. 152.—7. Erb. "Zur Kasuistik der intermittierenden angiosklerotischen Bewegungsstörungen," *ibid.*, 465.—8. Féré. "Note sur les rêves précurseurs de la migraine ophtalmique," *Rev. de méd.*, Paris, 1903, xxiii. 127.—9. Goldflam. "Zur Ätiologie und Symptomatologie des intermittierendes Hinkens," *Neurol. Centralbl.*, Leipz., 1903, xxi. 994.—10. Gowers. *Borderland of Epilepsy*, London, J. and A. Churchill, 1907. —11. Grasset. "La Claudication intermittente des centres nerveux," *Rev. neurol.*, Paris, 1906, x. 433.—12. Hagelstam. "Über intermittierenden Hinken," *Deutsche Ztschr. f. Nervenh.*, Leipz., 1901, xx. 65.—13. Harris. "Cause and Treatment of Headaches," *Lancet*, Lond., 1907, i. 276.—14. Heyerdahl. "Migraine" (112 cases), abst. *Jahresb. ü. Neurol. u. Psychiat.*, 1908, xi. 853.—15. Higier. "Rezidivierender Pseudotumoren des Gehirns," *Neurol. Centralbl.*, Leipz., 1910, xxix. 342.—16. Holtzapple. "Periodic Paralysis," *Journ. Amer. Med. Assoc.*, Chicago, 1905, xlv. 1224.—17. Hudovening. "Ein Fall von Migraine ophtalmique," *Orvosi Hetilap*, 1904, No. 7, abstr. *Jahresb. ü. Neurol. u. Psychiat.*, 1905, viii. 770. —18. Hunt. "Intermittent Claudication," *Publications Cornell Univ.* (Department Neurol.), 1907, ii.—19. Idelsohn. "Zur Casuistik u. Ätiologie des intermittierenden Hinken," *Deutsche Ztschr. f. Nervenheilk.*, Leipz., 1903, xxiv. 285.—20. Infeld. "Zur Kenntnis der bleibenden Folgen der Migränesanfallen," *Wien. klin. Wchnschr.*, 1901, xxviii. 673.—21. Jochmann. "Rezidivierenden Okulomotoriuslähmung," *Neurol. Centralbl.*, Leipz., 1906, xxiv. 871.—22. Kollaritz. "Über Migraine ophtalmoplégique," *Deutsche Ztschr. f. Nervenh.*, Leipz., 1904, xxvi. 128.—23. Köster. "Zur Kenntnis der rezidivierenden, alternierenden Lähmung des N. oculomotorius," *Neurol. Centralbl.*, Leipz., 1904, xxii. 995.—24. Lamy. "Le Syndrome migraine ophtalmique comme première manifestation dans un cas de syphilis cérébrale," abstr. *Jahresb. ü. Neurol. u. Psychiat.*, 1901, iv. 374.—25. Lukács. "Diplegia facialis hysterica," *ibid.*, 1902, v. 544.—26. Mitchell, S. Weir. *Clinical Lessons on Nervous Diseases*, Philadelphia, 1897.—27. Möbius. "Über period. Okulomotoriuslähmung," *Deutsche Ztschr. f. Nervenh.*, 1900, xvii. 294.—28. *Idem. Die Migräne*, Wien, Alf. Hölder, 1903.—29. Opin. "Migraine ophtalmique," *Progrès méd.*, Paris, 1903, 162.—30. Orbison. "Relapsing Facial Palsy," *Amer. Journ. Med. Sc.*, Phila., 1907, cxxxiii. 392.—31. Osler. "Cerebral Complications of Raynaud's Disease," *ibid.*, 1890, cxii. 522.—32. *Idem. Amer. Journ. Med. Sc.*, Phila., 1910, cxxxix. 469.—33. Paderstein. "Ophthalmoplegische Migräne und periodische Okulomotoriuslähmung," *Deutsche med. Wchnschr.*, 1907, xxxiii. 762, abstr. *Jahresb. ü. Neurol. u. Psychiat.*, 1908, xi. 641.—34. Parker, G. *Bristol Med.-Chir. Journ.*, 1909, xxviii.—35. Petit. "Paralysies faciales récidivantes et paralysies faciales à bascule," *Thèse de Paris*, 1905, abstr. *Jahresb. ü. Neurol. u. Psychiat.*, 1906, ix. 637.—36. Plavec. "Beiträge zur Erklärung der ophtalmoplégique Migraine," *Deutsche Ztschr. f. Nervenh.*, Leipz., 1907, xxxii. 183. —37. Rich. Quoted by Taylor, *Journ. Nerv. and Ment. Dis.*, N.Y., 1898, xxv. 637. – 38. Rossolimo. "Rezidivierende Facialislähmung bei Migräne," *Neurol. Centralbl.*, Leipz., 1901, xix. 744.—39. Russell, A. E. "Some Disorders of Cerebral Circulation and their Clinical Manifestations," Medical Publishing Co., Lond., 1909.—40. Russell, W. "On Intermittent Closing of Cerebral Arteries," *Brit. Med. Journ.*, 1909, ii. 1109.—41. Schilling. "Zur Frage der rezidivierenden Okulomotoriuslähmung," *München. med. Wchnschr.*, 1903, 776.—42. Simon. "Über die atypischen und frühzeitigen Fälle von intermittierenden Hinken," abstr. *Jahresb. ü. Neurol. u.*

Psychiat., 1906, ix. 313.—43. Spicer and Ormerod. "Recurrent Paralysis of Ocular Nerves," *Trans. Ophthalm. Soc. U.K.*, 1896, xvi. 277.—44. Spitzer. *Über Migräne*, Jena, G. Fischer, 1901 (in which references to literature of migraine and ophthalmic migraine up to this date will be found).—45. Strzeminski. "Über die typische wiederkehrende Okulomotoriuslähmung," *Arch. f. ophth.*, Leipz., 1900, li. 464, abstr. *Jahresb. ü. Neurol. u. Psychiat.*, 1901, iv. 496.—46. Thomas, H. M. "Recurrent Polyneuritis," *Phila. Med. Journ.*, 1898, i. 20.—47. Thomas, J. J. "Migraine and Hemianopsia," *Journ. Nerv. and Ment. Dis.*, N.Y., 1907, xxxiv. 153.—48. Zesas. "Über das intermittierendes Hinken," *Fortschr. der Med.*, 1906, No. 7, 185, abstr. *Jahresb. ü. Neurol. u. Psychiat.*, 1906, ix. 313.—49. Clarke, Michell. "Recurrent Motor Paralysis in Migraine," *Brit. Med. Journ.*, 1910, i. 1534.

J. M. C.

THE TICS

By J. S. Risien Russell, M.D., F.R.C.P.

History.—The word "tic" means a "jerk" or "twitch," and must be clearly distinguished from a spasm. Thanks to the French school, especially to Charcot and his pupils at the Salpêtrière, in the first instance, more or less order has been substituted for chaos in the large group of affections of the nervous system in which involuntary muscular contractions occur. Their labours were first devoted to the separation of the various forms of tic from the choreas, and that it is difficult to overestimate the importance of such a discrimination may be judged from the words of Charcot on this subject: "Entre le tic et la chorée il y a un abîme"; and again, "Sans doute, nosographiquement, les tics et la chorée représentent bien, comme je vous l'ai dit, deux affections radicalement distinctes."

Gilles de la Tourette, Letulle, and Guinon have all largely advanced the study of these disorders, and to Magnan and his pupils, notably Saury and Legrain, must also be accorded the credit due to them for their share in the elucidation of these problems. Noir's thesis on the tics was a notable contribution, and Brissaud, Pitres, and Grasset have all added to our knowledge of the subject, which has been further advanced by the labours of Meige and Feindel, who have enabled us to grasp the difference between a tic and a spasm, and to recognise what affections should be included in the former as opposed to the latter group of maladies. The English reader is accordingly especially indebted to Dr. S. A. K. Wilson for his admirable translation of Meige and Feindel's book.

Some confusion is liable to occur, because the word "tic" has also been applied to the painful affection of the face known as "tic douloureux," which is in no sense a tic in the present acceptation of the term, in that the muscular twitchings which result are purely of the nature of a reflex spasm. All modern writers on the subject, including Meige and Feindel, insist that a tic is essentially a mental process, although it has a physical outlet in a muscular movement, so that it is essentially different from a reflex spasm. It will be further evident that all such terms as "habit spasm" and "habit chorea" must be abandoned if confusion is to be avoided; the latter because tic is an affection quite distinct from chorea, and the former because the muscular contractions which occur in tic are not due to spasm. A spasm has been described as the motor reaction which results from irritation at some point in a reflex spinal or bulbo-spinal arc, and neither depends on consciousness nor is under the influence of the will, whereas a tic is a psychical affection in which a purposive co-ordinated act is performed for the fulfilment of some definite object, and in which consciousness is essential and the exercise of the will plays an important part.

No better means of securing a proper conception of the tics can be adopted than that of tracing the genesis of the affection in a concrete example, such as one of those selected by Meige and Feindel. An individual wears a collar, the frayed edge of which chafes the skin of the neck at some point, and as a result the head is abruptly moved so as to free the part from the painful stimulus. A purely spinal reflex movement of defence has so far resulted; but now that the individual has been warned he wishes to avoid the repetition of an unpleasant experience, and so voluntarily inclines his head so as to make it impossible for the painful stimulus to be repeated. What was only a spinal reflex has now become a conscious voluntary act of cortical origin. The following day the collar is replaced by one with its edge free from any defect such as caused the painful stimulus the day before. A few movements of the head are made to satisfy the wearer that there is no likelihood of a repetition of the previous unpleasant experience, and then the matter ends. Even if the idea that he should repeat the movement which is no longer necessary occurs, he is able to put it out of his mind by an effort of the will. It is different, however, in the case of a person who is ripe for the development of a tic, for he not only performs a few movements to satisfy himself that all is now well, but frequently repeats the brusque movements of the day before. Thus, although the irritation no longer exists, the movement that was at first instituted for its relief is continued unnecessarily, so that what was reasonable yesterday has become superfluous to-day, and if continued unchecked will become absurd. In a short time the circumstance which led to the first movement of defence is forgotten, and yet the individual continues to make similar movements which have now become meaningless and automatic. Another characteristic of the affection that may be discerned in a case of the kind is that although the tic has been started by an attempt to avoid the repetition of an abnormal sensation which was unpleasant, the fact that the discomfort no longer exists fails to bring satisfaction to the patient's mind. He dreads the recurrence of the abnormal sensation, and performs all sorts of gestures in order to make sure that it will not be repeated, and yet if he happens to rediscover it he experiences in this a mental satisfaction which is equal to that which came when he first learnt how to avoid the unpleasant sensation. This desire to search for a sensation is very common in the subjects of tic, as is also the impulse to repeat a functional act which similarly brings a peculiar gratification with its performance.

That habit plays an important part in the genesis of tics is well recognised, but those, like Meige and Feindel, who have devoted attention to this aspect of the subject, have insisted on the importance of recognising that every stereotyped act is by no means necessarily a tic. There are many automatic habits and mannerisms that are not tics, for no matter how absurd they may be, as Séglas has shewn, they may be wanting in certain features that are necessary to make them tics. The outstanding point of difference is that there is no mental suffering with the stereotyped acts, so that there is neither the imperiousness which makes the performance of the movement irresistible, nor the mental anguish experienced in the struggle to repress it. These bad habits and mannerisms are most likely to occur when the person is abstracted, as is well illustrated by a number of tricks common to normal individuals which may become most evident during the heat of debate or the excitement of some game requiring physical skill, whereas directly the attention is called to the trick it can be suppressed. The reverse is true of a tic, for the more the individual is distracted and the mind concentrated on other matters, the less likely is it that the tic will occur, whereas, when he is unoccupied it will assuredly assert itself. Mimicry, whether voluntary or involuntary, may beget a tic, whilst another may be born of an idea. Although some of the world's great men have been *tiqueurs*, a weakness of will is the underlying condition that permits of a tic, and the subjects of the malady commonly reveal in a number of ways a "mental infantilism," on which Meige and Feindel lay stress, and the characteristics of which were clearly recognised by Itard many years ago.

ETIOLOGY.—The subjects of tic are "degenerates" in the widest acceptation of the term; and a tic can only take root in suitable soil, as has been well expressed in Meige and Feindel's words, "not all who would may tic" (Wilson). The following conditions, however, influence the genesis of a tic:—

Heredity.—Direct heredity can sometimes be traced, in which case the same tic may affect different members of the same family; thus, Sir W. Gowers mentions a man, the subject of a life-long tic of the face, who had two children similarly affected. In all such cases the influence of imitation is of course an important factor; so that although heredity supplies proof of a neuropathic soil, imitation has probably determined the particular neurosis in most cases of the kind. More often the neuropathic taint is revealed by other neuroses in the parents or collaterals, so that alcoholism, hysteria, chorea, epilepsy, neurasthenia, psychasthenia, mental disorders, and even organic diseases of the nervous system are discovered in them.

Sex.—Females were formerly supposed to be more commonly affected than males, but sex does not appear to exert any influence on the frequency of the affection, except that torticollis tic is more common in men than in women (*vide* p. 629).

Age.—The malady may become manifest at any age after early childhood when psychical function has developed, but does not occur in children before the age of three or four years, and is most common about the time of puberty. The form of the tic, however, has an influence in this respect, for torticollis most commonly appears in adult life.

Race.—There is reason to suppose that there is a tendency for the disease to become more frequent as civilisation advances, and it is questionable if races still in a primitive state have attained a degree of mental development that is competent to enable them to manufacture a tic.

Habit.—In many cases the condition originates in some trick or habit so often repeated that it ultimately gets beyond the voluntary control of the individual. In some the trick is acquired without obvious reason, in others it originates in some cause which induces either voluntary or reflex movements. The movements may, however, persist as a tic after the exciting cause has been removed; thus, blinking of the lids, caused by conjunctivitis, may continue after the conjunctival affection is cured.

Imitation.—We have seen that some cases probably owe their origin to imitation; not necessarily a direct imitation, but rather an irresistible tendency to give vent to pent-up feelings of unrest engendered by the witnessing of similar movements in another.

Lowered General Health.—A state of lowered health, variously induced, as by previous acute illness, overwork, growing too fast, and the like, commonly precedes the onset of the affection; anaemia, again, may play a part, though its occurrence does not seem to be so common as in chorea. On the other hand, it may be that without any obvious impairment of general health, influences have been at work which have had a depressing effect on the nervous system, such as shock from an injury or fright, and mental overwork. In tic, as in chorea, some cases appear to be school-made.

Reflex Irritation.—Conjunctivitis causing blepharospasm has already been mentioned. Obstructive diseases of the nose, including adenoids and the like, are commonly present, and occasion sniffing movements of the face which, voluntary at first, subsequently become automatic.

Optical Defects.—Though deserving of separate consideration, there is no essential difference between these conditions and those just considered. Of 49 cases of Sinkler's series, which were examined by de Schweinitz and Thomson, errors of refraction were found in 41 of the patients; in 2 there was conjunctivitis, and in six there were defects in ocular balance.

Season.—No relation between tic and season is known; the cases appear to be distributed in about equal proportions over the different periods of the year.

Relation to Sydenham's Chorea.—Cases of simple tic have followed chorea, and the former condition has been said to lapse into the latter. In Sinkler's series of 143 cases simple tic and Sydenham's chorea coexisted in 2 patients, and in 9 the latter condition preceded the former.

PATHOLOGY.—The tics must be classed among the so-called functional affections of the nervous system, for no structural organic defect has been determined in the brain or any other part of the nervous system to account for the clinical manifestations. From a review of the cases in which organic lesions have been described, it is evident that diagnosis was invariably at fault, and that certain other conditions, including reflex spasms, have been mistaken for tics.

The most generally accepted view of the pathogeny of the tics, held by Brissaud and strongly supported by Meige and Feindel, is that the affection is a mental disorder, a genuine psychosis. Grasset, however, supposes that in the cerebral cortex two groups of perception-neurons exist, an inferior of psychical automatism and a superior of voluntary or free cerebration. In support of the existence of inferior psychical neurons he adduces as evidence a number of phenomena of which dreams and acts of distraction may be taken as examples in normal individuals, nightmares, automatic writing, and spiritualism in the nervous, and somnambulism, catalepsy, hysteria, and hypnotism in the diseased. The various acts that occur in such circumstances differ from mere reflex ones in that they are characterised by spontaneity, but they have not the freedom which belongs solely to the superior psychism. This conception of the cortex permits Grasset to suppose that there are three groups of tics: (*a*) non-mental tics which can reproduce movements of bulbo-medullary origin; (*b*) polygonal tics which are related to the inferior psychical neurons, and which are co-ordinated and psychical but not mental; and (*c*) a group related to the superior or free cerebration to which the term psychical tic is most appropriately applied, and which depends on an actual idea. Grasset's view is not accepted by Brissaud, or by Meige and Feindel who consider that what he regards as a bulbo-medullary tic is identical with a spasm, so that the only justification for the term would be to designate a tic which had taken its origin in a spasm, which is of course admitted as one way in which a tic may be manufactured. The distinction which Grasset draws between a polygonal tic and a purely psychical tic is not admitted except that the movement which has its origin in psychical activity degenerates into a tic, the repetition of whose movements is a manifestation of polygonal activity.

Blocq and Onanoff have attempted to draw a distinction between conscious and unconscious tics; Letulle considers all tics unconscious, but Guinon contends that "convulsive tic" is conscious. These conflicting views are reconciled by Meige and Feindel by supposing that the subjects are not conscious of the tic at the moment when it occurs, although they are able to appreciate it when their attention is called to the matter.

SYMPTOMS.—The symptoms vary according to the form of tic that is under consideration; hence a classification is necessary, even if for no other reason than to facilitate description.

The classification of the tics set forth by Noir is as follows:—

1. *Simple Tic.*—This is essentially the condition to which the term "habit-spasm" has been applied by Sir W. Gowers, and which Weir Mitchell designates "habit-chorea." The latter name is singularly inappropriate, as tic and chorea are different affections; and the former appellation, though having more to justify it in that some cases of the affection arise out of a trick or habit, is by no means strictly applicable to all cases of simple tic, seeing that many can in no way be ascribed to any trick or habit which has subsequently become automatic, and is also best avoided so as to prevent confusion between a tic and a spasm.

2. *Convulsive tic* or *maladie des tics convulsifs* is an affection in which, in addition to the motor disturbances which characterise the simple tic, explosive utterances and imperative ideas form a prominent part of the clinical picture. In France this form of tic is commonly spoken of as Gilles de la Tourette's disease, as this physician has made it the subject of much careful study. In some of its aspects this condition is so similar to the malady known in America as the jumping disease of Maine, among the Malays as latah (*vide* art. "Latah" in Vol. II. Part II. p. 767), and in Siberia as myriachit, that in all probability these affections belong to the same category, or are even identical.

3. *Co-ordinated tics* or *tics coordonnées* differ from the simple tics in the character of the movement, which, instead of being a simple jerk or twitch, consists in some complex movement exactly similar to those executed in the ordinary routine of life, the only difference being that the particular act is repeated time after time without obvious reason. Noir has given an admirable description of this variety of tic.

4. *Psychical tic* or *tic psychique* is essentially of the same nature as a motor tic, the spasmodic movement being replaced by its psychical equivalent—an imperative idea; or, as Charcot expressed it, "Il y a des tics dans la pensée comme dans le corps."

To these it is convenient to add two other forms:—

5. *Torticollis tic*, also known as *Mental torticollis.* In this form the automatic movements are limited to the muscles of the neck as a rule. The affection was formerly regarded as a spasmodic neurosis, its identity with the tics not being suspected.

6. *Saltatory tic* is another example of a spasmodic neurosis that has hitherto been separately described, because its kinship with the tics was not recognised.

Facial Spasm.—Some regard this affection as yet another form of tic, but while recognising the frequency with which facial movements occur in the subjects of tic alone, or in conjunction with automatic movements in other parts, it has been thought better to give this affection a separate description and not to include it as a form of tic (see p. 638).

Although there are an infinite number of tics according as different territories of the body are involved in the automatic movements, they do not call for separate description.

1. **Simple Tic.**—In simple tic movements occur which seem voluntary, but may be performed with lightning-like rapidity, and are automatic. The condition has been regarded as a form of chorea by Weir Mitchell, on the grounds that children are usually affected; that it is associated, as a rule, with a lowered state of health, irritability, and nervousness; that sometimes the condition lapses into chorea; and, lastly, that the remedies most effectual in this condition are those which have been found of greatest value in chorea. Plausible as these arguments may appear, and although in some instances it may be difficult to be certain whether the case be one of chorea minor or simple tic, there can be no question that the two conditions are entirely distinct. Most modern writers are of this opinion; and even Sinkler, who supported Weir Mitchell's view that simple tic is a form of chorea, had to admit that some cases of this kind must be classed with the various forms of tic.

The movements in tic are characterised by the suddenness and lightning-like rapidity with which they are executed. The form which the movement takes varies not only in different persons, but also in the same person, so that two or more kinds of movements may be prone to occur, now one and then another manifesting itself; or again a certain movement may exist alone for a considerable time, when another is added, or it takes the place of that which previously existed. The interval between the succeeding movements varies considerably; usually a few minutes elapse, but at other times the movements follow each other in such quick succession as to become almost incessant.

The distribution of the spasms also varies considerably; but the face is the part most commonly affected, and here blinking movements of the eyelids are more frequent than any other form of spasm; another common movement is that of alternate elevation and lowering of the eyebrows. Contraction of the zygomatic muscles, with drawing of the angle of the mouth to one side and then to the other, is also frequent. Sudden jerkings of the head are common; the head is in consequence displaced backwards, forwards, or laterally, or the movement may be one of rotation. The platysma is not uncommonly involved in the spasm, either alone or with other muscles of the neck. The upper limbs are also frequently affected; the most usual movement is shrugging of the shoulders, but, in other instances, various other rapid movements may be met with, such as flexion or extension of different joints or rotation of the whole limb. Tic movements of the lower limbs are very much less common, though sudden extension with stamping of the feet and the like may occur; or sudden flexion of the leg or thigh at irregular intervals during progression, constituting what has been spoken of as "spring-halt tic."

Affection of the muscles of respiration is common, and not only is the rhythm of respiration thus deranged, but various grunts and other similar sounds are emitted; these may be laryngeal in character and of varying intensity, or like a sob or sniff. Any of these sounds may be frequently repeated, and may be associated with some disordered movement of the face, trunk, or limbs. Another troublesome symptom is cough, which may be hacking or very loud and oft repeated.

According to Prof. Osler, cases in which there is a sudden start, due to a general contraction which passes through the muscles of the trunk and limbs with electric-like rapidity, may be conveniently included as a generalised form of simple tic with the cases we are now considering. The muscular contraction is almost instantaneous, passing off with great rapidity, to be repeated in similar fashion from time to time. It is to this tic that Henoch applied the name electric chorea. The name is unfortunate for two reasons: the condition is not a form of chorea at all, but belongs to the family of tics; moreover, it has been given to a totally distinct affection met with in Lombardy, and known as Dubini's disease (p. 554).

The movements in simple tic are increased by any excitement, especially under observation, though the effort to concentrate attention during medical examination may lessen their frequency, an inhibition which never occurs in chorea. An effort of the will may or may not control the movements, and sometimes the patient is able to resist the impulse to perform some act for a time; though in the end he is obliged to yield, as it were to an imperative mandate. The movements always cease during sleep. The contractions are usually much more limited in range than is common in ordinary chorea; but when they continue for a long time they may spread, so as to involve several groups of muscles. The tic movements may cease after a time, especially under proper treatment; or again they may persist to be a source of great annoyance throughout life.

2. **Convulsive Tics.**—Syn.: "*Maladie des tics convulsifs*"; "*Gilles de la Tourette's disease.*"—We have no very satisfactory name for this affection; for neither "convulsive tic" nor "maladie des tics convulsifs" implies much more than occurs in simple tic; whereas, as noted on p. 619, in this form of tic explosive utterances and psychical phenomena are added to the spasmodic movements, and form a striking clinical feature.

The affection is a neurosis characterised by sudden lightning-like muscular contractions, which are repeated without the slightest rhythm, and may be either limited or generalised. In addition to this the victims are liable to involuntary explosive utterances, and in this event obscene words are sometimes emitted (coprolalia), or there is an irresistible impulse to repeat words or sounds (echolalia), or to imitate gestures (echokinesis). Psychical troubles also occur, in which obsessions and imperative ideas play a prominent part.

To Charcot, Gilles de la Tourette, and Guinon we are indebted for a clear account of this affection; and neurology owes much to the school of the Salpêtrière for the separation of this form of tic from the hysterias, and from a large class of indefinite conditions in which spasmodic movements play the chief part. Although Charcot inspired his pupils to do the excellent work which has placed this affection on so secure a basis as an independent condition, in fairness to Trousseau it must be stated that he clearly recognised the affection and indicated its chief features.

The Salpêtrière school and various other writers have regarded the muscular tics as dominant in the early part of the clinical history of these cases, and echolalia, coprolalia, echokinesis and the like as subsequent accretions. Other writers, however, accord the first place in the clinical picture to the psychical tics which may occur exclusively at the beginning of the malady. Tokarski, by attempting to separate the two groups, has gone a step further, and would reserve the name "maladie des tics convulsifs" for the former class, and "myriachit" for the latter. The name "myriachit," however, as I have already said, has been long in use in Siberia to denote an affection which is probably identical with the form of tic now under discussion; and it is questionable how far a subdivision such as that suggested by Tokarski is warranted in the present state of our knowledge, though Noir upholds the distinction.

As already observed, four groups of symptoms characterise the clinical picture of the affection—automatic movements, uncontrollable utterances, an impulsive tendency to imitate by acts or words, and a peculiar mental state in which obsessions and imperative ideas play an important part.

The movements are, as a rule, sudden and lightning-like; though it is said by Chabbert that in exceptional cases they may be quite slow and deliberate; in any case they are repeated in the most irregular way possible, without the slightest rhythm. The resulting movement may have no resemblance to any ordinary voluntary act, or, on the other hand, movements of defence, gestures of contempt or defiance, and the like, may be forthcoming.

The muscles of the face and neck are especially prone to participate in the movements, whereas the limbs are less liable to be affected, the lower extremities escaping more often than the upper; but no part of the body is exempt, and in some cases there is scarcely a muscle that does not participate at one time or another—though, according to Kahler, generalised movements are very exceptional. In the face the muscular contractions result in all sorts of grimaces and jerky movements; the eyes are rolled in this direction or in that, the forehead wrinkled, as in frowning or in the expression of surprise; now the eyes are too widely opened, at the next moment they are closed. The sniffing movement, so common in simple tic, is frequently present, and various contortions occur about the lower part of the face and mouth, the latter being at one time opened, at another time closed; or mastication movements, perhaps with grinding of the teeth, may be seen. A number of movements of the tongue, including protrusion and retraction, may occur. The head is jerked in every conceivable direction by the action of the neck muscles; not uncommonly it is suddenly retracted, and a quick lateral movement is added; or the chin may be brought forcibly in contact with the chest; so, too, various lateral and rotatory movements of the head occur from time to time, and the platysma is frequently seen in action.

One of the most common movements of the upper limbs is that of shrugging of the shoulders, which may be bilateral, or now one and then the other shoulder is suddenly raised and then let fall. So, too, other movements of the shoulders occur, as for instance a twist as if to get rid of some discomfort, or sudden forward or backward movements of this part. The whole arm may be raised, or any of its component segments may be moved in this or that direction, the fingers participating in the abnormal movements, all of which are executed in the same brusque, jerky fashion.

The muscles of the trunk may take part in the spasms, when various movements of propulsion, retropulsion, or lateral oscillations of the body are seen; and these, alone, or in conjunction with automatic movements in the lower limbs, may interfere with progression. Isolated movements of the muscles of the trunk are, however, uncommon, though the salaam movements thus occur in some cases.

Although the lower limbs are more commonly exempt than the upper, yet, when affected, the movements may be quite as severe as in any other part of the body. A stamping movement of the foot is not uncommon, and the limb may be suddenly extended as in the act of kicking. Sudden flexion of the leg and thigh may result in a spring-halt movement; or the patient may suddenly stoop, or jump, and so on. These various anomalies give rise to the most grotesque modes of progression, for instance, the ordinary manner of walking may be suddenly interrupted by a few dancing steps, or by a jump on both feet, or a hop on one, by a sudden kick forward of one or other limb, and the like. In a case recorded by Gilles de la Tourette, the patient ran rapidly for a short distance, then touched his knee and performed the most extraordinary movements with his arm.

The automatic movements occur in attacks, and are not more or less continuous, as is the case in chorea; in each attack several groups of muscles may be in action simultaneously; or the contraction of one group may follow that of another, in which case there is a curious tendency to preserve the same order in successive attacks. As I have said, the tic may be limited in distribution, and the limitation may be to one side of the body—an event which, however, rarely obtains in the case of the face in which the affection is usually bilateral.

The patient is fully alive to the occurrence of the movements; indeed, he may be painfully conscious of the ridiculous grimaces and contortions which he is making, nevertheless he has little voluntary control over the movements. A voluntary movement of some kind is a surer device by which to escape the spasmodic movement than a mere effort of the will, although a very powerful effort of the will may be successful; but so great is the effort required that it is most fatiguing to the patient, and may make him feel so ill that the imperative movement is preferable to the malaise. The devices adopted by choreics to mask the involuntary movements by the performance of some voluntary one in quick succession to that of involuntary initiation is seen in the subjects of tic also; but with so little success, as a rule, that the victims of tic are thankful for solitude as the only way of escaping observation.

The movements always cease during sleep, though Féré mentions a case in which attacks sometimes occurred during sleep, in his opinion probably as the result of dreams. It is interesting that, as in epilepsy and certain other spasmodic affections, the movements may be entirely in abeyance during the course of any febrile disorder. On the other hand, observation, excitement, or emotion of any kind induce the attacks. Disorders of digestion and the like may increase the tendency to attacks, which in women may be much more frequent at the monthly period than at other times.

Apart from any interference by the automatic movements, voluntary movement is not affected; such patients may be able to perform acts requiring the greatest delicacy of co-ordination, such as playing the piano, writing, and the like, without the least evidence of any defect; indeed Guinon refers to one patient who could juggle with knives without a slip or mishap. The absence of any alteration in the character of the handwriting may prove an especially valuable point of distinction between this affection and certain of the other motor neuroses in which the handwriting is notably altered. There may, however, be some difficulty in writing if tic of a severe type affect the arm.

Although the spasmodic movements are usually the first phenomenon of the malady, yet a tendency to explosive utterance may be added; or the initial phenomenon of the affection may be of this character, and it may remain at any rate for a long time the sole symptom.

The *exclamatory tic* consists in the habitual emission of some irrelevant word, or parts of words, or sentences, or again of some inarticulate sound, resembling, it may be, that of some animal. In some cases obscene or blasphemous words are ejaculated (coprolalia). This is rather common, and is most embarrassing to the unfortunate patients, as, despite all their efforts, such expressions may be emitted most inopportunely. Coprolalia may be manifested in two ways, both of which may occur in the same patient: the words are either spoken in a loud voice, in which case muscular shocks accompany their emission, or they are uttered in a low voice, and are then unaccompanied by muscular spasms. The spasmodic utterance may be single, or be repeated several times in succession. In a case recorded by Gilles de la Tourette, a youth thus afflicted received a sound thrashing from some other boys who thought that the ejaculation "*couillon*" was directed at them.

Another of these impulses is to repeat like an echo (echolalia). Words or names may be repeated thus, or the cries of animals and the like; or again, the words echoed may be those of the patient himself. The echo is usually the end of a sentence, especially of the last word. Various peculiarities are, however, met with in different cases. A word unfamiliar to the patient or the name of a person is especially apt to impress itself in this way; in any case the word or phrase is echoed time after time.

Again, movements or gestures may be reproduced (echokinesis). The mimicry may be of a facial grimace, or of some movement of the limbs or trunk; and this feature of the malady it is which bears the greatest resemblance to the tricks of the jumpers of Maine, to the "latah" or "latak" of the Malays (*vide* Vol. II. Part II. p. 767), and to the "myriachit" of Siberia (Russian "miriatchit" = "playing the fool"). The subjects of this malady are irresistibly impelled to imitate some sound, grimace, or act, although by so doing they may cause pain to others, or bring dire anger upon themselves. Thus, in the form of tic now under consideration—for whatever the form of the uncontrollable impulse, whether the ejaculation of words or sounds, the echo or the mimicry—there is the same powerlessness in the will to prevent it. The impulse may be circumvented by various dodges, but generally with little success; thus voluntary speech may ward off an explosive utterance; but too often some ungainly sound, or an irrelevant word, suddenly interrupts the sentence, or for an intended word of welcome to a visitor one that is highly uncomplimentary may be substituted.

The mental peculiarities, which may form part of the clinical picture, are most remarkable, and belong to the class of imperative ideas—the "idée fixe" or "obsession mentale" of the French. As with the motor tics so ideas may become automatic, and, though aware of the mechanical repetition, the patient cannot help it, and these ideas may lead to importunate, impulsive acts, either meaningless and absurd or even injurious. Sometimes a physical basis can be found for the fixed ideas and automatic acts, but by no means in all cases.

A common form is that of an agonising search for a word or name which when found is repeated time after time; or some word may incessantly obtrude itself on the patient even until he is compelled to vocalise it; the victim may loathe the word or dread it, and may attempt to evade it, but without success. The feature of some cases is an irresistible impulse to count a certain number, or tap a given number of times before some act, or a tendency to touch certain objects.

The condition known as *insanity of doubt* is an abiding uncertainty whether a particular act has been executed correctly or has been forgotten; or there may be an uncontrollable desire to question or analyse the most ordinary acts of everyday life.

We meet with one or other of the various 'phobias—notably agoraphobia, or the dread of walking in open spaces; acrophobia, or the fear of heights; and mysophobia, when the subject is afraid to touch certain objects (*vide* also art. "Neurasthenia," p. 749).

When these various manifestations are associated in a given case they usually appear in definite order; most commonly the automatic movements occur first, and are followed by exclamations, coprolalia, echolalia, echokinesis, and fixed ideas—in the order named. It is more common, however, to meet with convulsive tics alone, and, as I have said, the spasmodic exclamations may occur alone for a long time. Echolalia, coprolalia, echokinesis, and the imperative ideas may all be present in the same patient; but more commonly only one or other of these symptoms is met with, or at any rate various combinations of two or three of them.

These fixed ideas obviously border upon insanity; moreover marked loss of will-power is evident in these patients and a loss of the power of attention. They are unable to sustain a conversation coherently; mental application becomes practically impossible; judgment is worthless, and memory suffers, even to the point of inability to read.

It must be recognised clearly that, although convulsive tic and hysteria are totally different affections, there is no reason, as Guinon has insisted, why the two neuroses should not coexist, just as epilepsy and hysteria may be associated in hystero-epilepsy. Not only is the combination possible, but cases are actually met with in which convulsive tic and hysteria are found so interdependent that the presence of manifestations obviously hysterical, or the stigmata of this neurosis, does not exclude the coexistence of spasmodic tic.

3. **Co-ordinated Tics.**—As already said, this form of tic differs from the simple tics in the more complex character of the movement. The tic may be no more than some act of everyday life, repeated time after time without obvious cause. The case mentioned by Sir W. Gowers, of a boy who was in the habit of bringing both his arms forward and then stooping, belongs to this group; as does another, in which a girl, when walking, would make a half-turn, as if looking for something that she had dropped. Prof. Osler refers to a child who was recovering from what was supposed to be chorea minor, and who first smelt and then blew on anything she took into her hands. He also describes a boy, the subject of facial tic, who used to bite his middle finger hard, and at the same time press his index-finger against the point of his nose. Sinkler has also recorded some similar cases—such as that of a girl who, when walking, would stop, rub the toe of her shoe against the calf of her other leg, and then go on as if nothing had happened. He also refers to a boy who, as the result of imitating the motor-man of a trolley car, acquired the habit of standing with his back to a door and swinging round first one and then the other arm, and giving vent to an explosive utterance.

It happens not uncommonly that a tic of this kind may be controlled more or less by an effort of the will; so that as long as the patient keeps the matter in mind he may be able to prevent its recurrence, whereas as soon as he forgets about it the movement occurs automatically, perhaps without his perception of it. In such cases continual efforts to control the movements may result in a permanent cure. The more usual movements which characterise simple tic may coexist in the same individual with those of a more complicated character.

Noir has admirably described the co-ordinated tics which are met with in feeble-minded children. Among the more common movements in such cases are balancing, jumping, rolling the head from side to side, striking the chest with the chin, and hitting the head or chest with the hand. The latter habit is what Rubinovitch has called "Krouomania," and, like the other movements, these are sometimes repeated in a rhythmical manner; the balancing, nodding, and rotation movements are more especially likely to preserve a definite rhythm. Or the subject may stoop from his chair, stretch himself out prone on the floor, raise his arms above his head, and so forth, all of which actions are performed from time to time in orderly sequence. Prof. Osler refers to a case at the Institution for Feeble-minded Children at Elwyn, in which an idiot (known as the "Dervish"), after rising from the floor, balancing, and making a few sweeping rotations, would execute a series of revolutions, in which, with his arms spread out, and moving but little from one spot, he increased the speed of the rotations until he spun round like a top, and finally fell exhausted. Some of the cases included by Noir in his account of tic occurred in blind subjects, in whom quick movements of a finger in front of the eyes were especially common. Noir considers that there is probably some perception of light in such cases, and that the tic is the outcome of the habit of producing shadows by movements of the finger in front of the eyes.

4. **Psychical Tics.**—There is but little to add to what has been said already on this subject under the head of convulsive tics (p. 621). A psychical tic has a like origin, and is equivalent to the motor variety. In the latter a movement occurs which the subject is unable to resist; in the former an irresistible impulse compels him to emit a certain word, perform a certain act, count a certain number before doing something, and so on.

In some instances the imperative conception is generated by some external stimulus or antecedent thought, whereas in other cases it seems quite spontaneous, and cannot be traced to any such cause. At times after the discharge there is temporary relief, but in other instances the victim, after a long struggle between volition and the irresistible impulse, ending in the victory of the latter, is left much exhausted.

I have already indicated that the motor and psychical tics may concur in the same subject, and that the former may occur without the latter; it now remains to be said that psychical tics may occur without motor equivalents. Moreover, as a motor tic may be of no serious moment, so a psychical tic may be equally insignificant; imperative ideas may persist for years, and, although often a source of annoyance to the victims, no more serious mental disturbance may result; as was more especially emphasised in this country by Hack Tuke.

The thoughts which incessantly recur are usually both foreign and unwelcome, and words which the victim feels compelled to utter are neither in conformity with his custom nor what he wishes to pass his lips; so that if they escape it is in spite of his efforts to keep them back. Those who always touch certain objects which they habitually pass may do so automatically, as a sort of habit; or this proclivity may be related to the mania for counting (arithmomania); or the patients may be impelled to the act by a dread that something undesirable will happen if they omit their observance. This is a common enough trick of children, and one usually of no consequence, even if it persist, as is sometimes the case, until adult life. No better example of this condition is to be found than the well-known one of Dr. Johnson, who, as he walked along the street, used to place his hand on certain posts; if he missed one, he would turn back and perform the accustomed ceremony before proceeding on his way. The antithesis of this *délire du toucher* is the dread of touching certain objects (mysophobia). In the case of a doctor, referred to by Hack Tuke, the imperative idea was that something serious would happen if he trod on the divisions between the flags of the pavement, so that, although fully alive to the absurdity of the notion, he avoided them.

Arithmomania, or the irresistible impulse to count a certain number before doing a thing, is well illustrated in the case of a girl, recorded by Prof. Osler, who had to tap on the edge of the bed nine times before getting into it; to count a hundred after brushing her teeth; to knock three times on a window by the side of the door, and a similar number of times on the door itself, before she would unlock it. The tendency in these cases of arithmomania may be to make endless calculations, to count certain letters in words, the number of books on a table, and so on.

Then there is the state in which a certain name or word is sought for with an intensity that becomes painful, and in which, when found at last, the person is impelled to repeat time after time (onomatomania). It may also manifest itself as an attribution of a disastrous significance to certain words.

What has been called the "*insanity of doubt*," or "*maladie du doute*," forms an important class of imperative ideas, a good example of which is Dr. de Watteville's case, related by Hack Tuke. The patient always had a feeling that he had done a thing wrongly; for instance, after posting a letter or putting something into a drawer, he had grave misgivings as to the accuracy of what he had done. So, too, on alighting from an omnibus he would begin to think that he had left something behind him. Although fully aware that there were no grounds for his misgivings, yet the ideas tormented him.

Both agoraphobia and acrophobia are common forms of imperative ideas. The former condition makes it well-nigh impossible for the affected person to venture across any large open space owing to the fear that something untoward will befall him; in the latter state the subject finds it difficult to resist a strong impulse to throw himself from any height, in spite of his being fully aware of the unreasonableness of this impulse. Neither giddiness nor suicidal intent plays any part in such cases. The condition of mysophobia, already referred to, belongs to the same class of fixed ideas, many other varieties of which might be mentioned, though to do so would not serve any very useful purpose. The fundamental characters of imperative ideas have been sufficiently set forth to allow the reader to multiply examples for himself at will.

As was said when convulsive tic was under consideration, no hard and fast line separates the mental state characterised by fixed ideas from that of true insanity, so that at any time the border may be crossed, and what was formerly recognised by the patient as absurd now becomes a delusion in which he firmly believes.

5. **Torticollis Tic.**—Brissaud first pointed out that a type of torticollis to which he applied the term "mental" is a tic. This is the affection hitherto known as spasmodic torticollis or spasmodic wry-neck in this country.

Etiology.—The malady usually affects adults, but it also occurs earlier in life; Meige and Feindel met with a case in a patient aged eighteen years, and Raymond and Janet with one four years younger. A neuropathic or psychopathic heredity is the rule, and the subjects exhibit various traits of nervous instability, but it commonly happens that some exciting factor can be traced as having started the tic. Thus, it may follow on an injury, a pain in the neck, a sudden movement of the head and the like, and prominent among such exciting causes are disorders of vision, many patients regarding the strain consequent on working in a bad light as responsible. Strabismus and ocular palsies have also been effective in virtue of the lateral position that the head has assumed in consequence; in a case under my care the genesis of the torticollis could be distinctly traced to the frequent turning of the head to one side, which was made necessary by the relative positions of his seat in the room and the door at which people entered to consult him.

Clinical Features.—The head is either turned to one side by a series of rapid or slower jerky movements caused by clonic contraction of the neck muscles, or it is persistently deviated in one direction as a result of tonic contraction of the muscles. The latter condition is an example of what has been spoken of as a tic of attitude. The sterno-mastoid is the muscle most often involved, though rarely if ever alone, the upper part of the trapezius on the same side and the splenius and other retrocollic muscles notably of the opposite side sharing in the contraction. The platysma, scaleni, and deeper-seated muscles on the anterior aspect of the neck are rarely affected, which accounts for the position which the head usually assumes in these cases. The face is turned to the opposite side from the sterno-mastoid which is in contraction, with the chin protruded; but the ear on the side of the muscle that is in contraction is approximated to the shoulder on this side as the head is inclined in this direction. The more the upper part of the trapezius and the splenius are in action the more the head will be drawn backwards, and when these muscles are in action on both sides the head is drawn backwards instead of being turned to one side. It rarely happens that even when the anterior neck muscles are involved in the contraction, they dominate the position; but this sometimes occurs, in which case the head is drawn forward and the chin depressed towards the chest.

Whatever the movement may be, or whatever the position which the head assumes, it is always one which can be voluntarily reproduced, in conformity with what obtains in all tics. In the earlier stages of the affection the movement is usually sufficiently under voluntary control to make it possible for the patient to prevent it by an effort of the will. Later this may become impossible, but even now the movement may be arrested by some simple act such as placing the index-finger on the chin, a device that is common in such cases, and one of those which have been characterised as "efficacious antagonistic gestures" in the tics. In severe cases the muscles of adjacent parts may become involved, so that those of the face, upper limb, and back may all participate, the muscles about the shoulder being especially liable to be affected.

In cases of long standing it is not uncommon to find some hypertrophy of the sterno-mastoid on the affected side as a result of the long-continued excessive action of the muscle, whilst the opposite sterno-mastoid may be in a state of atrophy from disuse. There is nothing in this, however, to justify Féré's belief that unilateral atrophy may sometimes be responsible for the overaction of the opposite sterno-mastoid, as the condition is much more likely to be a consequence than a cause in the circumstances in which it occurs.

Some degree of aching or neuralgic pain in the back of the neck and head is not uncommon, but this is not sufficient to make it probable that the tonic form will be confounded with the painful affection known as rheumatic torticollis. The mental sufferings of these patients may, however, be considerable, for the affection is commonly attended with a great deal of distress.

In common with all tics the torticollis can be traced to a mental origin, and it will usually be found that a feeling that the movement must be performed precedes the actual movement. Moreover, there is the impression that if the movement is executed it will bring comfort, instead of which it is followed by feelings of remorse, and yet repression of it by an effort of the will results in a struggle, the effects of which are equally distressing and exhausting.

Treatment.—The majority prove rebellious to all forms of treatment, so that even surgical intervention may only bring partial relief. Others are more amenable and yield to measures that are less severe, and some undergo spontaneous arrest.

6. **Saltatory Tic.**—The essential feature of this form of tic consists in alternate contractions of the flexor and extensor muscles of the legs, which may be sufficiently violent to throw the patient repeatedly into the air,—even to the ground unless he happen to be supported. In these or various jumping or hopping movements the feet leave the ground; but when the muscular contractions are less severe the heels are drawn up, while the toes remain on the floor. The movements are induced directly the sole of the foot touches the ground, and are repeated in rapid succession as long as the erect posture is maintained. In the sitting or recumbent postures there is usually a complete cessation of the

movements. The distribution of the muscular contractions varies a good deal, so that, although the legs are chiefly affected, the muscles of the trunk, neck, face, and upper limbs, may participate.

Diagnosis of Tics.—Difficulties in diagnosis depend largely on the stage of the affection, for when fully established, so that besides muscular contractions the patient emits inarticulate sounds or articulated words or is afflicted with coprolalia or echolalia, there can be no difficulty in diagnosis; no other malady gives rise to this group of symptoms. Even short of this, however, the diagnosis may be established by carefully tracing the tic to its origin, when the idea of movement is found to precede its exteriorisation, whilst the mental satisfaction which follows the movement and the dissatisfaction which results if it is repressed leave no room for doubt in the matter.

For a long time the majority of these affections were classed as varieties of chorea; but a more detailed study of the symptoms in the different classes of cases has led to their proper nosological order. Much confusion, however, still exists, and cases which belong properly to the group of tics are regarded by some as varieties of chorea.

In *Sydenham's chorea* the movements are inco-ordinate, muscular weakness is common, and voluntary movements are greatly interfered with, even where no actual loss of motor power is to be detected, and it is quite exceptional in chorea to see the movements of ordinary life. Volition exercises less control over the movements of chorea than obtains in the case of the tics. Again, chorea tends to recovery, however slowly, whereas the tics are, as a rule, incurable; on this difference we may have to depend largely for diagnosis in some cases, as for instance when a chorea is not seen until a late stage when a few facial grimaces alone persist. For although it may be impossible from the history of former movements to decide whether the case be one of tic or chorea, yet the latter ends in cure, whereas no such result obtains in convulsive tic. Certain laryngeal sounds and grunts may result from the spasmodic muscular contractions in chorea, but they are never articulated words; and coprolalia, echolalia, and echokinesis do not occur. Furthermore, although mental defects occur in chorea they are quite different from those of tic; they are those of insanity with derangement of intellect, whereas in tic the intellect is preserved, although will-power is diminished or abolished, and the patient falls under the sway of fixed ideas.

There ought to be no possibility of confounding tic with the two conditions which have been respectively described as "*Electric chorea*" (Dubini's disease) and *Bergeron's disease.* In the former of these affections the involuntary spasms are accompanied by convulsive attacks which leave a certain degree of paralysis in their wake, and nearly always end fatally, it may be in a few days or after some months (*vide* p. 554.) Bergeron's disease, on the other hand, is a benign affection in which the spasm in the face may be arrested by pressure on the facial nerve;

indeed the whole of the manifestations of the affection may be dispelled by an efficient emetic.

Nor can there be much likelihood of confounding tic with *chorea major*, which is not a form of chorea at all but an hysterical manifestation, in which the attacks of involuntary movements last a long time, and do not in the slightest resemble the attacks in tic. Automatic or habitual movements of ordinary life are reproduced, patients may dance (chorée saltatoire), imitate the movements of a smith at his anvil (chorée malléatoire), and the like. Moreover, such attacks may be evoked by an emotion, by pulling a limb, by percussion of a tendon, and so on. Such patients either present hysterical stigmata,—such as concentric limitation of the fields of vision, or cutaneous anaesthesia, or a history of hysterical manifestations can be obtained.

It is rare for *hysteria* to reproduce the whole of the manifestations that make up the complete picture of the maladie des tics; we may meet only with the movements which simulate those of tic, whilst at another time involuntary vocal phenomena may be present. The laryngeal sounds in hysteria may be grouped around an expiratory spasm (cough), or an inspiratory spasm (hiccup). Such phenomena occur quite suddenly and disappear quite as unexpectedly after having, it may be, resisted all sorts of treatment. The patients are quite indifferent to their condition, and may cough away all day without shewing the slightest signs of fatigue. When, indeed, tic is simulated by hysteria, by its spasms only, the movements may be identical. In all doubtful cases search must be made for the hysterical stigmata; but of course an hysterical subject may become affected with the maladie des tics, when, according to Guinon, the evolution of the movements alone can indicate the diagnosis; if they cease, the case is one of hysteria; if they persistently resist all therapeutic measures, then true maladie des tics has been added to the hysteria.

A condition which may be confounded with tic is *paramyoclonus multiplex* (p. 649). However, the phenomena of imitation are absent, as are spasmodic utterances and mental troubles. Moreover, the face, which is affected in tic, usually escapes in paramyoclonus; indeed the lower limbs, which are least commonly affected in tic, are among the most usual seats of the muscular contractions of paramyoclonus. Again, the spasms are much more simple, picking out individual muscles or parts of muscles, and usually symmetrical muscles; they never reproduce the gestures or movements of ordinary life. The contractions are isolated, or follow each other in quick succession, and do not constitute attacks, as in tic; furthermore, they may be evoked by cutaneous stimulation, percussion of a tendon, and so forth.

It is conceivable that a tic of limited distribution might be mistaken for a *localised spasm* of a totally different kind. But such localised spasms are uniform, they are limited to a single muscle, to a group of muscles supplied by one nerve, or by nerves so closely related in origin as to be involved in the same lesion; and there is not the multiplicity and difference of movement which make up attacks of tic.

The condition which Letulle has designated co-ordinated tics (*tics coordonnées*) may be attended with movements which resemble the impulsive acts of the psychical tics; but such movements are much more under the influence of the will. They can commonly be traced to some habit, and may not amount to a disease; moreover, some of them differ from convulsive tic in that by force of habit the movements are unconsciously performed; spasmodic utterances and psychical disturbances again form no part of the clinical picture. The subject of certain forms of co-ordinated tic can arrest the movements by a voluntary effort (p. 626), and such arrest is not accompanied by the distress and subsequent exacerbation of the movements which follow temporary suspension of the movement in the maladie des tics; moreover, in some cases, habitual control of the movements in this way may bring about a complete cure.

Facial and other contortions sometimes originate in some slightly painful condition, especially in children in whom ulceration of the mouth or lips, for instance, may set up grimaces. Search should be made for any cause of this kind, either present or past, for under suitable discipline the one form of movement ceases; if in spite of all such control it persist, it is probably a true tic.

Of organic affections of the nervous system, characterised by involuntary movements, *athetosis* and *post-hemiplegic chorea* only need be considered, neither of which indeed is likely to cause much difficulty. In the case of athetosis the position of the hand, its continual instability, and the impossibility of its retaining any object in its grasp, are sufficient to distinguish the affection from tic; and many other points of distinction might be adduced. So, too, in regard to post-hemiplegic chorea, the limb affected by the motor disorder atrophies, and the tendon-jerks become exaggerated. Moreover, in neither of these conditions are there any of the symptoms of maladie des tics other than the motor phenomena; for, though mental deterioration may occur, it takes some form of idiocy, never the characters of the fixed ideas.

PROGNOSIS.—Among the chief of the several factors which influence prognosis is duration; the longer the tic has persisted the more difficult it becomes to control it. On the other hand, if attended to early, more especially if the patient be a child, it may be corrected; and the cure thus brought about may be permanent. Another important element in prognosis is the age at which the manifestations first occur, for, according to Sir W. Gowers, when it begins late in life the affection is generally permanent. It must be remembered further that not only may the condition be permanent in any given case, but also that the range of its influence may be so increased that ultimately many more groups of muscles may become affected than those originally concerned.

Once established the form known as convulsive tic usually becomes progressively worse, continuously it may be, or with intervals during which it appears to be stationary, to improve, or even to become arrested;

such intervals being followed in turn by periods of further increase in the manifestations of the malady. The affection has, no doubt, very exceptionally ended in recovery; yet even if so favourable a result be obtained, it must be remembered that it is peculiarly prone to relapses. In formulating a prognosis it must always be remembered that psychical disturbances may occur, if they are not already present; and that they may end in insanity. Psychical disturbances further influence prognosis in another important way, for so long as the tic manifests itself only by motor and verbal explosions, the duration of life may not be materially curtailed, if at all. It is otherwise, however, when obsessions and impulses harrow the patient, more especially if he struggle to resist them; for of necessity they gain the ascendancy, and, exhausting himself by his efforts at resistance, he may in despair seek release in suicide.

We must always be mindful that although in simple tics, such as those limited to the face, the prognosis is relatively good in that such patients may reach adult life without further change, yet a violent emotion, a heated controversy, and the like, may induce a crisis in which new phenomena appear and do not subsequently disappear. When the affection reaches its extreme degree the patient is unable to follow any occupation, or even to live an ordinary life with comfort.

TREATMENT.—It is of primary importance that every case of the kind should be carefully investigated with a view to ascertain any cause for the condition, and to remove or counteract its influence. Yet, even when detected, the removal of the cause will not, of course, alone suffice to restore tranquillity, sufficient as it may have been to induce the morbid state in the first instance. A tic is easily acquired, but difficult to conquer, more especially when of long duration; so that we have not only to remove the cause, but also to adopt measures to assist the patient to suppress the imperative desire to tic. Thus it is not only necessary to treat the condition of the nose, or correct the error of refraction, as the case may be, but also to employ measures to build up the system generally to fit the patient for the struggle to overcome the tic.

In the majority of cases, indeed, the general health is impaired, and there may be, as we have already seen, a certain degree of anaemia. Improved hygienic surroundings, fresh air, or change of air, together with good food and tonics, are called for. All chalybeates are of value, but no drug exerts so favourable an influence as arsenic, given in gradually increasing doses until its toxic effects become manifest. Cases which do not benefit when the drug is administered by the mouth may, nevertheless, do so when it is given subcutaneously, as was first pointed out a good many years ago by Weir Mitchell. Strychnine is also of advantage, as a substitute for the arsenic, from time to time. In the more aggravated forms of the affection a sedative line of medicinal treatment may be called for, in which case some form of bromide should be given contemporaneously with nerve tonics.

The drugs that proved of most advantage in Hammond's hands are conium and atropine, and he has recorded some striking instances of cessation of the tic under their influence. He considers that the value of these remedies is enhanced by combining them with moderate doses of a bromide. In the case of atropine, Hammond gives $\frac{1}{100}$th of a grain as an initial dose, and gradually increases it up to $\frac{1}{40}$th of a grain: in the case of conium he finds the fluid extract or conine equally efficacious, and, beginning with five minims of the former, he increases the dose by one or two minims a day until the tic ceases, or the toxic effects of the drug become manifest; in the latter case, if the tic still persist, he reverts to the original dose, and gradually increases the amount again as in the first instance.

Gymnastics are of advantage, as in true chorea; and massage is also beneficial.

The patient should be encouraged to control the movements by an effort of the will. In children the promise of reward may be thus effectual; but we earnestly deprecate any actual or threatened punishment if such voluntary effort fail, or indeed be not steadily made.

Rest in bed is highly beneficial in some obstinate cases, and the patient should never be allowed to work, or to follow exciting pastimes; his life, indeed, should be as quiet as possible, short of monotony, and should be so regulated that everything is done punctually.

Suggestion has a recognised place in the treatment of the affection, and hypnotic suggestion is of service in any hysterical complications that may exist.

The essentials in treatment, however, consist in encouraging the patient to try to control the movements and in teaching him exercises which bring into action the muscles affected and others which necessitate action on the part of the antagonists. All such exercises should be carried out in front of a mirror, and should be graduated so that each séance is of short duration to begin with, and gradually increased, but never continued sufficiently long to produce fatigue. The patient should attempt to remain as motionless as if about to be photographed, but only for a few seconds at first, the time being gradually increased. Moreover, the attempts at immobilisation should be carried out at first in whatever position the tic is least often manifest, so that it may be the sitting posture to begin with; but as soon as he has learnt to exercise control in this position for five minutes or so he is encouraged to attempt immobilisation when standing, with the head, trunk, and limbs in different positions, when walking and when talking, all in proper order.

Concurrently with these exercises of immobilisation he must be taught how to carry out movements by the muscles in the affected region in a slow, regular, and accurate manner. Such exercises must be as simple as possible to begin with, and must be carried out for a short time only, the séances being repeated four or five times a day at the same hours in front of a mirror and under supervision.

When these various measures fail in cases of torticollis tic relief may yet be obtained by surgical intervention if the muscular contractions be not too widespread, so that the more limited the number of muscles involved the better the chances of success. Excision of a portion of the spina accessory nerve on the side of the affected sterno-mastoid is a necessary part of the operation, but is not enough alone if anything more than temporary relief is to be looked for. To secure permanent benefit the excision of a portion of this nerve should be accompanied by section of the posterior primary divisions of the upper cervical nerves on the opposite side.

The operation must not, however, be allowed to induce us to relax those other means that we have at our disposal for the treatment of such cases, but rather regarded as a measure that is calculated to make it more possible for the other methods to be effective.

What I have already said as to the incurability of the form known as *convulsive tic* prepares us for the utter impotence of therapeutic measures in nearly all these cases. Drugs of a sedative class are useless ; they can do no more than suspend the movements temporarily while the patient is under their influence ; nothing approaching a permanent cure attends such measures. Various tonics are serviceable in a general way, for all measures which are calculated to build up the system, and at the same time are not too stimulating, should be employed ; the various preparations of iron are especially useful. Change of circumstances is indispensable in the treatment of these cases, and isolation is of still greater service ; of like importance is strict moral and physical discipline, and encouragement in those healthy pursuits or hobbies which prevent introspection. An intellectual patient may divert his mind with study ; in other cases some mechanical occupation must be followed. Useful adjuncts in treatment are massage, passive movements, gymnastics, hydrotherapeutic measures, and the like. Static electricity appears to do good when employed for a long time, especially when combined with hydrotherapeutic measures and isolation.

If the patient be so tormented by fixed ideas as to be practically mad, the strictest guard should be kept over him.

J. S. Risien Russell.

REFERENCES

1. Allard. *Thèse de Lyon*, 1886.—2. Amard. *Traité analytique de la folie et des moyens de la guérir*, Lyon, 1807.—3. André. *Mercredi méd.*, Paris, 1891, ii. 325.—4. *Idem. Les nouvelles maladies nerveuses*, 1892.—5. Aretaeus. *De causis et natura diutinorum morborum*, Lipsiae, 1828.—6. Armangué y Tuset. *Mimicismo o neurosis imitante*, Barcelona, 1884. 7. Axenfeld. *Traité des névroses*, 1850.—8. Battistelli. *Riform. med.*, Naples, 1896, xii. 746, 759, 770.—9. Beard. *Journ. Nerv. and Ment. Dis.*, N.Y., 1880, vii. 487.—10. Beer. *Wien. med. Bl.*, 1891, xiv. 493 ; 1898, xxi. 423, 440, 455, 473, 489.—11. Bernt. *Monographia Choreae St. Viti*, Pragae, 1810.—12. Blocq et Onanoff. *Maladies nerveuses, séméiologie et diagnostic*, Paris, 1892.—13. Bosredon. *Compt. rend. Acad. des sc.*, Paris, 1856, xliii. 1009.—14. Boswell. *Life of Johnson*, i. 497.—15. Bouchut. *Du névrosisme*, 1877, 171.—16. Bourneville. *Compt. rend. du service des épileptiques et des enfants arriérés de Bicêtre de* 1880 à 1882.—17. *Idem. Recueil des mémoires, notes et observations sur l'idiotie*, Paris, 1891.—18. Bourneville et Noir. *Arch. de neurol.*, Paris, 1893, xxv. 228.—19. Bouteille. *Traité de la chorée*, 1818, 3rd part, 329.—20.

Breitmann. *Thèse de Paris*, 1888.—21. Bresler. *Neurol. Centralbl.*, Leipz., 1896, xv. 965.—22. Briquet. *Traité de l'hystérie*, Paris, 1850, 317, 477.—23. Brissaud. *Journ. de méd. et de chir. prat.*, 1894.—24. *Idem.* *Leçons sur les maladies nerveuses*, Paris, 1895, 1. s., 502.—25. Brunon. *Normandie méd.*, Rouen, 1890.—26. Buccola. *Riv. sper. di fren.*, Reggio-Emilia, 1880, vi. 155.—27. Burot. *Rev. d'hypnol.*, Paris, 1888, iii. 132.—28. Buss. *Neurol. Centralbl.*, Leipz., 1888, vii.—29. Cadiot, Gilbert, et Roger. *Rev. de méd.*, Paris, 1890, x. 431.—30. Cantilena. *Speriment.*, Firenze, 1880, xlvi. 274.—31. Catrou. *Thèse de Paris*, 1890.—32. Croce. *Arch. di psichiat.*, Torino, 1890, xi. 181.—33. Chabbert. *Arch. de neurol.*, Paris, 1893, xxv. 10.—34. Charcot. *Semaine méd.*, Paris, 1886, vi. 364.—35. *Idem.* *Leçons du Mardi*, 1887-88, 65, 105, 294; 1888-89, 13, 464.—36. *Idem.* *Ann. méd.-chir. franç. et étrang.*, Paris, 1889, v. 1.—37. *Idem.* *Arch. de neurol.*, Paris, 1892, xxiii. 69.—38. *Idem.* *Cliniques des maladies du système nerveux* (Guinon), 1893, ii. 454.—39. Charcot et Magnan. *Arch. de neurol.*, Paris, 1895, 137.—40. Chauvreau. *Thèse de Bordeaux*, 1888.—41. Culerre. *Ann. méd.-psychol.*, Paris, 1890, 7. s., xi. 25.—42. Dagonet. *Traité des maladies mentales*, Paris, 1876.—43. Dana. *Text-book of Nervous Dis.*, 2nd ed., London, 1894.—44. Dejerine. *L'hérédité dans le système nerveux*, Paris, 1886.—45. Descroizilles. *Rev. mens. de mal. des l'enf.*, Paris, 1890, viii. 337.—46. Dupouy. *Congrès de Toulouse*, 1887.—47. Esquirol. *Dict. des sc. méd.*, Paris, 1818, xxiii. 507.—48. *Idem.* *Des maladies mentales*, Paris, 1838, ii. 315.—49. Féré. *Arch. de physiol.*, Paris, 1876, viii. 247.—50. *Idem.* *Pathologie des émotions*, 1892.—51. *Idem.* *Rev. de méd.*, Paris, 1883, iii. 769; 1894, xiv. 755.—52. *Idem.* *Twentieth Century Pract. Med.*, London, 1897, x. 710.—53. Ferrus. *Gaz. des hôp. de Paris*, 1838, xii.—54. Finley. *Montreal Med. Journ.*, 1896-97, xxv. 718.—55. Foucherand. *Thèse de Lyon*, 1883.—56. Foulhioux. *Recherches sur la nature et sur le traitement de la danse de Saint Guy*, Lyon, 1847.—57. Gilles de la Tourette. *Arch. de neurol.*, Paris, 1884, viii. 68; *Ibid.*, 1885, ix. 19, 158.—58. *Idem.* *Progrès méd.*, Paris, 1893, 2. s., xvii. 30.—59. Gintrac. *Nouv. dict. de méd. et chir. prat.*, art. "Face," 1871.—60. Gowers. *Diseases of the Nervous System*, 2nd ed., London, 1893, vol. ii. 633.—61. Grasset. *Anatomie clinique des centres nerveux*, Paris, 1900.—62. *Idem.* *Rev. neurol.*, Paris, 1902, x. 782.—63. *Idem.* *Leçons de clinique méd.*, 3. s., fasc. I., Paris, 1896 (Congrès de Grenoble).—64. Grimaldi. *N. riv. di psichiat.*, Naples, 1892, i. 9.—65. Guinon. *Rev. de méd.*, Paris, 1886, vi. 511; 1887, vii. 509.—66. *Idem.* *Dict. encyclopédique des sc. méd.*, Paris, art. "Tic," 1887, 3. s., xvii. 555.—67. Hammond. *Med. Rec.*, N.Y., 1892, xli. 236.—68. Hare. *Internat. Clinics*, 1897-98, 7. s., i. 186.—69. Hirschfeld. *Ueber maladie des tics convulsifs*, Berlin, 1891.—70. Hunter. *Glasgow Med. Journ.*, 1898, l. 435.—71. Ireland. *On Idiocy and Imbecility*, London, 1877.—72. Itard. *Arch. gén. de méd.*, Paris, 1825, viii. 385.—73. Jaccoud. *Cliniques de Laribisière*, Paris, 1873, 169.—74. *Idem.* *Gaz. des hôp.*, Paris, 1886, lix. 1185.—75. Jackson, Hughlings. *Med. Press and Circ.*, London, 1871, ii. 504.—76. Janet et Raymond. *Névroses et idées fixes*, 2 vols., Paris, 1898.—77. Janowicz. *Thèse de Paris*, 1891.—78. Jolly. *Charité-Ann.*, Berlin, 1892, xvii. 740.—79. Lannois. *Thèse d'agrég.*, 1886.—80. Laycock. *Brit. and For. Med. Rev.*, London, 1845, xix. 311.—81. Legrain. *Thèse de Paris*, 1885.—82. Leroux. *Thèse de Paris*, 1886.—83. Letulle. *Nouv. dict. de méd. et de chir. prat.*, art. "Tic," Paris, 1885, xxxv. 608.—84. *Idem.* *Gaz. méd. de Paris*, 1883, 6. s., v. 536.—85. Luys. *Gaz. des hôp. de Paris*, 1880, liii. 1114, 1131, 1154, 1187.—86. Magnan. *Recherches sur les centres nerveux*, Paris, 1893, 2. s., 116.—87. Meige. *Les tics*, Paris, 1905.—88. Meige et Feindel. *Les tics et leur traitement*, Paris, 1902.—89. Mitchell, Weir. *Amer. Journ. Med. Sc.*, Phila., 1876, lxxii. 321.—90. *Idem.* *Lectures on Dis. of Nerv. Syst.*, London, 1881.—91. Noir. *Étude sur les tics*, Paris, 1893.—92. Osler. *On Chorea and Choreiform Affections*, London, 1894.—93. *Idem.* *Arch. Pediat.*, N.Y., 1897, xiv. 1.—94. O'Brien. *Journ. of the Straits Branch of the Royal Asiatic Society*, Singapore, June 1883.—95. Peretti. *Berlin. klin. Wchnschr.*, 1885, xxii. 824.—96. Pitres. *Gaz. méd. de Paris*, 1888, 7. s., v. 145.—97. *Idem.* *Journ. de méd. de Bordeaux*, 1901, xxxi. 106; 1904, xxxiv. 665; 1905, xxxv. 589 (Congrès de Grenoble, 1902).—98. Railton. *Med. Chron.*, Manchester, 1886, iv. 24.—99. Remouchamps. *Bull. Soc. de méd. ment. de Belg.*, Gand, 1895, 24.—100. De Renzi. *Gazz. d. clin.*, Napoli, 1892, iii. 177.—101. Ribot. *Maladies de la volonté*, 1884.—102. Roth. *Histoire de la musculation irrésistible ou de la chorée anormale*, Paris, 1850.—103. Roubinovitch. *Ann. méd.-psychol.*, Paris, 1892, xvi. 467.—104. Séglas. *Les troubles de language* *chez les aliénés*, Paris, 1892.—105. *Idem.* *Rev. neurol.*, Paris, 1901, ix. 114.—106. Siebert. *Deutsch. med. Wchnschr.*, Leipzig, 1896, xxii. 808.—107. Sinkler. *Trans. Assoc. Amer. Phys.*, Phila., 1896, xi. 149.—108. Sollier. *Psychologie de l'idiot et de l'imbécile*, Paris, 1892.—109. Tamburini. *Riv. sper. di fren.*, Reggio-Emilia, 1880, vi. 126 ; *Ibid.*, 1889, xv. 445.—110. Taylor, E. W. *Boston Med. and Surg. Journ.*, 1898, cxxxix. 62.—111. Thomson. *Trans. Med.-Chir. Soc. Edin.*, 1889-90, ix. 143.—112. Thornton. *Med. Rec.*, N.Y., 1885, xxviii. 713.—113. Tilley. *Proc. Roy. Soc. Med.*, London, 1910, iii. (Clin. Sect.), 93.—114. Tokarski. *Recueil des travaux dédiés au professeur Kojevnikoff*, Moscow, 1890.—115. du Tornéry. *Les maladies nerv. pendant l'antiquité gréco-romaine*, 1892.—116. Troisier. *Dict. encyclop. des sc. méd.*, Paris, art. "Face," 1877, 4. s., i. 18.—117. Trousseau. *Clin. méd. de l'Hôtel-Dieu*, Paris, 1868, iii. 257.—118. Tuke, Hack. *Brain*, London, 1894, xvii. 179.—119. Vanlair. *Rev. de méd.*, 1889, ix. 1.—120. Venturi. *Psichiatria*, Naples, 1884, ii. 39.—121. Vizzioli. *Giorn. de neuropatol.*, Naples, 1884, ii. 42.—122. *Idem.* *Boll. di clin.*, Milan, 1897, xiv. 193.—123. Voisin. *Mém. lu à l'Acad. roy. de méd.*, Feb. 24, 1843.—124. Wilson, S. A. K. "The Tics and their Treatment," *Transl. of Meige and Feindel's Book*, London, 1907.—125. Wilson. *Arch. Pediat.*, N.Y., 1897, xiv. 881.—126. Wille. *Monatschr. f. Psychiat. u. Neurol.*, Berlin, 1898, iv. 210.

J. S. R. R.

FACIAL SPASM

Synonyms.—"*Hemi-spasm of the face*" ; "*Facial tic*" ; "*Mimic spasm*" ; "*Convulsive tic*" ; "*Histrionic spasm of the face*" ; "*Tic non-douloureux*"

By J. S. Risien Russell, M.D., F.R.C.P.

Definition.—This is an affection in which spasms, usually clonic and sometimes tonic, occur in the muscles supplied by the facial nerve, usually on one and sometimes on both sides ; in either case the spasm may occur in all of the muscles, or only in those supplied by one or more branches of the nerve. The spasm may be either primary or secondary to paralysis, and in the former case it may be either what has been called idiopathic or due to some organic disease.

Causation.—It is very rare to meet with any direct hereditary transmission of this affection, though instances have been recorded by Sir W. Gowers, Piedagnel, and Rosenthal. A neurotic heredity, as manifested by epilepsy, insanity, and the like, in other members of the family, is sometimes observed. Possibly more than one member of a family may be affected, as observed by Blache, Delasiauve, Gintrac, Rosenthal, and others ; but most cases of this kind are open to the fallacy that probably a true tic (p. 614) and not a spasm was the condition present. Indeed, it is unfortunate that the term tic ever became applied to facial spasm, as the two affections are widely different, and ought not to be confused.

Women are much more frequently affected than men ; and the so-called idiopathic form of the affection usually occurs in advanced life, being rare in persons younger than about forty-five ; most cases occur between this age and sixty, though some may begin even later ; it is said that exceptional cases have occurred in people even as young as twenty. A lowered state of general health and, consequently perhaps, of the nutrition of the nervous system, is supposed to favour the occurrence of the affection, but in reality the patient's general health is usually very good. According to some authors, the most common cause of the condition is some mental emotion, such as a sudden shock, great grief, or long-continued anxiety, but no doubt some of the cases have been examples of tic that has been confused with facial spasm. One of the most striking instances of this effect of shock is the case mentioned by Romberg, in which facial spasm appeared in a woman on the sudden death of her husband. Direct irritation of the facial nerve has been proved in some cases in which tumour, aneurysm, or meningeal thickening has been discovered, and lesions of the petrous bone or middle ear may produce a like effect, as may parotid abscess and other lesions in this region.

Other authors insist on the importance of reflex irritation at some point in the bulbo-reflex arc as the essential cause, more especially irritation in some part of the distribution of the fifth cranial nerve, to which, in reflex action, the facial is specially related. The irritation may be in a carious tooth, and, according to Lannois and Porot, it is rare to meet with cases in which there has not been severe toothache or neuralgia, or failing these a history of injury to the face. Some disorder of the eye may be the cause, and Bethremirux refers to cases in which with lacrymal lesions the spasm has been relieved by catheterisation; and it is said that the orbital region has usually been involved when wounds of the face have had this consequence. Other such sources of irritation are otitis media, caries of the petrous portion of the temporal bone, pressure on the fifth nerve by a new growth or aneurysm at the base of the brain, abscess in the parotid region, an inflamed gland in the neighbourhood of the stylo-mastoid foramen, and the like. Facial spasm has followed migraine of many years' duration, and neuralgia has sometimes been the precursor of the facial spasm; wherein we are reminded that attacks of tic douloureux are often accompanied by contraction of the facial muscles. Exposure of the head and face to intense cold is regarded by some as a frequent cause, acting no doubt reflexly through the afferent channel of the fifth nerve.

Again, it has been supposed that irritation at a distance may lead to a like result; even uterine or intestinal irritation has been blamed. The condition has been known to come on during pregnancy, and to be recovered from on delivery; moreover, many cases begin about the climacteric period.

It is said that facial spasm may come about by repeated performance of some facial movement, which would seem to make it allied to tic, but in reality the two affections are widely different.

Finally, facial spasm may be brought about as the result of a tumour at the lower end of the pons, which irritates but has not as yet produced sufficient pressure on the facial nucleus or the emergent fibres of the nerve to cause paralysis, or in the region of the facial centre in the cerebral cortex. Not only in progressive lesions, such as tumours and abscess, in this situation has facial spasm been met with, but also with stationary lesions, such as a patch of softening, as in Berkley's case; or where there has been reason to suspect injury to the cortex during delivery. Blows and accidents to the head have seemed to be the cause in some cases, presumably as a result of contusion of the cerebral cortex; and Féré mentions a case in which a depression of the cranium in the region of the angular gyrus was attended with this result.

Pathology.—The failure to discover structural changes in cases which have accordingly been called idiopathic causes the pathology of the affection to be shrouded in much obscurity. Nevertheless, as facial spasm may be a symptom of organic disease, we are able to learn enough from cases of this class to make conjecture not only permissible but also profitable in our inquiries into the possible pathology of the idiopathic cases, whilst the definite relation which appears to exist between the malady and some source of reflex irritation teaches a lesson that is still more useful.

Although organic lesions have sometimes been found in the cerebral cortex, it cannot be said that these supply a satisfactory explanation of the mode of spasm in the majority of cases. It is true that clonic spasms result from cortical irritation, but that such spasms should be repeated time after time and year after year, and be confined to the anatomical distribution of a single nerve without further spread, and without causing even temporary paresis following the spasm, is contrary to the best established doctrines of discharging lesions of the cortex. An attempt has, naturally, been made to explain facial spasm on the hypothesis that it is a form of tic, but without success, for there are good grounds for regarding the condition as a genuine spasm, quite distinct from the psychomotor act which characterises a tic (see p. 614).

In these circumstances, the most probable view is that advocated in the first edition of this work, namely, that the facial nucleus in the pons is the starting-point of the discharges which result in the spasm. In short, the great weight of evidence is that the affection is a pure bulbo-reflex spasm, in which the cerebral cortex does not take any part, and in which the efferent channel is the seventh nerve and the afferent probably the fifth.

Symptoms.—All degrees of severity of spasm of the facial muscles are met with, both as regards intensity of the contractions and the range of their distribution; but in the typical and most prevalent form there are rapid clonic contractions which begin perhaps more or less slowly, and then the successive contractions are repeated more and more rapidly until, at the height of the paroxysm, the clonic passes into tonic contraction of the muscles. As the attack passes off, this form of spasm gives way to clonic jerks, which, as a rule, are slower and of greater excursion than those which precede the tonic stage. The clonic spasms may be repeated so rapidly as to have a quivering character, more especially as the paroxysm is reaching its height; the terminal clonic spasms, on the other hand, become separated by longer and longer intervals, and at the same time the excursions may be smaller and smaller, until they finally cease. Yet instead of the excursions of the final contractions becoming lessened in range, the range of the last contraction may be as great as any during the whole of the paroxysm.

Instead of the definite order of events just described, the spasms may involve the muscles in more or less instant tonic contraction—clonic spasms being either absent or of such small range as to be scarcely detectable; and there is a more or less abrupt termination equally wanting in distinct clonus.

All the muscles supplied by the facial nerve may be involved in the spasm, but the orbicularis palpebrarum and the zygomatici are more commonly affected than the others; even where all the muscles supplied by the facial are involved, these usually suffer in greatest degree, though, exceptionally, the levator labii superioris is more markedly affected than are the zygomatici. At times the onset is so deliberate as to allow us readily to note the muscle in which the spasm begins, whilst in other instances the onset is so rapid that it is practically impossible to say which muscle is the first to contract.

Instead of all the muscles supplied by the facial nerve being so affected, the spasm may be limited to certain of them, in which indeed it may be only very slight. Even where the spasms are subsequently general they are at first usually local, and in no part is it initiated more commonly than in the orbicularis palpebrarum, the resulting contraction being known as blepharospasm. Here the spasm may begin and end without extending its range. Where the orbicularis is thus affected the earliest manifestations may be so slight as to resemble that quivering of muscles popularly spoken of as "live blood."

Blepharospasm has received a good deal of attention, especially from von Graefe, and is the most important of the partial forms of facial spasm. There may be either tonic or clonic spasm of the eyelids, the latter being sometimes spoken of as "Nictitating spasm," and consisting in rapid winking movements of the lids in which both the upper and lower participate; or the twitching may be limited to one lid, in which case the lower is the more commonly affected. This form may occur, without obvious cause, as a phenomenon in the symptomatology of tic or as an hysterical manifestation. In the tonic variety there is persistent closure of the eyelids, the paroxysms lasting a few minutes, or it may be for hours; indeed, in rare cases the period has been extended to weeks or months. This form of spasm is usually reflex, the source of irritation being often in the ocular branches of the fifth, the condition being commonly associated with photophobia; but blepharospasm is sometimes associated with trigeminal neuralgia when the source of irritation appears to lie in some other branch of the fifth nerve. The spasm may be arrested by pressure on the points which correspond to the tender spots in trigeminal neuralgia, and nowhere is this so effective as at the infraorbital foramen. With removal of its cause blepharospasm may yield; in other cases it persists, and becomes a source of much annoyance.

When spasm, partial in distribution, affects the lower part of the face, in the distribution of the malar and labial branches, a unilateral or, exceptionally, a bilateral convulsive grin results, which has been variously designated "risus sardonicus," "risus caninus," or "cynic spasm."

When all the muscles supplied by the facial are affected, each paroxysm is separated from the next by an interval which varies considerably in different cases and in the same patient. Several hours may elapse without an attack, or they may be repeated in quick succession every few minutes. The duration of each paroxysm may be only a few seconds, or it may last several minutes.

Instead of the paroxysms of clonic and tonic spasms already described, there may sometimes be single momentary contractions repeated at short and more or less regular intervals, and resembling the results of excitation of the facial nerve by single induction shocks. Or at other times these isolated spasms may be repeated at irregular intervals.

The effect of the spasm in its typical form is to cause partial or complete closure of the eye, drawing of the angle of the mouth outwards, and intensification of the naso-labial groove. The forehead is wrinkled and the eyebrow raised or lowered according to the preponderance of muscular action. Or the angle of the mouth may be depressed while the skin of the chin is puckered and drawn upwards.

The muscles may be affected on one side only, or the spasm may be bilateral; but even in the latter case it usually begins on one side, and is commonly so limited for some time; moreover, even when it subsequently spreads to the opposite side, the side worse affected during a paroxysm is that in which the spasm usually begins.

It is worthy of note that although the spasm may be otherwise unilateral, yet orbiculares, corrugatores, or frontales may be in action; when the spasm is limited to the muscles of the upper part of the face both orbiculares or corrugatores are frequently involved, but it is unusual for the muscles of the lower part of the face to be affected equally on the two sides, except as part of a general spasm in which the whole of both sides of the face are affected. Then, again, the spasm is usually more intense on the side first affected; though this is by no means an invariable rule, for ultimately the spasm on the side more recently affected may become more intense; moreover, some paroxysms may begin on the side originally affected, and others on the side more recently involved. Both of these results I have observed in a man in whom the spasms were limited at first to one side of the face, and subsequently involved the other also; in him the spasms ceased for a time under treatment, when those on the side of the face first affected gave way before those on the opposite side. Moreover, when the spasms occurred on both sides of the face they would begin sometimes on one side, sometimes on the other.

Any emotional excitement tends to evoke the spasms, or to make them worse; as may any movements of the face, as in speaking, laughing, chewing, and the like. In an early stage of the affection, or when recovery is taking place, the spasms may arise only under such disturbing influences. On the other hand, the more quiet the patient the less the liability to spasm; mental and physical rest being both operative towards this end. Cold, which apparently may call forth facial spasm in some cases, is also a potent factor in increasing it when already in existence; warmth, on the other hand, has a beneficial effect. In a bright light, again, the spasms, as a rule, become very much worse, whereas by darkness they are calmed.

Evidence that the stapedius muscle is involved in the spasm is very exceptionally met with; but in cases of tonic spasm of the orbicularis palpebrarum, a noise in the ear has continued, not only while the blepharospasm lasted, but also for some time after cessation of all visible spasm. As Sir W. Gowers reminds us, many people can produce a rumbling noise in the ear by strongly contracting their orbiculares, especially if, at the same time, they attempt to turn the eyes upwards. In a case recorded by Moos, giddiness and conjugate turning of the eyes to the right during the height of the attack of spasm were regarded as the result of lowered pressure in the labyrinth and semicircular canals, due to spasm of the stapedius.

The spasm sometimes spreads beyond the domain of the facial nerve, in which case it may not only involve muscles supplied by other cranial nerves, but others also, which receive their supply from nerves of spinal origin. In consequence of this spread of the spasm the muscles of mastication, tongue, or eyes may be included; or the spasm may be of still wider range, including muscles of the neck and upper extremity; but it is more especially the muscles of the tongue, the masseters, and sterno-cleido-mastoids that are thus affected; implication of the ocular muscles is very exceptional. Whether such cases belong to the group under discussion is, however, open to question, for a feature of facial spasm is the strict limitation of the contraction of the muscles supplied by the facial nerve.

It is rare for the muscles of the palate to be involved in the spasm, though some well-marked instances of this have been recorded by Gowers, Leube, Oppenheim, Schütz, and others. Bilateral as well as unilateral spasm of the palate has been observed in conjunction with unilateral affection of the face; but in a case of the former, observed by Sir W. Gowers, the spasms of the face and those of the palate were not synchronous.

When there is no organic disease behind the paroxysms, no loss of motor power is met with in the muscles of the face, though of course all voluntary facial movements may be hampered by the spasms. The nerves and muscles usually respond normally on electrical excitation, except of course in organic disease of the nerve itself.

Instead of clonic contractions of the muscles of the face, the condition may be one of tonic spasm; but this is quite exceptional as an independent condition, though cases of the kind have been attributed to cold.

It is usually met with in paralysis of the muscles of the face, whether the result of a peripheral lesion of the facial nerve, as a part of a hemiplegia of cerebral origin, or as part of some general condition, such as paralysis agitans and tetanus, in both of which affections the condition is bilateral. Similar spasm may also be met with as an hysterical manifestation. The results of such spasm depend on whether the condition be bilateral or unilateral; but in any case the face may feel stiff, and all voluntary movement of the parts concerned in the spasm may be considerably interfered with. The palpebral fissure is narrowed on one or both sides, there is elevation of one or both eyebrows, and the angle of the mouth is either drawn to the side of unilateral spasm, or, if the spasm be bilateral, the condition may result in the "risus sardonicus."

Sensory symptoms are exceptional, though subjective and objective defects have been observed, both in regard to common sensibility and the special senses. As we have already noted, pain may be present, not as a part of the affection itself, but connected with some influence producing the facial spasm; the pain being referred to some part presided over by the fifth cranial nerve. In rare instances tender points have been present in some part of the distribution of this same nerve. Blunting of sensibility of the skin of the face has only been met with where some destructive process was present in connexion with the fifth nerve. Besides the subjective auditory phenomena, sight has been affected; it is conceivable that sparks, or similar subjective phenomena, may appear from sudden and powerful contraction of the orbicularis palpebrarum compressing the globe. No instance of subjective sensations of taste appears to have been met with, though loss of taste on the anterior part of the tongue has been present where there was reason to suppose that the nerve had been damaged.

Diagnosis.—Two main problems present themselves for our consideration in a case of supposed facial spasm: the one is, whether the genuine condition is present, or only some affection which simulates it; the other, whether, the case being one of true facial spasm, it is idiopathic, or dependent on some organic disease.

The first problem does not, as a rule, cause much difficulty. In *hysteria* there may be either a quivering movement of the muscles, which does not in the least resemble true facial spasm, or there may be tonic spasm, which affects all of the muscles of the face or chiefly the orbicularis palpebrarum. A common type is the glosso-labial spasm described by Charcot. There is no uniformity of spasm as in the affection under consideration, and even when clonic there is commonly a good deal of contracture associated. Moreover, hysterical stigmata usually make the diagnosis easy.

It is conceivable that cases of *chorea*, in which there has been but little affection of the limbs and a good deal of spasm of the face, may give rise to errors in diagnosis; but attention to the character of the spasm, and careful watch on the limbs, in which some spasm will always be noted in chorea, should prevent mistakes. There are many features which distinguish the affection from a *tic*. The movements are very sudden and rapid, resembling the twitchings evoked on electrical excitation rather than the co-ordinate purposive act expressive of any sentiment such as occurs in tic. The movements are strictly limited to the muscles supplied by the seventh nerve or one of its branches; they may continue during sleep, are beyond the control of the will, and neither preceded by any impulse nor followed by that satisfaction so characteristic of a tic.

A more difficult question, and certainly one of more importance in some of its aspects, is whether we are dealing with a case of idiopathic facial spasm, or with a result of organic disease. It is well first to exclude spasm resulting from facial paralysis, due either to an affection of the nerve or forming part of a hemiplegia. Here, besides the occasional attacks of spasm, there is the history of the previous attack of facial paralysis or of hemiplegia, and the detection of weakness of the movements of the affected side of the face, together with permanent contracture and overaction. Moreover, in the cases which follow hemiplegia, it may be possible to detect some evidence of weakness or contracture of the limbs of the affected side; or the tendon-jerks, or plantar reflexes, may afford us information of a valuable kind. In short, this form of facial spasm is not, as a rule, difficult to discriminate from the idiopathic variety. The cases which may present the greatest difficulty are those in which the facial spasm may be symptomatic of grave organic disease of the cerebral cortex in the region of the facial representation, in connexion with the facial nucleus in the pons, or the result of pressure on the nerve itself after its emergence from the pons, by some kind of tumour. Weakness of the facial movements, coming on concomitantly with the spasm on the same side, or progressively developing afterwards, strongly suggests organic disease; again, the detection of any other signs of organic affection in the parts presided over by others of the cranial nerves adds weight to the probabilities otherwise suggested. It may not be possible to say whether the organic disease, of which the facial spasm is a symptom, be situated in the cortex or in the pons, or whether its influence be exerted on the nerve after its emergence from the pons; but when only some of the muscles supplied by the nerve are affected, such as the zygomatici, then the cortex is the most likely seat of the generating lesion.

Prognosis.—In the absence of evidence of organic disease of which the facial spasm is but a symptom, there is nothing in the nature of the affection to jeopardise life; although indeed it robs the sufferer of much of the pleasure of life. There are few affections which cause their victims more annoyance than facial spasm, and in some cases the subjects become so worried by it that life to them scarcely seems worth having. Trivial as the affection may be, so far as any direct influence on the duration of life is concerned, it is usually most intractable; it either resists all treatment for years, or indeed to the end of the patient's life, or presents intermissions which may last for variable periods. Such cessations may be spontaneous, or may appear to result from treatment; in either case, after a variable interval, the spasm usually returns, and may eventually persist without any such intermissions. The earlier the case is seen, and the more definitely the spasm can be ascribed to some reflex source of irritation which is removable, the better does prognosis become; the longer the spasm has been in existence, and the more obscure its possible cause, the more unfavourable is the outlook. It must not be supposed, however, that because treatment is begun early, and a definite reflex cause of the spasm detected and removed, that a cure is confidently to be expected; for even in these cases the results are oftentimes most disappointing.

Treatment.—In the exceptional cases in which facial spasm is but a symptom of organic disease, such disease must be treated on the lines which are known to lead to improvement in the particular affection. Apart from this, and in the majority of cases of facial spasm, a careful search must be made for any possible sources of reflex irritation, and suitable measures adopted for their removal or suppression. Decayed teeth should be attended to, and where necessary extracted; the eyes should be examined, more especially with a view to the correction of any errors of refraction. In the case of exposure to cold the side of the face should be kept warm; and the side of the head and face may be bathed with hot water at frequent intervals—measures which may be supplemented by free diaphoresis.

Having removed or neutralised any possible cause, we have next to turn our attention to the hope of finding some treatment to render the neurons of the facial nerve less prone to independent discharge. There are two chief lines on which treatment may be pursued with a view to this end: the first is to try to calm the unstable centres by means of drugs which are known to exert a sedative action on the nervous system. Probably every drug of this kind has been thus tried, yet few of them have been the means of permanently arresting the spasms in more than a single case; and for the majority of them we cannot claim even this meagre measure of success. Morphine, alone or combined with atropine, has done most service, but the evil consequences which may follow the use of the drug are more mischievous than the malady itself. Bromides seem to have little influence on the spasm, if any; but success has been claimed both for conium and gelsemium.

The alternative line of treatment is to attempt to remedy what is possibly the fundamental defect in these cases, if our view of their pathology be correct; for, in the belief that we have to deal with an instability of the discharging nerve-centres, and supposing further that this instability largely depends on nutritional changes in the cells of the affected neurons, we may try to improve the nutrition of the neuron in the hope of rendering it more stable. With this object in view, nervine tonics have been tried extensively, notably arsenic, zinc, strychnine, and silver; but unhappily with little better result than with the sedative drugs. Iron and quinine are useful general tonics in these cases, and by building up the system generally we hope to influence, secondarily, the nutrition of the neurons which are at fault. This being the case, it is especially important that if the spasms appear to have been induced by depressing emotion, measures should be adopted to remove any such unfavourable influence as far as possible; and where any general impairment of the patient's health exists, we must endeavour to rectify the defect. This end may be best attained by complete isolation of the patient, absolute physical rest, as much mental calm as can be secured, forced feeding, general massage, and faradism.

Electricity.—Except when used to the body generally, as indicated above, faradism is contra-indicated; but some good may be expected from the use of galvanism locally. This mode of current should be employed in an uninterrupted or "stabile" manner, and only the gentlest currents, carefully measured by a galvanometer, ought to be used. A current of not more than two milliamperes should be employed, and care should be taken to introduce it gradually, and to shut it off as gradually. On the whole, it will be found of most advantage to make use of the sedative action of the current, by placing the anode over the trunk of the nerve in front of the ear, or on its branches in turn, while the cathode is placed at some indifferent point, such as the back of the neck. The method may be supplemented by placing a pole on each mastoid for a few minutes towards the end of each application. This form of treatment may be judiciously combined with massage of the face; and this latter means, and facial gymnastics, are especially useful in those cases in which the facial spasm arises out of some habitual movement.

Counter-irritation by means of a blister placed behind the ear, on the affected side, usually appears to diminish the amount of spasm, at any rate for a time; but no permanent good can be expected. So, too, counter-irritation of the cervical spine has been tried, and is said to have been successful in 1 case. When there are tender points in the distribution of the fifth cranial nerve, pressure on which stops the spasm, Sir W. Gowers recommends hypodermic injection of cocaine at these spots; others recommend blistering, or the application of the thermo-cautery to the painful points. Freezing of the cheek, by means of a spray, for a few minutes every day, or every other day, has been recommended by Weir Mitchell as affording relief in some cases.

Another and the most recent method of treating the affection is by the injection of absolute alcohol, as first recommended by Schlösser and subsequently practised by Brissaud and others. A solution of 60 to 80 per cent of alcohol is injected in front of the mastoid apophysis, at the exit of the facial nerve from the stylo-mastoid foramen, by means of a fine platinum needle introduced to a depth of about 1½ cm., very gradually until paresis of the face appears. The paresis passes off after a variable period, but has even persisted for several months, while cessation of the spasm has been secured for as long as two years or more; but it would seem unlikely that a radical cure could be effected in this way.

Stretching of the facial nerve is a procedure which at one time gave hopes of relief for the unfortunate victims of facial spasm, but these hopes have not been realised. To have any effect on the spasm the stretching must be powerful, and this is, of course, followed by paralysis of the muscles formerly in a state of spasm; experience has also shewn that, with returning power in the facial muscles, spasm once more re-asserts itself. The statistics of Mr. Godlee and of Keen both shew how little is to be expected from this method of dealing with facial spasm. The former observer collected 13 cases thus treated, and in only 1 of them was spasm absent two years after the operation; whilst of 20 cases, subsequently collected by Keen, only 1 was free from spasm longer than six months after the operation. Schott, after excluding all cases inadequately reported, and such as had not been observed sufficiently long after the operation, found that (including a case recorded by himself) 19 cases had been published from which the result of the operation could be gauged; in only 2 of this number was there cure (10 per cent), in 6 there was improvement (32 per cent), and in 11 failure (58 per cent). Though in so great a proportion of the cases there is this return of spasm, it is sometimes less pronounced than before the stretching of the nerve; in other cases, however, it is quite as severe as ever, so that it will readily be understood that this method of treating facial spasm is not one which can be enthusiastically recommended. With proper technique, and good antiseptic precautions, there is no special difficulty or danger about the operation; and the prognosis as regards recovery from the resulting facial paralysis is good. It is well to supplement the operation by the continuous use of sedatives for a few months, in the hope that the combined effect may bring about what so rarely happens when either measure is adopted alone.

J. S. Risien Russell.

REFERENCES

1. Abadie et Dupuy-Dutemps. *Rev. neurol.*, Paris, 1906, xiv. 196.—2. Baum. *Berlin. klin. Wchnschr.*, 1878, xv. 595.—3. Bell. *The Nervous System of the Human Body*, London, 1830, 160.—4. Bernhardt. *Ztschr. f. klin. Med.*, Berl., 1881, iii. 96.—5. *Idem. Deutsche med. Wchnschr.*, Berl., 1882, viii. 121, 403.—6. *Idem. Centralbl. f. Nervenh.*, Leipz., 1882, v. 405.—7. *Idem. Arch. f. Psychiat.*, Berl., 1884, xv. 777.—8. Brissaud. *Journ. de méd. et chir. prat.*, Paris, 1894, lxv. 49.—9. Brissaud, Sicard, et Tanon. *Bull. Soc. méd. des hôp. de Paris*, 1906, 3^{e} s., xxiii. 831.—10. Debron. *Arch. gén. de méd.*, Paris, 1864, iii. 641.—11. Eulenburg. *Centralbl. f. Nervenh.*, Leipz., 1880, iii.—12. *Idem. Real-Encyclopädie der gesamten Heilkunde*, Wien, 1881, vi. 28.—13. Féré. *Twentieth Cent. Pract. Med.*, Lond., 1897, x. 690.—14. François. *Essai sur les convulsions idiopathiques de la face*, Bruxelles, 1843.—15. Gowers. *Diseases of the Nervous System*, Lond., 2nd ed., 1893, vol. ii. 248.—16. von Graefe. *Arch. f. Ophth.*, Berl., 1854, i. 440; 1858, iv. 184.—17. Graves. *Dublin Med. Journ.*, 1843, xxii. 394.—18. Gray. *Amer. Journ. Neurol. and Psychiat.*, N.Y., 1883, i. 515.—19. Hall. *On the Diseases and Derangements of the Nervous System*, London, 1841, 342.—20. Hallion. *Traité de méd.* (Charcot, Bouchard, et Brissaud), 2me édit., Paris, 1905, x. 269.—21. Harris. *Lancet*, Lond., 1909, i. 1310.—22. Hobson. *Brain*, Lond., 1882, iv. 531.—23. Hoffmann. *Arch. f. Psychiat.*, Berl., 1882, xii. 259 —24. Kaufmann. *Centralbl. f. Chir.*, Leipz., 1885, xii. 33.—25. Keen, W. W. *Trans. Amer. Surg. Assoc.*, Phila., 1886, iv. 275.—26. Lannois et Porot. *Lyon méd.*, 1904, cii. 233.—27. Macnamara. *Westminster Hosp. Rep.*, Lond., 1907, xv. 48.—28. Meige. *Tics*, Paris, 1905.—29. Meige and Feindel. *Tics and their*

Treatment, Transl., Lond., 1907, 143, 267.—30. Mills. *Journ. Nerv. and Ment. Dis.*, N.Y., 1897, xxiv. 702.—31. Oppolzer. *Allg. Wien. med. Ztg.*, 1861, vi. 73, 82.—32. Patrick. *Journ. Nerv. and Ment. Dis.*, N.Y., 1909, xxxvi. 1.—33. Putnam. *Arch. of Med.*, N.Y., 1881, v. 103.—34. Raymond. *Bull. méd.*, Paris, 1897, xi. 142.—35. Remak. *Berlin. klin. Wchnschr.*, 1864, i. 209, 221, 229.—36. Roberts. *Phila. Polyclin.*, 1894, iii. 411.—37. Romberg. *A Manual of Nervous Diseases of Man*, London, 1853, i. 293.—38. Rosenthal. *A Clinical Treatise on Dis. of Nerv. Syst.*, N.Y., 1879, 467.—39. Ross. *Diseases of the Nervous System*, 1883, i. 463.—40. Salzmann. *Centralbl. f. Nervenh.*, Leipz., 1888, xi. 547.—41. Sano. *Journ. de neurol.*, Paris, 1899, iv.—42. Schlösser. *Fortschr. der Med.*, Berl., 1904, xxii. 554.—43. Schüssler. *Berlin. klin. Wchnschr.*, 1879, xvi. 684.—44. Schott. *Deutsche med. Wchnschr.*, Leipz., 1890, xvi. 967.—45. Schultze. *Virchows Arch.*, Berl., 1875, lxv. 385.—46. Shaw. *Weekly Med. Rev.*, St. Louis, 1891, xxiii. 231.—47. Southam. *Lancet*, Lond., 1881, ii. 370.—48. Sturge and Godlee. *Lancet*, Lond., 1880, ii. 814.—49. *Idem. Med. Times and Gaz.*, Lond., 1880, ii. 631.—50. Zesas. *Wien. med. Wchnschr.*, 1885, xxxv. 852, 883.

J. S. R. R.

PARAMYOCLONUS MULTIPLEX

Synonyms.—*Myoclonus; Multiple myoclonus; Myoclonus epilepticus; Myokimie; Myospasia Simplex; Spinal epilepsy; Friedreich's disease; Convulsive tremor.*

By J. S. Risien Russell, M.D., F.R.C.P.

This affection is characterised by sudden shock-like clonic contractions, usually of corresponding muscles on the two sides of the body; the spasms may not only be symmetrical, but also isochronous.

History.—The affection was isolated from the chaos of motor neuroses by Friedreich in 1881, the condition is therefore sometimes designated by his name; but this observer's name is more familiarly associated with hereditary ataxia. As Friedreich's description was taken from one patient, it was quite impossible for him to draw a clinical picture applicable to all such cases, so that many indubitable instances of this condition have not conformed in all respects to the clinical picture presented by him. On the other hand, from 1883 to 1890, a number of cases were published as examples of this malady which cannot be accepted.

In 1891, however, Unverricht was able to give a much wider conception of the affection in his monograph entitled "Über Myoclonie," which includes a description based on five cases personally observed, and a critical review of previous publications on the subject.

In searching amongst earlier publications, it is impossible to avoid being impressed by the similarity between the condition described by Friedreich and one to which attention was first called by the late Sir Russell Reynolds, in which, in the intervals between the attacks, in a case of epilepsy, there occurred sudden shock-like contractions of various muscles. This resemblance is emphasised by the fact that myoclonus and epilepsy are now known to be sometimes associated in the same individual. An important point of difference is to be noted, however, as in contrast to what obtains in paramyoclonus, the muscular contractions were sometimes sufficiently severe to throw the patient to the ground.

Paramyoclonus has been described by some writers as "spinal epilepsy," a designation to be avoided, for this appellation has long been identified with the clonic spasms which occur in the parts below a destructive lesion of the spinal cord.

It is questionable whether the condition described by Hammond as "Convulsive tremor," and considered by himself and some other American observers as identical with Friedreich's paramyoclonus, has any real claim to be so regarded.

Causation.—Little definite is known of the etiology of the affection. Direct heredity has been traced only in rare instances; and although indirect heredity in the shape of a neuropathic taint may be determined, this is by no means invariable. In not a few there has been a history of alcoholism in one of the parents.

That the affection may be congenital in the sense that the tissues become susceptible to it during uterine life, although the disease does not become manifest for a certain interval after birth, is suggested by cases such as those recorded by Unverricht, in which five children of the same family became affected between the ages of six and thirteen years. Whether such cases prove a congenital taint or not, they prove that the affection may run in a family. The possibility of simulation is made improbable by the fact that in one child, which was brought up away from the family, the disease appeared, nevertheless, at the time of life when it had broken out in the other members. Moreover, the association with epilepsy in Unverricht's case lends support to the view that the condition was genuine.

The tendency for the affection to manifest itself where there is a degenerative condition of the nervous system, whether congenital or acquired, is shewn by its frequent association with epilepsy—a disease in which certain tissues are prone to degeneration, or are actually undergoing this change.

That shock, mental or physical, may set up this spasmodic disorder, as in Friedreich's patient, there can be no question; but it is equally certain that no such exciting cause may be needed to evoke it; many cases have been recorded in which this cause was absent.

Again, the myoclonic spasm may manifest itself in the weakness of overwork or acute illness, as in several instances after severe and repeated attacks of malaria. It has been looked on, by Lemoine and Lemaire, as related to neurasthenia.

Pathology.—No changes have been found in the neuro-muscular system, except hypertrophy of the muscles in one case, and nothing certain is known of the pathology of the affection. Some authors, notably Möbius, Strümpell, Hirt, and Pitres, attribute myoclonus to hysteria;

others only admit that hysteria may present itself under the guise of myoclonus. Bötticher, who recognises two principal types of the affection, attributes the paramyoclonus of Friedreich to hysteria, while regarding the myoclonus of Unverricht as identical with Huntington's chorea (p. 549). That hysteria should manifest itself in a manner more or less resembling the clinical picture of a myoclonus is scarcely surprising, when we remember how closely hysteria may simulate the manifestations of other affections of the nervous system; but the following considerations, among others, indicate that all cases of paramyoclonus cannot be hysterical. Perhaps nothing is more opposed to the hysterical hypothesis than the association of myoclonus with epilepsy, unless we are to admit that this variety is distinct from that to which the term paramyoclonus is strictly applicable. Again, in the present malady there is little evidence of the concomitance of any of the usual stigmata, one or more of which we expect to find in a subject of hysteria. Moreover, were the condition hysterical, it would be not a little strange that men should be more commonly affected with myoclonus than women.

Another conjecture is that myoclonus belongs to the group of chronic choreas, but that there are important differences between the two conditions is evident from the remarks on their differential diagnosis (*vide* p. 656).

A third view finds favour with Lugaro and others. According to them the muscular spasms in myoclonus are the "exteriorisations," so to speak, of a general myoclonic state of the neurons of the central nervous system; a state that may find expression in the fibrillar trembling of neurasthenics, fibrillar chorea, or paramyoclonus of Friedreich, when the neurons of the anterior horns of the spinal cord are the seat of the morbid change; in electric chorea and common tic, when the motor neurons of the second order are involved; and "maladie des tics," when the psychomotor neurons are implicated.

And lastly must be mentioned Wagner's suggestion that we should perhaps look to the thyroid for an explanation of the condition, as the muscular spasms of animals after extirpation of the thyroid resemble those of paramyoclonus; and, indeed, that in a case of his such spasms were cured by thyreoiodin. The same observer and Jauragg have observed similar contractions in animals after removal of the parathyroids.

There is an equal want of unanimity of opinion as to the part of the neuro-muscular system that is at fault. Of the different views advanced, that of Popow, which supposes the defect to be in the muscles themselves, has little to recommend it from the point of view of the arguments he adduces in support of his hypothesis; nevertheless, the majority of observers are now agreed that the clinical manifestations of the malady depend on some disturbance of function in the lower motor neurons in the muscles.

Friedreich's view, subsequently supported by Unverricht, that the seat of disturbance lies in the anterior-horn cells of the spinal cord, has many adherents, including Löwenfeld, Marie, Ventick, and others; and one of the strongest arguments in its support is the symmetrical distribution, and in some cases the isochronicity, of the myoclonic spasms. Turtschaninow has succeeded in evoking clonic muscular contractions experimentally in dogs, the spasms resembling the myoclonic contractions observed in Unverricht's cases; and he found that the contractions continued even after ablation of the cerebrum, section of the cerebral peduncles, or transverse section of the spinal cord high up.

Most of those authors who have opposed the supposition that the spinal cord is the seat of the disturbance, have advanced, as an alternative hypothesis, that derangement of the motor neurons of the cerebral cortex is concerned in the spasms; among them may be mentioned Rubino, Seppilli, Minkowski, Grawitz, and Raymond. It has been urged that the frequent association of epilepsy with myoclonus is a strong argument in favour of the cerebral origin, as the former disease is essentially an affection of the cortex cerebri. The fact that the myoclonic spasms are increased before and after the epileptic attacks has been regarded as additional evidence pointing to the probable dependence of the two conditions on some defect in the same part of the central nervous system. The clonic character of the spasms has been regarded as another point in favour of the cortical basis of the affection; and it has been stated further by some writers, such as Raymond, that, besides the influence of moral causes in evoking or exaggerating the spasms, the intellectual faculties of the subjects of myoclonus are rarely quite normal. The chief points urged by Unverricht and others against the attribution to the cerebral cortex are that the spasms are symmetrical and often isochronous, that they involve single muscles or parts of muscles, and that even when groups of muscles are simultaneously involved they are not such as act together in synergic combination; whereas in all spasms known to be of cerebral origin the muscles involved are those which are brought into action synergically, and which result in co-ordinated movements like those of volition; so that flexion of the elbow, for instance, may be a consequence of the contraction of the group of muscles concerned in bringing about this movement, but that isolated contraction of a muscle, as for instance of the supinator longus, is never met with. It has further been pointed out that the most delicate electrical stimulus applied to the cerebral cortex can only evoke contractions of a group of muscles concerned in some particular movement, and never of an isolated muscle.

The last suggestion that we have to consider presupposes that epileptic attacks may form part of the clinical picture of paramyoclonus, and it is accordingly believed that the motor neurons both of the brain and spinal cord may be affected; and that the neuroclonic state existing in the neurons of the anterior horns of the spinal cord or their homologues in the medulla oblongata and pons, may give rise to myoclonic spasms; whereas an altered state in the neurons of the cerebral cortex gives rise to epileptic attacks.

Symptoms.—According to Friedreich's original description, the following are the characteristic manifestations; namely, clonic spasms in a number of symmetrical muscles of the upper and lower limbs, interfering in no way with motor power and co-ordination of movement, ceasing during sleep and during voluntary movements. The locomotor effects of the spasms is either nil or but very slight, and the muscles of the face and trunk escape. The nutrition of the affected muscles is unaffected, and their mechanical and electrical irritability is unaltered. In his case there was no affection of sensation, the superficial reflexes were increased, and there was also marked increase of the knee-jerks.

The case which enabled Friedreich to give this description of the affection was the result of a severe shock, and the spasms, after lasting several years, ceased for a time, but returned, and, according to Schultze, never ceased again until the patient died. Excellent as is his description, and applicable as it is to some cases of the affection, it requires some modification when we attempt to regard it as a type of the class of cases that must be included in the same category as that of Friedreich, for they differ from it in several important particulars. In the first place, the intensity of the spasms differs in different cases, so that in some a definite though slight locomotor effect is evident, whilst in others, and more usually, this is entirely absent. The extent of the invasion of different parts of the body also varies; and both the face and trunk muscles, which escaped in Friedreich's case, may be involved. Moreover, voluntary movement has by no means invariably an inhibiting effect on the spasms; on the contrary, in some cases such movements evoke the spasms, or intensify them. Then again the knee-jerks, which in Friedreich's patient were markedly increased and are often in excess, may in many cases be normal or even diminished.

These preliminary remarks will clear our view of the class of cases to be included in this affection, whether we hold to the name originally given to it by Friedreich, or whether we follow Unverricht, whose appellation, though shorter, is less comprehensive. With the broader conception of the affection indicated above, we are now in a position to consider the various symptoms of the malady more in detail.

The spasmodic movements have been described as lightning-like in rapidity, but although they resemble the contraction which is obtained from a muscle by a single faradic excitation, they are commonly quite deliberate, though rapid, and by no means "lightning-like." Usually the sudden contraction in one muscle is followed by an equally sudden contraction in another; in other instances the spasm is repeated in the same muscle several times in succession. There is no rhythm in the succession of the shocks; they may occur singly or in batches separated by quiet intervals; as many as forty or fifty contractions may occur in the course of a minute.

The spasms may seize muscles singly, parts of muscles, or whole groups of them; but the great characteristic of the distribution of the spasm is that it attacks muscles which are not supplied exclusively by a single nerve, or which cannot possibly be thrown singly into contraction by volition; even when a group of muscles is involved simultaneously in spasm the combination is without any co-ordinated function, and is such as no effort of the will can reproduce.

Bastianelli considers that there is a special group of cases of myoclonus characterised by fibrillar contractions of the muscles, in which those of the face may participate. He states that in these cases the electrical excitability of the muscles is sometimes increased; and there is always an unduly persistent contraction after the excitation has been discontinued. Paraesthesias and paralgesias form part of the clinical picture of this group, to which Giovanni Biancone has given the name "Myokimie."

Another characteristic feature of the distribution of the spasms is that they may be strictly symmetrical on the two sides of the body. Symmetry is not invariably observed, however, for, in exceptional cases, one side of the body has been affected alone, as in a case described by Chauffard. Not only may the muscular spasms be even strictly symmetrical, but they may occur simultaneously in the same muscles on the two sides. The latter, however, is by no means the rule; for instead of being isochronous, the spasm on one side may be followed by contraction of the same muscle on the opposite side after an interval which indeed is usually of very short duration.

Certain muscles are much more commonly concerned than others, although in any given case nearly the whole of the voluntary muscles of the body may be affected, with the exception of the ocular muscles, which are always spared. The muscles of the limbs are affected more frequently than those of the trunk, and the latter more commonly than those of the face; and, as we have already seen, the muscles both of the trunk and face escaped in Friedreich's case, those of the limbs being affected alone. All the muscles of the limbs are not equally prone to be affected; those of the proximal segments are usually involved, whilst those of the distal segments either escape or are much less affected, and the most peripheral parts remain quite free from spasm. The pectorals and deltoids are especially liable to be affected, as are the biceps, triceps, and supinator longus; in the legs the quadriceps extensor group, and the adductors, and, in the trunk, the abdominal recti, obliques, and the erector spinae. But even the diaphragm and cremasters may share in the spasms.

The extent to which any locomotor effect is produced by the spasms varies. As in Friedreich's case, the absence of any locomotor effect of the muscular contractions is still regarded as one of the characteristic features of the affection. Indeed the spasms are not usually observed until the patient's clothes are removed, so as to reveal the muscular contractions. Occasionally the contraction of the muscle is sufficiently powerful to produce a perceptible though only slight movement of the limb.

Attitude appears to have a distinct influence on the spasms; for, as a rule, they are less pronounced when the patient is standing than when sitting, and when sitting than when lying recumbent; a state of things no doubt closely associated with the next problem, namely, that of the influence of voluntary movements on the spasms. This we may consider from two standpoints—the influence of the former in inhibiting, accentuating, or evoking the latter; and the degree in which the latter interfere with the former.

It will be remembered that one of the characteristic features in Friedreich's first case was that voluntary movements inhibited the myoclonic spasms, so that all such spasms ceased during the performance of a voluntary act. This has been noted in other recorded cases of myoclonus; but it is as common to meet with cases in which, instead of diminishing the spasm, voluntary movements actually augment it, or even evoke the involuntary spasms in parts quiescent up to the moment of the volition. Similarly, any form of mental excitement or physical fatigue will usually augment the spasms. In some cases all spasms are in abeyance so long as the patient remains quiescent; whilst in others the spasms go on quite independently of repose, and may indeed, as we have already seen, be more marked in the recumbent than in any other posture. In nearly all cases the spasms cease entirely during sleep; but in one, recorded by Schütte, although the more pronounced shocks ceased, fibrillar contractions persisted.

Voluntary movements are not generally interfered with by the myoclonic spasms, even when the former evoke or intensify the latter. There is no loss of motor power, nor is there any affection of co-ordination of movement, other than interference with a voluntary act by the unexpected intrusion of some unbidden muscular contraction.

The muscles do not atrophy, and their mechanical excitability is said to be normal; but nevertheless percussion of a muscle, or irritation of the skin, may suffice to evoke or increase the myoclonic contractions. Exposure to cold may have a like effect, as may excitement.

The electrical excitability of the affected muscles is unaltered, but the application of the current usually intensifies the spasm; in some instances an undue persistence of the contraction of the muscle after shutting off the current has been noted.

Myoclonus and Epilepsy.—This association was present in Unverricht's cases; and has been comparatively frequent. Two instances of the kind were shewn at a meeting of the Neurological Society of London by Dr. Ferrier in 1897; the patients were brothers, and the proposal was made to designate such cases "myoclonus epilepticus,"—to distinguish them from cases of myoclonus unassociated with epilepsy. This association was met with by Dr. Mott in four members of a family, in which all the others were epileptic, and an uncle was said to have suffered from epilepsy. The epileptic attacks commonly occur at night, and it is not unusual to be able to foretell a fit, as the myoclonic contractions become much worse during the period immediately preceding it. The spasms may be more intense for a time after an epileptic fit: in other instances, however, they are considerably less marked after the epileptic attack.

There are, however, those who regard the cases in which there is this association as quite distinct from paramyoclonus, and as due to functional disturbance of the cerebral cortex.

Sensory Phenomena.—If we except the class of cases which Biancone proposes to call "Myokimie," in which subjective sensory disturbances constitute an important part of the morbid group, there is a total absence of sensory defect in myoclonus.

Reflexes.—The superficial reflexes are commonly increased, as in Friedreich's case, though not necessarily so; the tendon-jerks may be either unaltered, increased, or diminished. The sphincters are unaffected.

Psychical Disturbances.—Mental disturbances are exceptional, and according to some observers entirely absent, although Raymond regards the intellectual faculties of the subjects of this disease as rarely quite normal. In 2 cases observed by Bresler there was progressive enfeeblement of intelligence, and a fatal issue; but in cases of the kind it is difficult to avoid the suspicion that hereditary or some allied form of degenerative chorea has been mistaken for paramyoclonus.

Diagnosis.—The affections with which paramyoclonus is likely to be confused are chorea, hereditary chorea, electric chorea, tic, and hysteria.

Chorea.—The spasmodic movements in chorea resemble voluntary movements, and are the result of contractions of groups of muscles in synergic combination, which occasion marked displacements of the limbs and other parts. The movements are commonly unilateral; an extremely rare event in myoclonus, in which affection the face is by no means always affected, whereas facial grimaces are among the most common manifestations of chorea.

Hereditary Chorea.—Much of what has been said of ordinary chorea is applicable to this and allied forms of degenerative chorea in which progressive mental deterioration supplies a further point of difference in the majority of cases; but it is noteworthy that in this variety of chorea the muscular contractions may be so slight in the earlier stages that there may be little or no resulting movement of the limbs.

Electric Chorea.—If by electric chorea we mean the affection described by Dubini (*vide* p. 554), there should be little difficulty in diagnosis, in spite of the occurrence of epileptic convulsions; Dubini's disease is attended with high temperature, progressive paralysis with muscular atrophy, and loss of faradic excitability of the muscles; it runs an unfavourable course and terminates in death. In the hysterical affection which has been called electric chorea the usual stigmata of hysteria are present, as a rule, and serve to distinguish it; moreover, as in chorea, the contractions are of synergic groups of muscles, such as can be brought into action by an effort of the will, not of isolated muscles or of parts of these. The condition described by Henoch as electric chorea appears to be none other than a form of tic or paramyoclonus; and in one of his cases the condition was associated with epilepsy.

Tic.—The muscular contractions are not of the spinal type seen in myoclonus, but are clearly of cortical origin, and result in some psychomotor act such as can only be accomplished by the synergic action of a group of muscles. Moreover, the movements can be inhibited for a variable period by an effort of the will, although ultimately a movement that is gross and purposive will result which is altogether unlike anything seen in paramyoclonus.

In the variety known as Gilles de la Tourette's disease ("maladie des tics convulsifs," p. 621), in addition to the more usual psychomotor acts and gestures encountered in the ordinary varieties of tic, there are added other impulsive tendencies which the patient cannot resist, and which may be exhibited by the imitation of some gesture (echokinesis), the repetition of words (echolalia), or a tendency to blurt out obscene words (coprolalia). From this it will be obvious that there is little real similarity between the two affections; and yet there can be little doubt that cases have been recorded as paramyoclonus that have really been examples of tic.

Hysteria.—The diagnosis of paramyoclonus from hysteria would not be easy if we were obliged to admit that cases like that recorded by Hoffmann are in reality cases of hysteria in the guise of paramyoclonus. If, however, we attribute to hysteria only such spasmodic movements as can be reproduced by an effort of the will (the proper position to take in the matter, it seems to me), then the distinction between the spasms of hysteria and those of paramyoclonus at once becomes evident. Moreover, in hysteria the ambulatory effect of the spasms is much greater, the spasmodic movements are much more influenced by observation and excitement, and it would be unusual not to find some of the stigmata of this affection in addition to the spasmodic disorder.

Prognosis.—The outlook is unfavourable in genuine cases of the affection. Except that its course is liable to be interrupted by pulmonary tuberculosis or some other intercurrent fatal complication, the subjects of paramyoclonus may live a long time. They usually die without enjoying any period of remission of the spasms, and relapse has been the rule when a cure seemed secured. It is probable that hysterical simulation of myoclonus claims most of the cases in which cures are supposed to have been effected in what appears to be an incurable malady.

Treatment.—It is naturally of primary importance that measures should be adopted to improve the general condition, and to bring about a more stable condition of the motor neurons of the central nervous system by improving their nutrition. Although the affection manifests itself so often without any evidence of an exciting cause, such should always be sought for, and if still found in action should be promptly dealt with; for it must be remembered that the longer some perverted action of the motor neurons continues the more difficult it becomes to break them of a habit established by frequent repetition.

Cures have naturally been credited to numerous remedies, including various forms of electricity, owing to favourable results that have been obtained in cases that have really been examples of hysteria simulating myoclonus. Thus, the constant current to the spine, static and high-frequency currents, and faradic electricity to the muscles, have each had their advocates.

Hydropathic methods have also found favour in the treatment of myoclonus, either alone or variously combined with electrical treatment.

Of drugs arsenic has a good reputation. Chloral controls the spasms temporarily, but when its use has to be long continued it loses its effect. The bromides have much less influence over this condition than over epilepsy. Hyoscine has been tried without notable effect, and cocaine has been given subcutaneously and as a subarachnoid injection, but is not to be recommended. Thyroid gland substance in different forms has been equally unfruitful of good results in this affection. Psycho-therapy can only be expected to do good where hysteria or tic has been mistaken for myoclonus.

J. S. Risien Russell.

REFERENCES

1. Bechterew. *Arch. f. Psychiat.*, Berl., 1888, xix. 88.—2. Biancone. *Riv. sper. di fren.*, Reggio-Emilia, 1898, xxiv. 313.—3. Binder. *Über Myoclonie*, Berl., 1894.—4. Boettiger. *Berl. klin. Wchnschr.*, 1896, xxxiii. 142.—5. Bregmann. *Arch. f. Psychiat.*, Berl., 1895, xxvii. 588.—6. Burzio. *Ann. di freniat.*, Torino, 1898, viii. 165.—7. Céry et Jaudon. *Union méd. du nord-est*, Reims, 1896, xx. 133.—8. Colleville. *Gaz. hebdom.*, Paris, 1893, xxx. 548.—9. Curschmann. *Lehrbuch der Nervenkrankheiten*, Berl., 1909, S. 739.—10. D'Allocco. *Riforma med.*, Napoli, 1897, xiii. 223, 237, 247.—11. Desterac. *Arch. d'électric. méd.*, Bordeaux, 1898, vi. 281.—12. *Idem. Ann. d'électrobiol.*, Paris, 1898, i. 605.—13. Feinberg. *Ztschr. f. klin. Med.*, Berl., 1893, xxiii. 431.—14. Ferrannini. *Riforma med.*, Napoli, 1897, xiii. 771.—15. Faldella. *Riv. speriment. di freniat.*, Reggio-Emilia, 1888, xiv. 193.—16. *Idem. Neurol. Centralbl.*, Leipz., 1889, viii. 238.—17. Friedreich. *Virchows Arch.*, Berl., 1881, lxxxvi. 421.—18. Fry. *Journ. Nerv. and Ment. Dis.*, N.Y., 1888, xv. 397.—19. Garnier et Santenoise. *Ann. méd.-psychol.*, Paris, 1898, vii. 393.—20. Goldflam. *Neurol. Centralbl.*, Leipz., 1892, xi. 97.—21. Gucci. *Riv. speriment. di freniat.*, Reggio-Emilia, 1892, xviii. 515.—22. von Hoesslin. *München. med. Wchnschr.*, 1896, xliii. 271.—23. Hoffmann. *Deutsche med. Wchnschr.*, Berl., 1896, xxii. 36.—24. Homén. *Arch. de neurol.*, Paris, 1887, xiii. 200.—25. *Idem. Neurol. Centralbl.*, Leipz., 1891, x. 385.—26. Huchard et Fiessinger. *Rev. de méd.*, Paris, 1905, xxv. 741.—27. Kny. *Arch. f. Psychiat.*, Berl., 1888, xix. 577.—28. Köppen. *Ibid.*, 1888, xix. 707.—29. Kovalevsky. *Arch. ital. per le mal. nerv.*, Milano, 1887, xxiv. 288.—30. Krewer. *Deutsche Ztschr. f. Nervenh.*, Leipz., 1896, ix. 62.—31. Lemoine. *Rev. de méd.*, Paris, 1892, xii. 882.—32. Lemoine et Lemaire. *Ibid.*, 1889, ix. 1018.—33. Lenoble et Aubineau. *Ibid.*, 1906, xxvi. 471.—34. Levi. *Bull. Soc. anat. de Par.*, 1895, lxx. 331.—35. Löwenfeld. *Ärztlicher Intelligenzbl.*, München, 1883, xxx. 151, 164.—36. Lugaro. *Riv. di patol. nerv. e ment.*, Firenze, 1896, i. 389.—37. Lundborg. *Neurol. Centralbl.*, Leipz., 1904, xxiii. 162.—38. Marie. *Progr. méd.*, Paris, 1886, iii. 152, 241.—39. Marina. *Arch. f. Psychiat.*, Berl., 1888, xix. 684.—40. Minkowski. *Mittheil. aus der medic. Klinik zu Königsberg*, 1888, 59.—41. Mirallié. *Gaz. méd. de Nantes*, 1897-98, xvi. 346.—42. Moser. *Med. Rec.*, *N.Y.*, 1898, liv. 518.—43. Mott. *Arch. of Neurol.*, London, 1907, iii. 320.—44. Papillon. *Rev. mens. des mal. de l'enf.*, Paris, 1907, xxv. 126.—45. Peiper. *Deutsche med. Wchnschr.*, Berl., 1890, xvi. 399.—46. Raymond. *Clinique des maladies du système nerveux*, Paris, 1896, 589.—47. Remak. *Arch. f. Psychiat.*, Berl., 1884, xv. 853.—48. Schetalow. *München. med. Wchnschr.*, 1895, xlii. 696.—49. Schultze. *Neurol. Centralbl.*, Leipz., 1886, v. 363.—50. Schütte. *Ibid.*, 1897, xvi. 11.—51. Seeligmüller. *Deutsche med. Wchnschr.*, Berl., 1886, xii. 405 : 1887, xiii. 1117.—52. Seppilli. *Riv. sper. di freniat.*, Reggio-Emilia, 1895, xxi. 326.—53. Silvestrini. *Med. contemp.*, Napoli, 1884, i. 83.—54. von Söldner. *Neurol. Centralbl.*, Leipz., 1896, xv. 612.—55. Soury. *Ann. méd.-psychol.*, Paris, 1897, v. 399.—56. Spitzka. *Journ. Nerv. and Ment. Dis.*, N.Y., 1887, xiv. 626.—57. Starr. *Ibid.*, 416.—58. Stembo. *Berlin. klin Wchnschr.*, 1896, xxxiii. 983.—59. Taylor, F. *Med.-Chir. Trans.*, London, 1906, lxxxix. 419.—60. Unverricht. *Deutsches Arch. f. klin. Med.*,

Leipz., 1890, xlvi. 413.—61. *Idem. Die Myoclonie*, Leipz. u. Wien, 1891.—62. *Idem. Deutsche Ztschr. f. Nervenh.*, Leipz., 1895, vii. 32.—63. Variot et Papillon. *Arch. de physiol.*, Paris, 1891, 5. s., iii. 218.—64. Ziehen. *Arch. f. Psychiat.*, Berl., 1888, xix. 465.

J. S. R. R.

ECLAMPSIA NUTANS

Synonyms.—*Epilepsia nutans; Salaam convulsions; Spasmus nutans; Tic salaam.*

By J. S. Risien Russell, M.D., F.R.C.P.

Newnham furnished the first satisfactorily recorded account of cases of this kind in 1849; a year later Wiltshire published a case together with an account of the salient features of the affection. In this same year two cases were recorded in Germany by Faber, and, according to him, Romberg would seem to have been the only German observer who had previously noticed it.

The malady is one of childhood, nearly all cases occurring between the period of first dentition and puberty: it is characterised by peculiar bowing movements of the body, the whole trunk moving forward and downward, so that sometimes the head nearly touches the knee; in other cases, however, the body and head are only bent slightly forward. This movement occurs in paroxysms which may be repeated as often as 50 to 140 times a day; and each attack may be made up of 20 to 100 bowings. In other instances the attack has occurred once a day only, and that at some stated hour, as for instance, in one of Faber's cases, at 11 a.m.; a second attack sometimes occurred in the evening. Sometimes the patients are free from attacks for intervals of a few days. Nodding movements of the head accompany these "Salaam convulsions," as they have been called; moreover, nodding movements of the head may be the first abnormal phenomenon, the bowing forward of the trunk being added subsequently, as in the case of his own child recorded by C. West in 1841.

The severer bowing attacks have been observed to follow sleep; and it is said that their severity appears to be somewhat proportional to the depth of the preceding sleep. During sleep, however, all such movements are in complete abeyance. When in the attack the child often seems bewildered, but consciousness is not completely lost; and, though after a paroxysm the patients may be drowsy and exhausted, in other cases they are quite bright and happy as soon as the bowings have ceased.

As the case progresses, more general automatic movements are observed to occur, and these may take the form of epileptiform or tetanoid attacks; and subsequently paralysis, of paraplegic or hemiplegic type, has been met with in some instances; though this does not appear to be an essential part of the malady. In regard to the epileptiform attacks, momentary loss of consciousness has been observed in some instances, immediately preceding the convulsions in the limbs; in other cases of eclampsia nutans no affection of consciousness has been noted. The epileptiform attacks may increase in frequency, and indeed the child may ultimately become quite definitely epileptic.

There is a pronounced alteration of the mental faculties in these patients, so that, although apparently perfectly normal in intelligence before the onset of the malady, they subsequently have an imbecile appearance, and their mental faculties do not give evidence of having reached a due stage of development. Finally, complete imbecility or idiocy may result, from which the patient may, however, recover, though slowly and with difficulty.

This affection does not appear to have attracted much individual attention during recent years, no doubt because it is now generally recognised as a manifestation of epilepsy, but many of those who have furnished us with accounts of the condition already described as "head-nodding" have insisted that that affection is to be distinguished from eclampsia nutans, which latter condition they assert to be an independent malady, and, moreover, an epileptic manifestation.

Faber was of opinion that the malady could not be regarded as epilepsy, seeing that loss of consciousness, one of the principal symptoms of an epileptic attack, is sometimes absent, as in one of his own cases. That true epileptic attacks may occur in association with the other manifestations of the affection has long been recognised, and the general belief now is that the condition is epileptic.

In considering wherein the malady differs from that which will be described directly under the title of head-nodding (p. 661) there are several points deserving of attention. Although cases of eclampsia nutans have been met with in families free from any hereditary taint of neurosis or mental affection, in other instances the converse has been notable. Then, again, the condition manifests itself later in childhood than head-nodding, so that, although it may appear at six months, it may not do so until the child is as many years of age. So, too, the more general automatic movements prone to occur with "salaam convulsions" are foreign to the ordinary cases of head-nodding. Again, although some mental defect has been observed in certain cases of head-nodding, it has never attained the degree met with in eclampsia nutans; furthermore, the mental defects have been transitory.

J. S. Risien Russell.

REFERENCES

1. Barnes. *Liverpool and Manchester Med. and Surg. Rep.*, 1873, i. 54.—2. Barton. *Amer. Journ. Med. Sc.*, Phila., 1843, xxxi. 243.—3. Bennett. *Ibid.*, 364.—4. Bidwell. *New York Journ. of Med.*, 1851, vii. 342.—5. Cox. *Dublin Quart. Journ. of Med.*, 1863, xxxv. 344.—6. Davidson. *Liverpool Med. and Surg. Rep.*, 1869, iii. 39.—7. Ebert. *Ann. d. Charité-Krankenh. zu Berlin*, 1850, i. 762.—8. Faber. *Journ. f. Kinderk.*, 1850, xiv. 260.—9. *Idem.* *Schmidts Jahrb.*, 1850,

lxvii. 213.—10. *Idem.* *London Journ. of Med.* 1850, ii. 569, 590.—11. Finlayson. *Glasg. Med. Journ.*, 1877, ix. 352.—12. Newnham. Clay's *Brit. Rec. of Obst. Med. and Surg.*, Manchester, 1849, ii.—13. Niemeyer. *Textbook of Pract. of Med.*, New York, ii. 318.—14. Steiner. *Compendium of Children's Diseases* (transl. by Lawson Tait), London, 1874, 82.—15. Tanner's *Prac. of Med.*, London, 6th ed. 1869, i. 463.—16. *Idem.* *On the Diseases of Infancy and Childhood*, by Meadows, London, 2nd ed., 191.—17. Thomson, J. *Festschrift in Honor of Abraham Jacobi*, New York, 1900, 65.—18. Watson. *Brit. Med. Journ.*, 1860, ii. 757.—19. West, C. *Lancet*, London, 1841, i. 724.—20. *Idem.* *Amer. Journ. Med. Sc.*, Phila., 1841, xxviii. 187.—21. *Idem.* *On the Diseases of Infancy and Childhood*, London, 5th ed. 201.—22. Wiltshire. *Lancet*, London, 1850, i. 485.—23. *Idem.* *London Journ. of Med.*, 1850, ii. 590.

J. S. R. R.

HEAD-NODDING

Synonyms.—*Head-jerking; Nodding spasm; Spasmus nutans; Salaam spasm; Gyrospasm of the head; Eclampsia rotans.*

By J. S. Risien Russell, M.D., F.R.C.P.

Introduction.—This is an affection of early infancy in which nodding, lateral, or rotatory movements of the head occur from time to time, with or without nystagmus.

Of the names that have been proposed for the condition, "Head-nodding," imperfect as it is, is on the whole the most satisfactory; some of the others suggest a spasmodic character, whereas the movements are peculiarly free from spasm; and others again are apt to lead to confusion with a somewhat different affection—known variously as "epilepsia nutans," "eclampsia nutans," and "tic salaam,"—which is a form of epilepsy (*vide* p. 659).

The earliest records of head-nodding are by Ebert in 1850, and by Romberg and Henoch in 1851. In 1890 W. B. Hadden published a paper on the subject in the *Lancet*, and subsequently another account, based on a larger number of observations, in the St. Thomas's Hospital Reports for the same year. Two years later Peterson of New York recorded his experience of 5 cases of the affection in a paper entitled "Gyrospasm of the Head in Infants." Among those who early called attention to the condition in this country were Sir Stephen Mackenzie and Dr. Gee.

Causation.—No special hereditary influences appear to have been traced in the large majority of these cases; but in exceptional instances more than one member of the same family has been affected. The condition is essentially one of early infancy, most cases occurring under one year of age, the majority between six and twelve months; though Henoch observed it in a child aged three years, Prof. Still in one of four, and Mouat in one of five. The age position in a family seems to make no difference, and, according to Hadden's figures, males and females appear equally liable, though Aldrich alleges a preponderance of females, a state of things which obtained in Raudnitz's series of cases.

Henoch attaches especial importance to dentition as an immediate cause, although other forms of reflex irritation may be similarly operative, a view that is supported by Soltmann and Prof. Still. Hadden, however, met with cases in which dentition neither caused nor aggravated the movements; and in most of his cases the usual local and general signs of disturbed dentition were absent. Moreover, head-nodding may make its appearance at a time when teething is not active; for instance at the age of two months, and even younger. Peterson's observations also fail to support Henoch's view. It is, however, worthy of note that relapses have occurred in cutting of a tooth, and that in cases of head-nodding teething is occasionally late and slow, even in the absence of rickets.

The children are usually in excellent health, and there appears to be no reason to regard artificial feeding as a factor in causation. Although only 2 cases in Hadden's first series presented signs of rickets, this condition was evident in 9 out of the 21 cases of his second series, and is present in the majority of cases, so that Kassowitz and Stamm both blame this affection. Hochsinger believes that tetany is an etiological factor because it and head-nodding occur in the same children and so constantly occur in the winter. Congenital syphilis appears to play no part in the etiology. Any debilitating disease may determine or aggravate the movements, so that diarrhoea, diphtheria, whooping-cough, measles, aphthous stomatitis, and scurvy have played this part.

The patients have, as a rule, been born in natural labour; but after birth a history of falls or blows on the head has been obtained often enough to justify the belief that such traumatic influences may play a part in the causation; indeed, Peterson, Hirsch, and others regard trauma as the most potent of the proximate causes; and Lange believes that a central injury is the cause in all cases. This view, however, does not receive any support from Rietschel's observations.

There does not appear to be any close relation between this affection and the convulsive state in general. In only three of Hadden's second series of cases was there a personal history of convulsions, although in six instances other children in the family had had convulsions.

The children have a peculiar way of looking at objects; but most careful testing of vision has failed to discover any defect, and the ophthalmoscopic appearances have been normal. Defective light has been regarded as a cause by Raudnitz and others, but there is little justification for this view, although Dr. J. Thomson and Prof. Still find that the affection usually begins during the dark months of the year.

Pathology.—No anatomical lesion has been demonstrated to account for the clinical manifestations which are in accord with those of a functional and, it may be, reflex neurosis as opposed to a disease with an organic cause.

Hadden regarded the symptoms as due to instability of the motor centres of the cerebral cortex with consequent disarrangement of movements during their evolution in the infant. He points with much reason to the fact that the first acquired movements of the young child are those of the head and eyeballs, which do not become harmoniously associated until the fourth month; indeed, for a considerable time after this date the voluntary movements of the head are ataxic. If, moreover, evolution in some of these children is unusually rapid, as seems to be the case, there is still greater reason why the movements of the head and eyes, being precocious, should be specially liable to disarrangement.

In short, the voluntary or purposive movements of the head and eyes are not at that age sufficiently registered in the psychomotor centres of the cortex, and dissolution results from the inability of the cortical centres to cope with work which has been too early expected of them.

Others have regarded the condition as a functional disorder, the result of congenital or acquired nervous instability, in which some peripheral irritation acts as the exciting cause; and some regard it as more of the nature of a habit or trick, and thus allied to the tics of later childhood.

Symptoms.—Two symptoms are pre-eminent in the clinical picture of this affection—spontaneous movements of the head and nystagmus. This statement is in accord with my own experience, and is further supported by most of the published records of such cases; though at variance with the experience of Peterson, who concluded that nystagmus is not usually associated with the head movements, but is only an occasional concomitant.

Although the affection is spoken of as "head-nodding," as a matter of fact a pure nodding movement, such as is made in giving assent, is rare. When it occurs it exactly resembles the movements of the head of a mandarin doll. In one of Henoch's cases the whole of the body was bent forward as well as the head, so that the movement more closely resembled a salaam; but it seems possible that this was a case of eclampsia nutans rather than one of the class we are now considering.

A lateral movement of the head, such as that which signifies negation, is the most common. Hadden evidently distinguished simple lateral movements of the head from rotary movements; but this distinction is not obvious in the writings of others, and in the cases which I have myself seen the lateral movement was one of rotation. In all of the five cases observed by Peterson the movement was simply rotatory, even without any nodding.

The nodding or lateral movements may be present alone; but more commonly they are variously combined in the same case; thus the one movement may be observed at one time, whilst the other is seen at another; they may alternate during the same period of the affection, at one moment the one, at the next the other manifesting itself; or the one movement may run so rapidly into the other as to make it difficult to say which is the dominant factor in the combined movement which results. Henoch found that in many cases the rotation movements were most marked and the nodding only slight; though he met with nodding more frequently than did Hadden, who observed pure nodding in a single instance only, and in this case the child's mother said that lateral movements were present sometimes.

The movements of the head are rhythmical, but vary in rate in different cases; they are usually slow, though in some instances they are much more rapid; they may be almost constant, but more usually occur at varying intervals. Nearly all observers are agreed that the movements are especially prone to occur during efforts at fixation and during excitement; Peterson, on the contrary, stated that they became arrested when the child's attention was called to an object. The movements only occur when the head is unsupported, so that they always cease when the child lies down. They cease when it is asleep, and, according to Mills, when its eyes are covered.

Zappert, Ungar, Weyl, and Oppenheim have all observed cases in which rhythmical movements of the head occurred only during sleep, lasting with short intermissions throughout the night, and have sometimes been noticed when the child has slept during the day. Opinions as to the nature of the condition have wavered between tic and spasmus nutans; but strongly opposed to both is the occurrence of the movements during sleep, so that Zappert supposes that they are more allied to the conditions described by Binder and Cahen, that they begin as voluntary conscious movements before sleep, and then become stereotyped, so as subsequently to become automatic and unconscious.

As I have already said, nystagmus is usually present, and, like the head movements, it may be almost continuous, or may occur from time to time at varying intervals. It may be vertical, horizontal, or rotatory; and, although usually present in both, one eye may be affected alone; even when present in both, it is usually more pronounced in the one eye than in the other; moreover, the character of the nystagmus may be different in the two eyes.

The nystagmus may come on simultaneously with the head movements, may precede them, or be the only clinical manifestation of the affection. When nystagmus and head movements are associated at the same period of the affection, they may occur simultaneously, or the two conditions may alternate, so that at one moment head movements are seen, and at the next they are replaced by nystagmus. The nystagmus is much more rapid than the head movements, and has an independent rhythm even when the two phenomena occur simultaneously. The usual rate of nystagmus is about four to six movements per second. It cannot be said that any one variety of nystagmus is constantly related to any given movement of the head (as, for instance, vertical with nodding, and horizontal with lateral movements); for, although in some cases such a relation obtains, in others every variety of combination is met with. When the nystagmus is unilateral the head may move either to that side or away from it.

The nystagmus, when absent, may be evoked by any attempts at fixation, and also by forcibly restraining the head movements; so, too, either of these devices will accentuate nystagmus already present. It has been noticed further that the nystagmus may be more pronounced in certain positions of the eyes than in others; thus, in one of Hadden's cases it was most marked when the eyes were directed to one side, and least so when they were turned in the opposite direction; in another child the nystagmus was especially evident when the eyes were directed upwards. Moreover, the character of the nystagmus may change with alteration of the position of the eyes, as in Gordon Norrie's case, in which a horizontal nystagmus tended to become vertical when the eyes were turned upwards. The eyelids shared in the movements of the globes in one of Hadden's cases.

Another ocular defect that has been met with in some cases of head-nodding is strabismus, which appears usually to be convergent. Instances of this association have been reported by Henoch, Gee, Hadden, Peterson, and Mouat; and slight transient squint was noted by Aldrich. In one of Hadden's cases, in which nystagmus was absent, the movements of the head were preceded by lateral deviation of the eyes.

The pupils usually present no abnormality, though the condition of hippus was present in Norrie's case and in one of Hadden's. The latter observer also found the reaction of the pupils on convergence poor in one case, and in another in which nystagmus was limited to one eye the pupil on that side acted better consensually than on direct stimulation. In all the cases examined the discs have been found normal; in two of Hadden's patients the fundus was light in colour; in one of these there was a slight crescent of atrophy round the disc, in the other there was a deficiency of retinal pigment.

I have said that there has been reason to suspect faulty vision in some cases, though careful testing of sight has failed to establish any defect of the kind. The suspicion arose in three of Hadden's patients, but in none of them was Marcus Gunn able to determine any defects of sight or abnormal ophthalmoscopic appearances. Nevertheless, a characteristic feature of the malady is the peculiar way in which the child looks at objects out of the corners of its eyes, and tilts its head backwards and slightly to the opposite side. The child is liable to stare in an absent-minded manner from time to time, but does not change colour, lose consciousness, or present any other epileptiform manifestations. In none of these cases has a personal or family history of convulsions been obtained, and none of them has been known to fall into genuine epilepsy later in life.

Mentally the children are usually particularly bright, in some instances even precocious, taking notice unusually early; on the other hand, in a few exceptional cases, there has been distinct mental defect. In the latter case, it has usually been that the child, intelligent and happy when it first came under observation for the head movements, subsequently became cross and passionate, or less intelligent, irritable, and forgetful; in one of Hadden's cases, a child whose intelligence was above the average had occasional outbursts of laughter. But so far as can be ascertained, even in cases of this kind, no permanent mental defect results.

Diagnosis.—The affection must not be confused with a rare congenital affection in which the head-nodding and nystagmus are permanent and not transitory, as in spasmus nutans. It must also be distinguished from head-rolling, which appears to be a voluntary movement, is not associated with nystagmus, and occurs chiefly when the child is recumbent. A form of epilepsy, spoken of as eclampsia nutans, in which the head is jerked forward, has been confused with spasmus nutans, but the absence of loss of consciousness, change of colour, and convulsions should serve to prevent mistakes (*vide* also p. 659).

Prognosis is highly satisfactory; in time total cessation both of the head movement and of the nystagmus has been noted in the majority of the cases, though the latter usually persists longer than the former. The duration, however, varies in different cases, and whereas in some all movements may cease in the course of a few weeks, in others they continue for months. Moreover, after they have ceased, they tend to recur under any exciting or debilitating influence; or the nystagmus may recur without the head movements. In one of Hadden's cases nystagmus was still present after a period of seventeen months.

Treatment.—It is probable that without treatment most of these cases would end in recovery in the course of a few months, or it might be of years; but they certainly appear to be favourably influenced by treatment. If any exciting cause is determined it must be removed. Irrespective of what view is held as regards the cause, there can be no doubt that fresh air, sunshine, and improved hygienic conditions in general are important factors in the treatment. The drug which appears to be of most service is one of the bromide salts, given in doses of one and a half to three grains, three times a day, to infants at the age when the affection is most commonly met with. Professor Still finds phenazone better in some cases, alone or combined with bromide. Belladonna may be combined with the bromide, and later steel wine or syrup of the phosphate of iron may be substituted for sedatives. The latter measures are especially indicated in cases in which nutrition is poor, or when there are any evidences of rickets, when cod-liver oil or some preparation of malt will also be found useful; whilst, in support of his view that rickets is the cause of the affection, Kassowitz considers phosphorus beneficial in its treatment.

J. S. Risien Russell.

REFERENCES

1. Aldrich. *Amer. Journ. Med. Sc.*, Phila., 1899, cxvii. 158.—2. Bannatyne. *Lancet*, Lond., 1895, ii. 1163.—3. Buzzard, T. *Ibid.*, 1886, i. 834.—4. Dickson. *Ophthal. Rev.*, Lond., 1895, xiv. 845.—5. Gee. *St. Barth. Hosp. Rep.*, Lond., 1886, xxii. 26.—6. Hadden. *Lancet*, Lond., 1890, i. 1293, 1349, 1416.—7. *Idem. St. Thomas's Hosp. Rep.*, Lond., 1890, xx. 205.—8. Henoch. *Lect. on Dis. of Child.*, New

Syd. Soc., Transl., Lond., 1889, 192.—9. Hirsch. *Med. News*, N.Y., 1898, i. 538.—10. Hochsinger. *Jahrb. f. Kinderh.*, Leipz., 1905, lxii. 562.—11. Jackson, Hughlings. *Lancet*, Lond., 1889, ii. 839.—12. Lange. *Verhandl. d. Versamml. d. Gesellsch. f. Kinderh. deutsch. Naturf. u. Ärzte*, 1897, Wiesb., 1898, xiv. 231.—13. M'Gillivray. *Ophthal. Rev.*, Lond., 1895, xiv. 252.—14. Mackenzie. *Lancet*, Lond., 1886, i. 833.—15. Mills. *Amer. Textbook Dis. of Child.* (Starr), 2nd edit., Phila., 1898, 713.—16. Mouat. *Lancet*, 1906, ii. 397.—17. Norrie. *Centralbl. f. prakt. Augenh.*, Leipz., 1888, xii. 229.—18. *Idem. Ophthal. Rev.*, Lond., 1888, vii. 355.—19. Oddo. *Bull. méd.*, Paris, 1898, xii. 953.—20. Osler. *On Chorea and Choreiform Affections*, Lond., 1894, 89.—21. Peterson. *Med. News*, Phila., 1892. lxi. 374.—22. Raudnitz. *Jahrb. f. Kinderh.*, Leipz., 1897, xlv. 145.—23. *Idem. Verhandl. d. Gesells. f. Kinderh.*, 1902, 132.—24. Rietschel. *Charité-Ann.*, Berl., 1906, xxx. 152.—25. Romberg und Henoch. *Klinische Wahrnehmungen und Beobachtungen*, Berl., 1851.—26. Soltmann. Gerhardt's *Handb. d. Kinderkrankh.*, Tübingen, 1877, i. 197.—27. Stamm. *Arch. f. Kinderh.*, Stuttg., 1901, xxxii. 259.—28. Still. *Lancet*, Lond., 1906, ii. 207.—29. *Idem. Clin. Journ.*, Lond., 1906, xxix. 90.—30. Thomson, J. *Festschrift in Honor of A. Jacobi*, N.Y., 1900, 65.—31. Wholey. *Journ. Amer. Med. Assoc.*, Chicago, 1907, xlviii. 475.—32. Wilson. *New York Med. Journ.*, 1898, lxviii. 770.—33. Zappert. *Jahrb. f. Kinderh.*, Leipz., 1905, lxii. 70.

J. S. R. R.

OCCUPATION-NEUROSES[1]

Synonym.—*Craft-Neuroses.*

By Henry Head, M.D., F.R.S.

Definition.—Occupation-neuroses may be defined as affections of the nervous system produced by a handicraft, and interfering with the patient's professional activity.

Introduction.—Now it is obvious that such disability may depend ultimately upon many different causes; and yet, in spite of profound etiological differences, we classify these neuroses together, because in every case the patient complains that he is thereby incapacitated from carrying out his daily work.

The occupations dealt with in this article are complicated acts brought to perfection by education and practice. They have been acquired by voluntary effort, long sustained and carefully directed, but finally become semi-automatic. They have been developed by forcing a neuro-muscular mechanism originally developed for locomotion or prehension to carry out the highest acts invented by civilised man. We cannot wonder, therefore, that the slightest disturbance in this mechanism will destroy these highly complex acts which require for their perfect performance a facility not even possessed by man in his uncivilised state. To a clerk the cause of his inability to write is a matter of no material moment; his fingers refuse to obey his will, and he cannot earn his living. But to the physician the cause is all important. Attempts have been made to place the fault in the muscles, in the nerves, in the cerebral innervation, or even in psychical abnormalities. All attempts to bring these occupation-neuroses under one anatomical or physiological category have failed. We shall, therefore, deal with each professional disability separately, indicating the various causes which may underlie it.

---

[1] Throughout this article much use has been made of the account of these neuroses given by the late Dr. G. Vivian Poore in the first edition of this work (1899, VIII. 3-26).

It has often been stated that the particular cramp, spasm, or pain of which the patient complains is evoked solely by his professional activity. This may be so in a limited number of instances, but in the majority it is certainly untrue. Acts which do not affect the same neuro-muscular mechanism could not be expected to cause pain or cramp, and in the larger number of occupation-neuroses no other non-professional act can be found which throws the same muscles into play.

Examination of the Patient.—However healthy he may appear, every patient complaining of an occupation-neurosis should undergo a complete examination as if for life assurance. By this means the examiner will detect a neurotic heredity, or obtain a history of some congenital or acquired nervous fault. Abnormalities in speech or gait, hemiparesis or monoplegia, apraxia, abnormalities of the pupils or knee-jerks, will be at once discovered by a routine examination of the nervous system. Local muscular wasting, general tremor, or ataxia must be sought for in all four limbs. The condition of the blood-vessels must be carefully noted, and the possibility of renal inadequacy considered, particularly with a view to prognosis. The discovery of alcoholic excess or habitual drug-taking, especially for insomnia, may be all-important for treatment.

Having excluded degenerative changes, congenital defects, and ordinary bodily disease, we next examine the patient for the cause of his special defect. After he is stripped to the waist, the movements of the shoulders and scapulae are first examined. When the arms are raised above the head, and the palms are brought together at the middle line, the position of the two arms is examined; a want of symmetry may betray muscular weakness on one side. Then the patient must be made to shrug the shoulders, to make the shoulder-blades meet by trussing back the elbows, and to hold the arms in front of him at the level of the shoulders. In this way any defective action of the trapezius, levator anguli scapulae, rhomboids, and serratus magnus is made manifest. While the arm is extended at the level of the shoulders, both limbs must be narrowly scanned for tremor. Then the arms, forearms, and hands should be examined for general want of nutrition, and for wasting of particular muscles, especially in the hands between the metacarpal bones, and in the ball of the thumb. The grasp should be noted, and the movements of the hand and fingers carefully tested.

Next, we must ascertain if any of the nerve-trunks of the arms are definitely tender. This examination requires care, because normal nerve-trunks are somewhat tender, and it is only by comparison of one side with the other that we can arrive at any useful conclusion. The patient stands facing the examiner, with both hands resting, palms upwards, on the examiner's shoulders. It is important that the patient's arms should be really at rest, and as free from all muscular efforts as possible. The physician then examines the nerve-trunks of both limbs simultaneously by means of gentle pressure on the ulnars behind the olecranon, on the musculo-spirals at the inner borders of the supinator longus, and on the median at the elbows, on the inner side of the biceps tendon. The simultaneous examination of the medians can only be made if the examiner crosses his hands. If the examination of the nerve-trunks on the two sides be made simultaneously, there is greater likelihood of the pressure being equal; and if the examiner find a distinct difference in the tenderness of the nerves, he is justified in recording it as a symptom of importance. The patient is, of course, in ignorance of the object of the examination, and when the nerve-tenderness is well-marked his response to a gentle pressure of the thumb is startling alike to examiner and examinee. This comparison of the two sides can only be trustworthy when the nutrition of the two is equal; if one arm be wasted, the comparison is no longer possible. The search for tender nerves is important in all these cases; and not seldom one of the palmar twigs of the median or ulnar is found to be tender, or tenderness is detected along the course of the suprascapular, circumflex, or anterior thoracic nerves.

Sensation should then be carefully tested in the affected limb. Sensibility to touch, pain, and temperature will not in all probability be abolished, but the patient may complain that a touch with cotton-wool, or the sensation produced by dragging the point of the pin gently over the skin, is abnormal over certain parts of the affected limb. Care must be taken to compare the effect of these tests applied to similar parts of the two limbs.

Attention must next be directed to the bones, joints, and ligaments. A painful or creaking shoulder-joint with incipient ankylosis, stiffness of the elbow or wrist, and nodules on the phalanges, have all to be looked for. While investigating the mobility of the joints, knowledge will be gained of the condition of the tendons. A small amount of teno-synovitis will greatly interfere with function.

The patient, still stripped to the waist in a warm room, should now be made to carry out the particular act of which he complains. If it be to write, give him a sheet of paper and a good pen, see that he has a comfortable chair and table, and ask him to write his name and address, and the date. A great deal may often be learnt by watching his attempts to perform the deranged action. Occasionally nothing amiss is to be detected, except perhaps that the writing may be produced rather slowly. The patient may tell you that the effort to write causes pain; but, so far as any change in the mode of production or in the product itself is concerned, there is nothing amiss. This, of course, is important both in diagnosis and prognosis. At the other end of the scale is the man who is scarcely able to make a mark upon paper; whose efforts are accompanied by most tumultuous movements of the shoulder; who is obliged to fix his arm securely before beginning to write; who grasps the pen with all his might, and perhaps drips with perspiration as he makes an abortive attempt to sign his name. It often happens that the ordinary method of holding the pen is altered; if this be so, the patient should be asked to write with the pen held in the conventional manner, when his difficulties will be accentuated. For it is not the "faculty" of writing which is deranged, but only certain methods of executing it. In well-marked cases the pen is grasped very tightly, the wrist and forearm do not rest on the desk or table, and, in very severe and long-established cases, there is much tumultuous action of the shoulder-muscles. There are one or two points in these disorderly movements which deserve attention, one being the conduct of the right index-finger. The patient often complains that he cannot write if the index-finger be placed upon the penholder; and, when he tries to write in this way, it will be observed that the index-finger ceases after a time to press upon the penholder, slowly leaving it and slipping upwards until the pen evades his grasp. This, the most common of all the failures in writing, will receive further consideration (p. 673).

It is most important, if possible, to make each patient bring the tool or instrument with which he works. It may happen that some fault is at once evident which can be corrected, with temporary or permanent benefit, and the patient is rarely able to describe phenomena, evident to the examiner.

Lastly, in most cases it is wise to compare the electrical reactions of the affected groups of muscles with those of the opposite limb. A loss of reaction to faradism, with a slow reaction to the galvanic current, the so-called reaction of degeneration, is never found in uncomplicated cases of occupation-neuroses. The presence of such changes will, therefore, serve to warn the physician that the symptoms of which the patient complains are a part only of some organic disease. Dr. Poore maintained that in a very large percentage of cases of writers' cramp and allied neuroses a faradic current which evokes a vigorous contraction from the sound limb has no such effect when applied under exactly similar conditions on the diseased side. He noticed a similar diminution in the galvanic excitability. This change is, with rare exceptions, confined to the small muscles of the hand. Of the interossei the first dorsal was affected far more frequently than the others, and may be regarded as the muscle in which this impairment of irritability to faradism can be most readily demonstrated.

"Writers' Cramp."—The profession in which, above all others, such impotency is prone to declare itself is that of the clerk. Cases of difficulty in writing are far more common than all the other occupation-neuroses taken together. Dr. Poore stated that out of many hundred cases of such disabilities of various forms, at least 90 per cent were cases of "writing difficulty." This numerical preponderance is due in part, no doubt, to the fact that the occupation of the clerk outnumbers all other occupations in which special neuroses are apt to occur. Writing, to a greater or less extent, is the "occupation" of all educated persons, a fact which alone is sufficient to account for a preponderance of palsy in writing;

nevertheless, there is no doubt that the act of writing is, in its nature, one peculiarly liable to derangement.

A man who takes a pen into his hand and rapidly, almost automatically, covers a sheet of paper with orderly sentences, is apt to forget the labour and sorrow of his writing lessons. Those who can recall their own early efforts, or who closely watch a little child receiving its first instruction in writing, will recognise that the "faculty" is a painfully acquired art. The little child can succeed in making his clumsy pot-hooks only by dint of considerable muscular exertion; and the boy or girl who is able to wield a knife and fork without obvious effort has to strain every muscle in order to obtain that necessary combination of firmness and mobility which goes to the production of the simplest written characters. Some persons, even amongst the educated classes, never learn to write anything but a painfully childish hand. In order that the shoulder of the writing arm may work from a fixed point, the trunk needs a maximum amount of support by the child; and this end he attains by twisting one or both feet round the legs of the chair, and by leaning with the left arm on the desk or table. If uncontrolled, the child is tolerably sure to rest his right elbow and the whole of the forearm on the table in order to afford a fixed point for wrist-movement, and the progress of the pen is generally accompanied by associated movements of the muscles of the opposite hand, the face, the tongue, and probably of the feet and toes also. In short, when the child attempts to write, almost every muscle of the body is engaged more or less in the effort.

Finally, the child is taught to write with the pen held firmly between the thumb, index, and middle fingers, and with the shaft pointing towards the right shoulder. The little finger rests firmly on the paper, and each stroke is made by flexing and extending the fingers and thumb that grasp the pen. The pen is both held and moved by the action of the same small muscles. Moreover, many clerks habitually use a hard steel nib which necessitates the exertion of considerable pressure to produce that difference between up and down strokes so admired by the calligraphist.

Such a method of carrying out a series of fine and complex movements, recently acquired in the history of the race, is certain to lead to fatigue and disability, provided that the patient is compelled to write much and with rapidity. But I have never seen a case of primary writers' cramp in any one who habitually used the fingers for holding the pen only, and made all movements by which the letters were formed by means of the forearm or wrist. The most vicious feature in the act of writing, as taught by the writing-master, is the use of the same groups of small muscles for both guiding and holding the pen. I have never seen a case of writers' cramp in an artist who used the pen like a brush.

Writers' cramp is much commoner in men than in women, solely because excessive writing is more liable to form part of the daily work of a man than of a woman. I saw a lady who had developed this condition in consequence of the amount of writing entailed by her daughter's marriage.

The disease in its primary form appears, as a rule, between the ages of twenty-five and forty-five; in old persons some general degenerative change should always be suspected. The bulk of the patients are in the prime of life, and it is in vigorous adults that we are most likely to find true writers' cramp.

Hereditary constitution plays a considerable part among the causes of this condition. By this we do not mean that writers' cramp is handed down like accessory fingers and toes, but that as the handwriting of father and son may be almost indistinguishable, so the method of using a pen may be identical. This method of holding and guiding the pen is responsible for the development of chronic fatigue and so, ultimately, of the difficulty in writing. Moreover, the son whose method of writing so closely resembles that of his father will probably inherit his neuropathic nature, and be liable to become easily exhausted, irritable, and sleepless. I have seen one instance in which the father kept a journal and, before the development of writers' cramp, his writing was almost identical with that of his son; both developed difficulty in writing at the age of forty-four. Both father and son wrote a beautiful hand, holding the pen in the conventional manner. Where the difficulty in writing supervenes on shock or psychasthenic states, collateral neuroses will be found, such as tic, neurasthenia, epilepsy, eccentricity, and inability to earn a living. This is the so-called "neuropathic heredity."

Attempts have been made to classify "writers' cramp" according to the form it may assume. Thus, a "spasmodic," a "tremulous," and a "neuralgic" variety have been described. But, although such names express the leading symptom, they have no etiological or practical significance. Since writers' cramp may arise under extremely diverse conditions any classification, to be useful clinically, must take notice of its cause rather than of the form of difficulty in writing. I propose, therefore, to consider writers' cramp under four main groups. (1) The primary form, which arises in a patient otherwise in perfect health. Here at first the difficulty occurs only when the patient writes. (2) The form in which the difficulty in writing arises as a secondary consequence of some condition causing neuro-muscular weakness of the right arm, such as an injury or an attack of neuritis. (3) Those cases in which the difficulty in writing is a symptom of some general organic or functional disease, such as arthritis, senility, paralysis agitans, or neurasthenia. (4) Cases in which the difficulty in writing is due to psychical causes, such as fear, or belongs to that form of motor disturbance of mental origin which we now call tic.

1. *Primary Writers' Cramp.*—The following case is a characteristic instance of this form of the disease:—

A merchant, of healthy parentage, began to find at the age of thirty-five that it was increasingly difficult to hold his pen. After writing for some time his index-finger left the holder. He strove hard to hold the pen between the thumb and middle finger, but ultimately one or other would also become extended and the pen would fall from his hand. He then learnt to write with his left hand; but ten years later an exactly similar condition came on in the fingers of this limb also. He had no pain except an occasional aching in the small muscles of the right or of the left hand if he wrote much. He held his pen in the usual pernicious manner and pressed hard on the shaft at the same time, making all movements with the small muscles of the fingers. He had no difficulty in using either hand for any other movement however delicate, except writing, and could shave perfectly. He was absolutely healthy in every way except that his hair had become white somewhat early and his arteries were perhaps a little hard for his age.

Frequently the index-finger becomes pressed more and more firmly against the pen until at last the violent flexion at all joints thrusts the point of the pen into the paper, or the holder is propelled from between the fingers and thumb.

The amount of pain in these cases varies greatly. In some, the act of writing is accompanied by a dull aching in the small muscles of the first interosseous space, or in the extensors of the forearm, which disappears entirely if the hand is not used for writing. Sometimes this pain is the main feature of the disease. As soon as the patient takes a pen into his hand the pain begins, and rapidly becomes so intolerable that he is compelled to cease writing. In the earlier stages of such cases the pain is always situated in some part of the limb below the elbow.

Sometimes the disability takes the form of a coarse tremor. Immediately the pen is held tightly between the thumb, index, and middle fingers, coarse tremulous movements begin, usually in the long extensors of the fingers and radial extensors of the wrist, of such violence that the writing becomes a meaningless scrawl. This form of disability is almost always associated with a good deal of aching, and even tenderness of the muscles affected by the tremor. Sometimes this primary disability will come on without excessive writing, or at an early age. In such cases we are frequently told that one of the parents suffered from the same complaint. I saw a young man of eighteen who developed writers' cramp after a civil service examination. In a gentleman, director of a family business, true writers' cramp came on at the age of twenty-eight from signing cheques; his father had suffered from the same disease, and he had, therefore, abstained from other writing and employed a secretary or a typewriter.

If the difficulty in writing belongs to this primary group the prognosis is absolutely bad. The patient will never be able to write again for long periods. After a long rest from writing, with massage or treatment with the constant current, he may regain his power of writing with a pencil or signing his name legibly to cheques with a pen, but he will never be able to make use of writing for professional purposes. It is therefore of the greatest importance to diagnose correctly whether the difficulty is primary or secondary. This will be considered on p. 674.

2. *Secondary to some Conditions causing Neuro-muscular Weakness of the Arm.*—A young clerk, riding home on his bicycle one night, had a bad

side-slip. Next day his right arm ached, especially after taking down a number of letters in shorthand. Gradually the general pain and aching passed away, but writing, and especially shorthand, with which he was much occupied, caused pain in the muscles of the forearm. Now it is characteristic of the free method of writing necessary for shorthand that it never leads to primary writers' cramp. Moreover, this young man had pain when he shaved, and particularly when he put on the brake of his bicycle with the fingers of the right hand. In the same way one of my colleagues developed a gradual difficulty in writing after fracturing his clavicle in a bicycle accident. In both cases two months' rest from writing led to absolute recovery.

Sometimes a man whose profession necessitates much writing is seized with an attack of what is usually called brachial neuritis. He has probably had previous warnings in the form of lumbago or pains in the sciatic region. The pain generally lies over the pre-axial portion of the arm, forearm, and hand, including the thumb and thenar eminence. This necessitates a few days' rest, and he is then forced to resume his business. Gradually the pain becomes peculiarly associated with the act of writing, although it can be evoked equally well by a day's golfing. Tender points will usually be found about the shoulder, arm, or forearm; and the main nerve-trunks, such as the musculo-spiral, may or may not be tender.

A young musician developed an attack of pain over the pre-axial aspect of the forearm during the composition of an opera. He rested for a few weeks, but found that the pain came on again whenever he worked at his score, and, finally, writing became impossible. But this pain could also be evoked by carrying a heavy teapot or by bicycling. The nerve-trunks were not tender, and I could not find any change in sensation or in the electrical reactions of the muscles. He entirely recovered in consequence of the help given him by two of his friends, who became his amanuenses until the work was finished.

In this secondary form the prognosis is good, provided the patient can rest the arm entirely for three months. As the prognosis is so different in the primary and secondary forms, it is important to differentiate clearly between them. The following points will help to distinguish them from one another.

The primary writing-disability, whether it take the form of spasm, tremor, or pain, has arisen gradually during the act of writing. In the early stage it cannot be evoked by other widely-diffused movements such as shaving, carrying a cup, or golfing. The pain is usually situated in the forearm or hand.

The secondary form has arisen as the sequel to an attack of brachial neuritis, neuralgic pain, or to an injury. Writing causes more discomfort and unsteadiness than any other activity, but shaving is difficult, and carrying a weight or golfing also evokes pain. Moreover, the pain is not uncommonly situated not only in the forearm but in the arm and shoulder.

3. *The Difficulty in Writing may be a Symptom of some General Disease.*—Arthritis is a not uncommon cause of difficulty in writing both in young and middle-aged professional women. A young schoolmistress who taught writing and games consulted me for intense pain which went up her arm to the shoulder and prevented her from writing. This was particularly the case after she had played hockey. But it disappeared entirely in the holidays, when she neither wrote nor played games. Her wrist shewed early arthritic changes, and the interphalangeal joints of the right hand were swollen and tender to manipulation. A similar difficulty in writing may arise in elderly persons in consequence of gouty changes in the joints.

Occasionally tabes dorsalis manifests itself first as a difficulty in writing. But in this case, and also where the handwriting has become gravely affected by dementia paralytica (general paralysis of the insane), other signs point to the true nature of the disease.

But paralysis agitans, or the onset of a senile tremor, may lead to great difficulty in diagnosis. In an elderly patient of my own the tremor which destroyed the clearness of his writing was also evoked by an attempt to button his clothes or when he used a knife and fork. It was also present, though to a less degree, in the left hand which he never used for writing.

The general fatigue which is the essence of neurasthenia is liable to produce a difficulty in writing. But here there is no spasm and little pain. The tremor, if present, is purely the sign of fatigue, and the patient's handwriting undergoes little or no alteration.

Thus, a lady, well known in the world of art, found she was less and less able to write without producing a feeling of intense nervous fatigue. This condition came on after anxiety and worry. After about ten lines her writing gradually shewed signs of fatigue; it sprawled across the paper, the o's and the a's were left widely open, and the letters at the end of the word were slurred. But there was no tremor, no cramp, and no pain along the nerves or in the muscles. When at her worst, she could paint as well as ever, and she recovered entirely with rest and regulation of her life.

4. *Difficulty in Writing due to Mental Causes.*—Our knowledge of this group is due entirely to the work of French observers who have placed the subject of the tics on a sound basis. A tic is a co-ordinated purposive movement of the voluntary type which serves no direct object. At first it may arise in response to peripheral irritation as an expression of emotion or as an associated habit. But ultimately the desire to carry out the particular movement of which the tic consists becomes intense apart from the conditions under which it may have started. The essential factor in all tics, that have not sunk down to the level of unconscious habits, is the desire to perform a certain movement which precedes the act itself. This can be controlled to a varying extent and, unlike a chorea, is less frequent and troublesome under observation.

A tic may be occasionally of such a nature that it affects writing.

A young woman under my care suffered from an habitual movement which consisted of flexing the elbow and extending the wrist. This greatly interfered with her writing, but also prevented her from sewing, from playing the piano, and from using a typewriter effectually. She also had difficulty in holding a tea-cup without spilling the contents. In all cases the actual movement was preceded by the desire to make it and the fear of consequences. She was an excitable young woman of twenty-five, and as a girl of twelve had suffered from a nodding and a blinking tic. The movement of which she complained when she came to me began when she was at school, in consequence of anxiety about writing and playing the piano. But it did not become a serious hindrance until she took up typewriting as a profession.

In most cases the tics which interfere with writing or other professional occupations arise in consequence of some emotional disturbance or discomfort. But fear can have an inhibiting effect upon writing even without leading to the development of an habitual movement such as a tic.

I saw a shorthand clerk who was frightened of his principal ; so long as he worked in the outer office he could write with perfect ease. But as soon as he was called in to the head of the firm his power of writing in shorthand to dictation became slower and slower. Finally his hand was so unsteady that the characters became completely illegible. At the same time the long extensor muscles of his right forearm became tired and hot. He was taken off the duty of working with this particular member of the firm and found he could write with ease. Then the fear and anxiety were removed by suggestion, and he is now able to carry out all the duties of his profession.

Typewriters' Cramp.—A true occupation-neurosis due to typewriting must be uncommon ; for, in spite of the large number of women engaged in this occupation, I have seen one case only.

A woman, aged forty-five, began to notice pain at the base of her right thumb and over the first metacarpal. She then used the left thumb for the space bar ; but this soon produced a similar feeling in the left hand. After long spells of work both forearms seemed to become stiff or "cramped," and this feeling sometimes extended as high as the shoulder. The fingers lost power and refused to work unless forced to do so. This use of the will had a most distressing effect upon her, and produced great general fatigue. The nerves were not tender, and there were no sensory changes. The arms always felt heavy and tired, but she could sew or play golf without difficulty. She was a nervous woman, overworked, and earning her living with difficulty.

De Lépinay gives an instance in which the disability took the form of a sudden straightening of one or more fingers with sudden flexion or extension of the wrist. This patient had previously suffered from writers' cramp.

In the greater number of cases in which a typewriter becomes unable to use her instrument she complains of pain. Moreover, this pain will usually be found to be associated with some general state ; it will be evoked by occupations other than typewriting, and will be most prominent on writing. In fact, the difficulty usually corresponds to that we have called secondary writers' cramp (p. 673).

Telegraphists' Cramp.—Telegraphists who use the Morse key may suffer from tremor or pain on using the instrument. An occupation-neurosis, the exact equivalent of writers' cramp, due to the use of the Morse key, undoubtedly exists. Moreover, this telegraphists' cramp is almost invariably accompanied by writers' cramp, which shews itself when a message is transcribed. The telegraphist has not only difficulty in using the key, but suffers from an equivalent difficulty when he attempts to write. This telegraphists' cramp may take the form of tremor, of inability to keep the finger on the key, or of pain. Not infrequently all these affections are present in the one case. Of the two cases, described by the late Dr. Poore, the following was the more satisfactory—

A man, aged thirty-nine, had worked a Morse key for nineteen years. He had as much difficulty in writing as in telegraphing, and it was noticeable that all delicate acts—such as shaving, holding a tea-cup, or wielding a salt-spoon—were seriously impeded by tremor. The tremor affected all the muscles of the right upper limb, including the pectoralis major; but it was most marked when the hand was prone in the telegraphist's position. The median and musculo-spiral nerves on the right side were distinctly tender.

I have seen a man, aged twenty-six, otherwise healthy, who complained of a "cramped feeling" in the right arm when using the Morse key. But he said this was even worse when he attempted to write. He was a telegraphist, and three months before I saw him began to notice pain across the back of the hand and up both flexor and extensor aspects of the right forearm. He was free from this cramping pain on Monday morning and on a holiday, but it grew worse as the week advanced. The nerves were not tender, sensation was unaffected, and the electrical reactions were normal.

Pianists' Cramp.—Closely allied to the two previous conditions is pianists' cramp. Here, too, the disability may assume different forms. Dr. Poore gave a description of nine cases; in two of them the trouble consisted in a difficulty in executing some movement of the fingers, whilst seven patients complained of pain and consequent weakness of the arm.

I saw a young woman of twenty who, for four years, had been a student of music and had practised the piano for many hours daily. She complained of weakness in the fingers when she attempted to strike the notes, and when she tried to play the piano her forearm ached. She indicated the extensors in the forearm as the seat of the greatest aching. I could not find any abnormal signs; the nerve-trunks were not tender, sensation was unaffected, and the muscles reacted normally.

In one of Dr. Poore's cases all the fingers of the left hand were said "to run together" when playing staccato passages, owing, apparently, to a failure of the extensor communis digitorum to raise them steadily in rhythmic sequence. Another patient complained that "the left forefinger would not keep off the keys." De Lépinay describes the case of a musician who noticed that he could not extend the little finger of the right hand when playing the oboe. Later the ring-finger failed in a similar manner, and he also found difficulty in playing the piano. Finally all the fingers of the right hand became affected, and he ceased to be able to use either instrument. He could perform a large number of other movements with this hand, but careful examination revealed that all acts demanding an orderly activity of the extensors were badly performed. Thus, when writing he held the pen in a peculiar manner. Brissaud believed that this case was one of neuritis, mainly of the musculo-spiral nerve, without sensory changes. Here again we find the same tendency for the pianists' "cramp" to be primary or secondary. The primary variety may assume the form of debility of movement, tremor, or pain. The secondary form may depend on neuritis, neuralgia, or a number of general diseases.

Violinists' Cramp.—Every violinist develops corns on the index and middle fingers of his left hand. These corns may become tender and cause radiating pain up the arm, which prevents him from pressing his fingers firmly on the string. In one instance, tenderness of these corns was found to be the cause of a supposed violinists' cramp. But a true occupation-neurosis analogous to primary writers' cramp may occur in those who play a stringed instrument. Such a condition, however, cannot be of common occurrence, for Dr. Poore gives one case only, although he described several instances of inability to play the violin or violoncello, arising from various local or general conditions. Tenosynovitis, neuritis, and occasionally a form of injury to the nerve-twigs as they pass through the extensor muscles at the elbow (p. 680) are more common causes of difficulty in playing the violin. In all such cases other movements of the hand, particularly grasping with the fingers, will produce pain and, apart from this pain, the orderly sequence of movements remains undisturbed.

Sewing Cramp.—Dr. Poore described an excellent example of this condition exactly analogous to primary writers' cramp.

The patient was a tailor, aged fifty-seven, well-built and temperate, and free from all general disease. For about a year he had experienced "weakness" of the right thumb and forefinger, which made it difficult for him to ply his needle. He held it in the usual way between the thumb and index-finger, with the upper end against the thimble worn on the middle finger. After he had made a few stitches through the cloth the needle eluded his grasp. The thumb and forefinger gave way, and the needle was no longer grasped with sufficient firmness to push it through the fabric. Dr. Poore stated that the median nerve was tender, and that a tender spot was found at the base of the thenar eminence. Sensation was duller over the "median" area of the right palm and the irritability to the faradic current of the first dorsal interosseous and of the muscles of the thenar eminence was less than that of similar muscles in the left hand.

This case was an excellent instance of a "fatigue" disease in which the difficulty in muscular action only occurred when the patient attempted to carry out the movement that had produced the chronic fatigue.

Muscular Weakness produced by the Use of the Scissors.—Tailors' cutters who use scissors of large size are liable to suffer from a weakness of the thumb and fingers of the right hand. But this condition seems to be produced by pressure upon the nerves to the thenar eminence and to the skin of the back of the thumb by the handle of the scissors. For in the case reported by Savill the muscles of the thenar eminence were wasted, and the dorsum of the thumb was partly anaesthetic. This disability bears no relation to the true occupation-neuroses.

Muscular Inability associated with the Use of the Hammer.—Since Duchenne first described the case of a tin-worker who suffered from a painful contraction of the deltoid and biceps when holding his hammer, "hammermen's cramp" has been described as an occupation-neurosis comparable to writers' cramp. But examination of the published cases of this disease shews that most of them certainly do not belong to this group. The "hephaestic palsy" of Frank Smith seems to have been nothing more than hemiplegia or monoplegia of cerebral origin occurring in men who used the hammer. Dr. Poore's case of gold-beaters' cramp, according to his own diagnosis, was one of senile tremor occurring in an overworked and ill-nourished man with degenerated arteries. It must not be classed with the true professional ailments caused by fatigue, in which an otherwise strong and healthy subject finds himself incapable of some one delicate manipulation. De Lépinay's case comes nearer to being a true occupation-neurosis. A tin-worker began to suffer from a progressive difficulty in movements of the arm and shoulder when directed towards the use of the hammer. It produced great pain and a sensation of extreme fatigue.

I have seen many blacksmiths and men accustomed to use a hammer who complained that they could not hold it in one or other hand. But in every case the disability depended on the presence of pain in the neighbourhood of the elbow, evoked by attempting to grasp anything firmly in the hand. This condition seems to be a distinct clinical entity of frequent occurrence, but, so far as I have been able to discover, it has not been definitely described. Many cases of "hammermen's cramp" seem to have been instances of this disease, which I believe to be closely allied to meralgia paraesthetica in the leg (Bernhardt's disease) (*vide* also Vol. VII. p. 400).

The patient begins to complain of pain situated over the insertion of the long extensors of the fingers just below the external condyle. The pain is never entirely absent, but is greatly increased when he attempts to grasp with the hand of the affected extremity. Should the patient's occupation necessitate any movement in which the long extensors participate, he will complain of professional disability. Thus, I have seen a blacksmith, a ladler of metal for stereotyping, and a Government clerk all of whom suffered from this complaint; the first could not use his hammer, the second his ladle, and the third his pen, without bringing on the pain so severely that work became impossible. The Government clerk gave the following history of the onset of the pain. Coming home one winter night from a week-end in the country, he arrived at the station to find no cab. He was therefore obliged to carry a heavy bag some distance to his home. Next morning he noticed pain over the extensors of the forearm, which was easier when the arm was flexed. It was greatly increased by writing or by handing a plate: grasping the spade while gardening produced severe pain. It gradually improved to such an extent that he was free from pain unless he wrote for long periods or performed any act which necessitated grasping. In these cases the pain seems also to produce a curious inhibiting effect upon the strength of the grasp, so that the patient complains that his hand "feels weak"; and yet there is no weakness of any muscles of the hand. Extension is both painful and difficult, and so the wrist and fingers are no longer fixed, and the flexors cannot act for want of their normal support. This was well seen in the case of the ladler of metal, who was no longer able to employ his right hand as a fulcrum when using his long ladle.

I believe this condition is produced by injury to the nerve-twigs which pass through the extensors to come to the surface just below the external condyle. This spot is intensely tender to deep pressure, and there is generally a patch of tenderness to the point of a dragged pin, with or without a little diminution in cutaneous tactile sensibility, over a patch a few inches in length situated just below the external condyle. No other nerve-trunks are tender, and the muscular reactions do not shew any change.

Complete rest of the arm with blisters applied to the tender spot will generally cure the disease in six weeks. But it is liable to drag on if the arm is used, and may fall into a chronic intractable condition.

Other Forms of Occupation-Neurosis.—Tremor and pain affecting the arm, whatever their cause, will interfere with any occupation depending on the use of the upper extremity. This disability will be described as a professional neurosis by those who do not recognise the difference between a primary and a secondary form. Thus, there is scarcely a known occupation to which a "cramp" has not been attributed.

But disabilities corresponding apparently to primary writers' cramp have been described more particularly for the following occupations:—"Bricklayers' cramp" (Poore), in which the difficulty consisted in using the trowel; "sawyers' cramp" (Poore); "milkers' cramp" (Basedow), in which the grasp of the teat became impossible; the "waiters' hand" (Remak), produced by carrying glasses and plates; "barbers' cramp" (Oppenheim), in which an extension of the hand and fingers came on every time he attempted to shave his clients. But, as Oppenheim rightly points out, the more common professional disability in this trade is the fear of slipping and producing a cut; de Lépinay gives an excellent instance of this "phobia" (Régis). "Compositors' cramp" (Poore), in which the patient lost the power of holding his "stick" in the left hand,

and "skin-dressers' cramp," produced by the use of the scraping knife used in preparing the skins, have also been described.

In all occupations involving prolonged strain there is a liability to break down. Dr. Poore described a case of "sawyers' cramp" in a man who made packing-cases "by the piece"; in him was found definite tenderness of the suprascapular nerve and of the nerve-twigs (anterior thoracic) piercing the pectoralis major. The pectoralis major and the supraspinatus muscles were slightly wasted. Another interesting case (Poore) was that of a man whose work consisted in covering pickle-jars with bladder. Here it was the left hand which became impotent, and the muscles affected were the flexors of the finger which tightly grasped the top of the jar.

Among occupation-neuroses affecting the lower limbs the so-called "dancers' cramp" must be mentioned. At first sight it appears strange that an exercise so essentially rhythmical as dancing should be liable to functional derangement. It appears, however, that the stage-dancer only is affected, and of these such only as perform the "pas des pointes" on the tips of the toes, a position which necessitates prolonged contraction and great strain of the muscles of the calf to maintain the necessary extension of the foot.

Oppenheim describes a trumpet-player who was seized with a cramp in the orbicularis oris whenever he put the instrument to his lips, and a similar condition has been described in watch-makers, in whom cramp of the orbicularis palpebrarum can be produced by holding the watch-maker's magnifying glass in the eye.

Diagnosis.—It will be gathered from what has gone before that occupation-neuroses are, after all, merely symptoms of many conditions; and that, when we are confronted by them, our chief duty is to make out the cause of the breakdown, and to determine whether this break-down is, so to speak, accidental, or whether there are reasonable grounds for supposing that it is definitely due to the too frequent repetition of the particular act in which the occupation consists. The latter conclusion can never be justified until, by careful examination, all other causes of break-down have been excluded. Having excluded congenital defects, and those degenerative and other changes of the nerve-centres which are fairly well known, and to which special names are assigned, we arrive at our diagnosis of occupation-neurosis mainly by way of exclusion.

It has often been stated that the occupation-movement only is deranged. This is certainly untrue for all those forms of disability which arise secondarily to injury or other disease. In these conditions, which form a large proportion of the cases of "professional cramp" other movements such as playing golf, handing a plate, or applying the brake of a bicycle will evoke the same pain, tremor, or cramp.

In the primary forms, not associated with any other disease, movements differing so grossly from those of the patient's profession can be carried out with ease. But even in the primary form of writers' cramp, fine movements produced by the same muscles as those used professionally will not uncommonly shew some deviation from their normal accuracy.

The **prognosis** of craft-palsies is not uncommonly a matter of some difficulty. It necessarily hangs upon the diagnosis. If, as I have argued, an alleged occupation-neurosis is but a symptom of many different pathological conditions, the prognosis differs widely in different cases. We may form the opinion that the trouble is an early sign of senile degeneration, not really dependent upon the occupation, and not likely to be greatly influenced in its course by treatment or regimen; or we may recognise it as caused by an accidental condition, such as injury, neuritis, or rheumatism, which may be curable; or, again, we may recognise it as a genuine occupation-neurosis which, if indeed without influence on the expectation of life, demands nevertheless an instant remission of the injurious occupation.

If in a case of writers' cramp, for example (*a*) the writing is gravely affected; (*b*) there has been overwork with the pen; (*c*) there is no evidence of neuritis; and (*d*) other occupations not demanding fine movements of the writing muscles can be carried out without difficulty by the affected limb, a very grave prognosis must be given with regard to the recovery and maintenance of the normal power of penmanship.

Treatment, like prognosis, depends upon diagnosis. It is almost always necessary to prescribe *rest*, more or less absolute, from the occupation; but this advice must not be given without taking all the circumstances into consideration. If we come to the conclusion that rest is not likely to relieve the disease to any great extent, perhaps the best advice, and that which will best serve the interests of the patient, is to get him to ease his work if possible, but to permit him to go on with it. If, for example, the disorder be due to incipient senile tremor or ataxia in the upper limbs, it is clearly to the patient's interest that he should make full use of whatsoever power may remain to him, and he should be told to continue his work, and to make the best of it. When a patient is suffering from a disease which is usually progressive and is little influenced by treatment, we clearly do him a great disservice if we deprive him of the privilege of earning his living a day earlier than necessary. This is especially true in the case of an artisan. It is astonishing how long an ataxic patient will manage to "rub along," and earn, or partly earn, his living after the physician has made this diagnosis. We must not lose sight of the uncertainty of the rate of progress.

If, however, a young, delicate, and neurotic clerk, who has come out of the country into a city office, complains of a difficulty in writing, the best advice, if he be "without encumbrance," is to change his occupation instantly. The name given to the best-known occupation-neurosis, namely, "writers' cramp," is a name full of terror for the clerk whose sole means of livelihood is penmanship. He asks with trepidation, "Have I got writers' cramp?" by which he unconsciously means, "Is my difficulty incurable?" Often this question is not to be answered offhand; but in any case it is necessary to relieve the patient from his occupation for a time, and my general rule is to forbid him to write for three months. This injunction is a hard one, and in most cases a compromise has to be made. In large mercantile houses it is often possible to find a change of occupation for a valuable servant; and many of my patients have been sent on a voyage on the business of the firm, or have been employed as overlookers at the warehouses, or in some form of travelling; or they have had the services of a shorthand clerk. Such brief notes as a man, thus occupied, finds it necessary to take should be made with a soft pencil, for a pencil, upon which some pressure can be made, is easier to write with than a pen, and serves the unwilling hand very much as a walking-stick serves a man with a weak leg. It is much better for the patient to have employment of some kind, provided it does not entail prolonged writing. These patients are often worried and apprehensive about their condition, and if they be deprived of all employment they are very apt to brood over their troubles, and to become hypochondriacal.

Patients often think that their ills can be cured by gymnastics, such as the use of dumb-bells; such a proceeding cannot do good, and, if the muscles be overstrained, may do harm. I have known several instances in which an advertising gymnast has failed entirely to relieve patients suffering from writers' cramp. I always discountenance any kind of violent exercise with the arms and recommend any gentle exercise which does not involve any prolonged strain, and does not cause pain, stiffness, or uneasiness. There is no object to be attained by keeping the arm and hand in a sling (which I have frequently known to be recommended); there is no reason why the patient should be debarred from making ordinary use of the limb.

Pianists, to whom the abandonment of employment means a serious money loss, I allow to go on teaching; but I absolutely forbid any public performances which entails many hours of severe practice daily. All practising must be abandoned.

I have spoken of tenderness of the nerves as a common symptom. Sometimes the tenderness is primary, but more frequently it is secondary. The tenderness of the nerves is usually accompanied by some aching of the limb, and the patients often complain that they find great difficulty in placing it in a painless position; that they "cannot make it comfortable when in bed." This condition is nearly always benefited by *counter-irritation* over the trunks of those nerves which are tender, and at points where they are most superficial. Counter-irritation may be carried out in many ways. If the pain be severe it is a good plan to apply blistering fluid or a blistering plaster; but if less severe I recommend the application of strips of capsicum plaster. This should be made with india-rubber and spread upon thin perforated linen. It should interfere as little as possible with the free movement of the limb; and, if it be mixed with the india-rubber medium, the patient can wash as usual without great risk of removing it. The plaster may be worn until it comes off, and, if necessary, it may be renewed once or twice.

Massage or Electricity.—Neither massage nor electricity must be used so long as the nerves are tender, for in such circumstances they are positively harmful. It is very important that the masseur should be gentle in the exercise of his craft; for if he use too much force he is likely to do harm rather than good. It is also of great importance that special attention should be given to the intrinsic muscles of the hand, such as the interossei and lumbricales, and the muscles of the thenar and hypothenar eminences. The whole limb and hand should be rubbed; and if there be any suspicion of rheumatic thickening in and around the joints, these should be submitted to passive movements. A limb thus treated will, in favourable cases, get plump and strong, although to begin with it may have been in a very different condition.

I have never known much benefit, if any, result from faradising the muscles. *The galvanic current* alone is to be used, in combination with rhythmical movements of the fatigued muscles. A current of moderate intensity is employed, of about 5 milliamperes, which produces a slight contraction of the muscles without causing pain or burning. The positive rheophore, which is of large area, should be fastened to the nape of the neck with a bandage. The negative rheophore, soaked in hot water, is then placed seriatim over the affected muscles and (where possible) the nerves supplying them. While the rheophore is thus placed, and the current still passing, the patient should be made to exercise these muscles thoroughly and rhythmically. The effect of the current, when thus used, is most refreshing, and, as a rule, removes any sense of fatigue. Finally, the whole limb should be sponged, and is left reddened and glowing. Galvanism thus used stimulates the nutrition of the part, and in some cases the result is most satisfactory.

These methods are useful in cases of neuritis, in chronic fatigue of the muscles, or where the muscles have been deranged by local accidental causes. But in saying this it is necessary to repeat with emphasis that neither of these methods is of any use so long as the nerve-trunks, nerve-twigs, or muscles are tender.

These methods of treatment, when successful, are to be looked upon as curative; but it must be remembered that a man who suffers from an occupation-neurosis must be regarded as disposed to these troubles; and it is generally necessary to warn him that, unless he is able to "ease off" his work, he will be liable to a recurrence. I have already alluded to cases in which a man has gradually developed more than one professional cramp.

Mechanical methods of overcoming occupation-neuroses are temporary subterfuges. A man who cannot write with a pen often has no difficulty in doing so with a piece of chalk on a blackboard or other large surface. He finds some relief from a penholder which offers a big grasp to the writing hand, and many patterns of penholders have been made for this object. Big cork penholders, globular penholders, or those which slip on the finger and are retained without the apposition of the thumb, have also been tried, and in many cases they afford relief, at any rate for a time. Yet this relief is never complete; for it is impossible to write with any great rapidity with such an apparatus. The best of them dates from Duchenne, and resembles a small bun made of wood, to which is fixed an ordinary penholder carried by an adjustable clamp. The wooden mass is held in the palm of the hand, and the fingers do not touch the penholder. By this means a man can be brought to sign his name or to write short letters; but he cannot write freely or for any prolonged period.

For writing a few lines there is no objection to the use of the left hand; but in aggravated cases of writers' cramp the use of the left hand for "quill-driving" is not without danger, not only to the left hand, but to the right hand also. It is probable that when a man has learnt to write with his right hand, the normal method of writing with the left hand would be to write backwards, and to produce what is known as mirror-writing, which can only be readily deciphered when it is held before a mirror. Mirror-writing is of no commercial value; and persons who have learnt to write with the left hand, and who produce normal writing flowing from left to right, find the act laborious, and the rapidity of production never very great. The labour of writing in the normal fashion with the left hand is greater than it is with the right hand, and persons disposed to occupation-neuroses are very apt to break down with the left hand as they have done with the right. Further, it must be remembered that to learn to write with the left hand is a labour and a worry, and is often accompanied by associated movement of other muscles, notably of the muscles of the right hand or forearm. I have seen a case of writers' cramp in which the patient had learnt to write with the left hand, yet he was quite unable to do so without performing unconscious movements with the fingers of the right hand, which were never still while he was writing with his left. It seems probable that in writing with the left hand some fatigue is occasionally produced in the right limb. There is no harm in learning to make use of the left hand for minor acts of writing; but there must always be a risk of its "giving out," just as the right hand had previously done.

The only apparatus which is of undoubted and permanent use to the sufferer from "writers' cramp" is a typewriter. I have not so far seen a case of primary writers' cramp in which the patient was not able to use a typewriter with ease and comfort. In fact it is often a useful means of differentiating between the primary and secondary forms. For if the disability is due to brachial neuritis, arthritis, or some similar cause, the use of the typewriter will evoke as much pain or tremor as writing. A busy man can now obtain a machine so small that he can carry it about without difficulty. But, unfortunately, no form of machine will help the bank clerk to enter up his books. For tradesmen and all those who can determine the form in which their accounts shall be kept, I recommend loose-leaf books. Each leaf can be placed on the typewriter, and no figures need be entered by hand.

With regard to the general health little need be said. Whatever is found amiss should be corrected, if possible. If the patient be exhausted by overwork, he must seek complete rest and change; and we must treat such conditions as gout or rheumatism on ordinary dietetic and medical lines. Alcohol is a danger to some of these patients, and I have seen one definitely cured by abandoning its use. A man finds, perhaps, that a stimulant enables him to get through a certain amount of work; but if he indulge this manner of overcoming his difficulty, he soon falls into the state of the chronic drunkard who takes a glass of spirits to "steady the hand." The temporary relief leaves him worse than before, and the need of the stimulant steadily increases. Such patients should be dissuaded in the strongest way from a habit so dangerous.

Re-education.—Some patients can certainly be taught to write from the elbow or wrist. If given a rounded nib and a large penholder, and trained never to move the fingers, a few sufferers from writers' cramp can then write sufficiently to sign cheques or write a short letter. Macé de Lépinay gives an ingenious method of re-educating the patient to write with the hand upside down (supine). Seat the patient with his right side against the table, the left edge of the seat being turned towards the left. The forearm and hand rest supine upon the table, and the pen with a large handle is held between the pulps of the index and middle fingers by means of the thumb. With practice some patients are said to be able to write fairly easily by this method, which effectually avoids all possibility of moving the fingers whilst writing.

In a few cases in which fear plays a part, suggestion is useful, as in the case on p. 676. But in primary writers' cramp I have not seen any success from any form of suggestive or hypnotic treatment.

If the disability is due to a tic, careful exercises in control must be carried out for a considerable period under medical supervision. These are described on p. 635.

HENRY HEAD.

REFERENCES

1. DUCHENNE. *De l'électrisation localisée*, 3rd ed., pp. 1021 *et seq.*—2. GOWERS, Sir W. In his *Textbook on Diseases of the Brain and Spinal Cord*, and in most of the treatises on nervous diseases, a section is devoted to Occupation-Neuroses under various synonyms.—3. MACÉ de LÉPINAY. *Nouv. icon. de la Salpêtrière*, Paris, 1909, xxii. 65, 189, 288.—4. OPPENHEIM. *Lehrbuch d. Nervenkrankheiten*, 1908, 5te Aufl., Bd. ii. S. 1457.—5. POORE, G. V. *Med.-Chir. Trans.*, Lond., 1878, lxi. 111.—6. *Idem.* *Textbook of Electricity*, 1876, p. 217.—7. *Idem.* *Nervous Affections of the Hand*, London, 1897.

H. H.

HYSTERIA

By J. A. Ormerod, M.D., F.R.C.P.

Introductory.—The name "hysteria" has the sanction of widespread and long-established usage, and we adopt it chiefly on this account. The objections to it are obvious; not only that it has become etymologically meaningless, but also that to many minds it has the disagreeable connotation of a certain moral feebleness in the patient, and of unreality in the symptoms. Other names have been proposed, but they also are not free from objection. "Neuro-mimesis" lays too much stress on the resemblances, often superficial, between the symptoms of hysteria and of other diseases. To substitute the word "functional" for "hysterical," as is now often done, avoids, indeed, the unpleasant connotations, but introduces an arbitrary limitation of meaning; for it can be said of many diseases other than hysteria—epilepsy, for example—that they are functional in so far as we are ignorant of their organic basis; so that, properly speaking, whilst all hysteria is functional, all functional disease is not hysterical.

More important than the mere name is the definition of it. On the one hand, hysteria is no mere limbo wherein to thrust all ill-understood nervous cases; on the other, our definition of it is unsatisfactory, so long as our knowledge of its intimate nature is incomplete. Short of this, most physicians will admit that there exists indeed a natural group of cases to which this or some similar name must be applied; but that its limits are somewhat vague, and that cases constantly occur of which the classification must for the present be doubtful. Among French physicians, to whose labours in this field we owe so much, there has been a tendency to include more and more under the title of hysteria; whilst certain English authors incline in the contrary direction. Thus, Dr. T. Buzzard (8), writing of the diagnosis between hysteria and organic disease, says that many symptoms considered characteristic of hysteria will eventually be relegated to disseminated sclerosis. Dr. Bastian, again, would subdivide the large class of palsies which are indifferently called functional or hysterical, and limit the term "hysterical" to those only which have such well-recognised characters as fits and emotional manifestations. There is, indeed, great difficulty in pronouncing upon the true nature of "functional" cases which do not present a full quota of hysterical symptoms; for we are bereft of the sure guide of morbid anatomy, and, unless we would adopt some arbitrary definition, we must be content with the resemblances and affiliations which they present to better-defined cases. A similar proposal is made by Dr. Head, who, with regard to cutaneous sensory affections, would limit the term "hysterical" to those of a "psychical" type. This would relieve us of much difficulty in the definition of hysteria, but would introduce clinical limitations of this name which, for the purposes of the present article, we can hardly consent to observe.

Nevertheless, let us endeavour to make some general statements as to the characters of hysteria. We look both for the constant traits of an hysterical diathesis and to the very variable manifestations arising from it, and judge partly of the crop from the soil and partly of the soil from the crop. The diathesis is readily and generally recognised when the patient is a woman, adolescent or in early womanhood, emotional and impressionable, and inclined to such minor neuroses as palpitations, globus, headaches, and other pains. But since hysteria may exist in the opposite sex, and in patients of a different type, we must welcome the attempt—due chiefly to the physicians of the Salpêtrière school—to find more precisely bodily signs of the hysterical state. The signs or "stigmata" described by them are mainly three: to wit (i.) the presence of anaesthesia; (ii.) concentric limitation of the fields of vision; (iii.) the presence of hystero-genetic zones. Of these I shall speak in detail later. Important as these signs are, there is often some difficulty in utilising them practically; for, first, they do not exist in every case; secondly, for the discovery of anaesthesia and of the field contraction a minute and skilled examination may be necessary; lastly, the hystero-genetic zones do not, at least in this country, commonly reveal themselves in a convincing form, but are usually reduced to mere areas of tenderness, the diagnostic meaning of which is not very clear. The hysterical diathesis, therefore, may be difficult to diagnose; and both for this reason, and for the still more cogent one that an hysterical patient may suffer from organic disease as well, we must next ask what are the characters of the various manifestations of hysteria. Much may be said of this in detail; not much, perhaps, that is at once distinctive and of universal application, but we may note the following general points:—

(*a*) Many symptoms are paroxysmal, leaving the patient entirely free in the intervals; of these the hysterical "fit" is the most notable. Others, though not exactly paroxysmal, are prone to appear and disappear with extreme brusqueness. In particular, sudden and complete disappearance of a grave paralysis suggests hysteria.

(*b*) Even when obstinate and of long standing they are susceptible, in the long run, of complete (and possibly sudden) resolution.

(*c*) They are far more dominated by mental and normal influences than are the symptoms of other, and particularly of organic, nervous diseases. Or they may seem to possess a sort of spontaneity, arising and departing without assignable cause. Or they may be subjected to some sort of artificial control (such as "ovarian" pressure) which would be impossible in organic disease. Sometimes the dependence of an obscure symptom upon some obviously hysterical state, as when a paralysis follows an hysterical fit, may reveal its true nature.

(*d*) Among individual symptoms, however characteristic, there is no regular train of sequence from which we can construct a typical course for the disease as a whole. Indeed there is often incongruity and want of proportion among the symptoms themselves, as when well-marked hemianaesthesia exists with little or no hemiplegia, or a severe paraplegia without sphincter paralysis, or when the symptoms seem inconsistent with the patient's general state.

(*e*) Lastly, there are no known organic lesions to which we can assign the symptoms. If such be found at a necropsy, it may be taken that the diagnosis of hysteria was so far wrong. This is not, indeed, as might be imagined, a wholly unpractical criterion during life; for it is on this ground that we hold certain symptoms known to be indicative of structural disease, such as optic neuritis and atrophy, or electrical reaction of degeneration, to be inconsistent with unmixed hysteria; and we may fairly extend this reasoning and say that the more closely any set of symptoms coincides with such as would flow from a known organic disease, the less likely are they to be hysterical. We do not, however, affirm the contrary proposition, namely, that symptoms are hysterical because they resemble no known organic disease; this is an assumption which often leads to error.

ETIOLOGY.—As to *sex*, it is hardly necessary to mention the greater prevalency of hysteria in women, seeing that it was long thought to appertain to the female sex exclusively: it is now, however, often recognised in boys, and not very rarely in men. Briquet's figures give 11 male to 204 female hysterics; and later French writers give a much larger proportion of males. As to *age*, the outbreak is commonest between the years of fifteen and twenty; but it is common enough in childhood (before ten), and may even take place in the very young. No *race* is exempt from it, and in the Middle Ages, as demonological histories shew, it was as common and as severe as it is now. It is, therefore, no special appanage of modern civilisation, nor yet, we may add, of the upper classes of society Its great *frequency* may be gathered from the statistics of Briquet, who found that of all his hospital patients about one quarter were hysterical; and, from the statement of Sydenham, that hysteric disorders constitute one moiety of chronic distempers (and one-sixth of all the diseases to which mankind is liable).

Heredity.—In the formation of the hysterical diathesis, the most potent, and some authors say the only essential factor is an hereditary nervous taint; and the disease may either be inherited directly, as when a hysterical mother bears children who become hysterical; or the heredity may be more general, a neurotic taint, such as insanity, epilepsy, alcoholism, or some other neurosis, being manifested in the ancestry or collaterals.

Other *constitutional traits*, such as tuberculosis (Grasset), the arthritic diathesis (Laycock), plumbism, syphilis, have been noted in connexion with hysteria, but probably have quite a subordinate place. There is no direct or special connexion between hysteria and disease of the sexual organs.

Among the *proximate causes* of the disease we must first mention depressing emotion, either of an acute or cumulative kind. Among the first kind rank terrors, as from the perils of fire, water, or violence; the unwonted sights of death, or accident, or convulsive fits; sudden griefs, as from loss of kindred; severe revulsions of feeling, as from seduction, desertion, or disappointment in love: on such shocks the hysterical symptoms may follow at once, or after an interval of variable length, during which it may be said the patient has "never been herself." Of the second kind are the trials of poverty; of family and business anxiety; ill-treatment by relatives or employers; unhappy marriages; the ennui and disappointment of a purposeless or misdirected life; courses of unhealthy excitement, spiritualism, or religionism. For a woman predisposed to hysteria the nursing of a sick relative is a particularly dangerous strain. Another set of causes appears to be more directly material—prolonged ill-health of all kinds, and notably anaemia; amenorrhoea; pelvic troubles necessitating elaborate local treatment; tedious convalescence from enteric or other acute disease; chronic intoxication with agents that specially affect the nervous system (the possibility of secret alcoholism is to be weighed when hysteria makes its appearance in middle-aged women); chronic organic nervous diseases, whereof hysteria may be a most puzzling complication. Physical injury is likewise a well-recognised cause of hysteria, though it may be hard in such cases to apportion the bodily and the mental factors (see p. 765). Lastly, hysteria may be contagious by imitation, and thus take the form of an epidemic. This usually takes place in small and predisposed societies, such as schools, nunneries, remote and superstitious villages; and the phenomena are such as appeal strikingly to the imagination, such as religious ecstasies, strange forms of fits, saltatory spasms, and laryngeal noises in the form of bellowings, barkings, or mewings.

Hypotheses of Hysteria.—What is the nature of hysteria? What lies at the root of all its manifold and strange manifestations? As yet no thoroughly satisfactory answer can be given. The ancient supposition that the symptoms were due to wanderings of the uterus about the body, absurd though it appears, was at least capable of proof or disproof. Anatomy disproved it; and by degrees it has become evident that the primary seat of the disease is to be sought in the nervous system. But no structural lesion has yet been found there, neither does it at present appear probable that it will be. In the absence of disease demonstrable to the eye, there remain certain other suppositions: one, a vasomotor hypothesis, namely that the abnormalities of nervous function are due to spasm and relaxation of the arteries supplying the nervous system, is mainly calculated to explain palsies of definite localisation, and capable of sudden recovery; but seems inadequate to explain palsies which may last for months or years, or indeed many other symptoms of the disease. Secondly, we may suppose the disease to be due to defective nutrition of the nerve-elements, whereby, though their structural aspect be unchanged, their chemical composition is so altered as to produce irregular or defective action.

And this seems probable enough; but in the absence of detailed support or application, it is at present no more than a hypothesis.

Looking, then, to the difficulties of a purely anatomical explanation, it is not surprising that recent writers should have changed the ground, and should approach the question from the psychological side. Briquet maintains that hysteria is essentially concerned with the emotions; it is a disorder of that part of the nervous system which subserves the emotional nature. He urges with much force the part that emotions play in the causation of the disease, and draws a parallel, perhaps less convincing, between the effects of violent emotion in the healthy, and the phenomena of an hysterical fit. But it is difficult in this way to explain more permanent hysterical symptoms—anaesthesias, palsies, and the like; neither is it by any means certain that all hysterical patients are of an excitable temperament. To explain such palsies mental processes other than those of emotion have been invoked. Thus, Sir Russell Reynolds has said that certain paralyses are dependent on idea; the patient, if possessed with the idea that he cannot move, appears physically incapable of moving. Charcot, following this up, demonstrated analogies between certain hysterical palsies and those producible by hypnotic suggestion; and, applying this particularly to paralysis in trauma, he says that just as an injury may produce in any one a state of temporary numbness and uselessness of an injured limb (local shock), so in the hysterical this state may form the basis, by suggestion, of a more declared and permanent motor and sensory paralysis. It is practically a generalisation from such observations to say, with Babinski, that all the phenomena of hysteria are due to suggestion, or with Möbius, that hysteria is wholly "ideo-genic"; that ideas or representations which in the healthy are under the control of conscious reflection and volition, in the hysterical produce bodily actions or incapacities by an automatic process which lies beyond the control or even the knowledge of the patient. Thus hysteria would be a disorder of cerebral function, and might be called a sort of insanity. But to classify hysteric patients with the insane would be an obvious clinical error, as even the strongest advocates of its psychical origin would admit. Thus, Dr. Donkin says: "In the majority of cases intellectual disorder is not conspicuous, and mental abnormality is mainly evidenced by exaggerated impressionability or tumultuous emotion"; and again, "The mental condition of many hysterics is only paroxysmally abnormal in times of excitement or stress," which indeed might be said of other than hysterical subjects. To put the psychical hypothesis of hysteria on a more definite foundation, and to specify the particular forms of mental disability which characterise the disease, is the object of Janet's interesting speculations. Starting from the facts of hysterical anaesthesia, this author remarks that to some instances of this a certain unreality attaches; that is, the patient has not indeed the consciousness of sensation, but yet all the elements of a sensation are present, except the personal perception of it. Similarly, with hysterical amnesia (on the frequency of which he is inclined to insist) memories can be shewn to exist, but the patient cannot personally perceive or utilise them. The defect is not in the apparatus of sense or memory, but is an inability so to appropriate the data supplied thereby as to make them part of the personal consciousness. It results from this defective power of psychological synthesis that, whilst a healthy mind can grasp and weld into one state of consciousness the numerous impressions which simultaneously pour in upon it, the hysterical cannot take in so much at a time; and there arises a "limitation of the field of consciousness" comparable to the well-known limitation of the field of vision. Burdened by such deficiency the patient ceases to attend to the impressions which concern her least till she becomes, in the sphere of sensation, anaesthetic; or, in the sphere of memory, amnesic. And, since strong momentary impressions are thus less controlled by other present impressions or memories of the past, she becomes a prey to momentary impulse; and presents an apparent mobility and inconsistency of character, a susceptibility to external suggestion, and a seeming feebleness of will. Further, the habitual relegation of impressions to a lower sphere not only impairs and limits the higher personal consciousness, but tends to foster at its expense a "secondary" subconscious mental state. The elements of such a state exist in all of us; sometimes it can be brought into prominence by such processes as reveries, hypnotism, or somnambulism. In somnambulism, which Janet considers as closely allied to hysteria, the secondary consciousness takes control of the whole individual, to the exclusion of his "primary" or personal consciousness. The patient thus lives two lives in alternation, the normal and the somnambulistic; and though the events of the latter are forgotten in the normal state, their threads are taken up again whenever the somnambulistic state recurs. Hysterical fits, according to Janet, are virtually instances of somnambulism, parts of a drama played by the secondary consciousness, and reiterated at intervals independently of the patient's personality or of her actual surroundings. Further, these two mental states not only alternate, but coexist; hence, when the primary consciousness is feeble, and the secondary correspondingly obtrusive, a field is opened in the latter for the growth of fixed ideas, which, independently of the will and even without the patient's knowledge, may produce paralysis and other of the more permanent hysterical phenomena.

This, so far as we know, is the most precise and elaborately reasoned hypothesis of hysteria from the psychological standpoint. It remains to be seen if it can be successfully pursued into further details. Why, for instance, to take anaesthesia, should the patient be specially prone to lose perception of sensations coming from the left half of the body? Why should analgesia be common, seeing that the sense of pain is a most obtrusive and most useful form of sensation? How shall we explain the regular localisation of tender points? Can we reasonably regard a common hysterical fit, whose manifestations are very similar in many individuals, as representing the thread of the particular patient's subconsciousness? It may be so in a special stage of the hystero-epileptic fit; but such cases are surely exceptional. These are minor points perhaps; but as to the "subconsciousness" itself, which appears to be essential to the hypothesis of paralysis from idea, is it not premature to base a hypothesis upon a condition of which we have so very little knowledge? Again, there are hysterical phenomena—visceral, vasomotor, and nutritional—to which it is extremely difficult to apply any psychical explanation at all; for they appear to lie outside the sphere of those higher cerebral centres which are regarded as the organ of mind. Lastly, granting that it may be necessary, in consequence of our present state of ignorance of the workings of the highest cerebral centres, to speak of these in transcendental terms, yet psychology alone cannot supply an explanation of disease, which, to satisfy the physician, must be physical.

Breuer and Freud have a special hypothesis of hysteria. They hold the very prevalent view that this disease and many allied states are mental disorders, and the symptoms of them indicative of what is going on in the "secondary" or subconscious mental life of the individual. But hysteria, they say, has this special characteristic, that it originates in some shock or disagreeable feeling experienced in connexion with the sexual emotions, usually something that has happened in childhood. The patient suppresses this occurrence, not merely keeping silence about it, but also banishing it from conscious remembrance, either by strenuous effort of the will, or by substituting for it some less disagreeable idea. Still the original memory remains in the lower consciousness as a permanent source of disturbance ("foreign body"), and reigning there, unmitigated by the wear and tear of the daily conscious life, asserts itself from time to time by the production of fits and other hysterical phenomena. The events of the secondary consciousness can be recalled during hypnotism, or by persistent and skilful questioning, and if the patient be then made to give to her interlocutor a full account of the original occurrence, she obtains mental relief, and the hysterical symptom disappears for good. This curiously revives the old conception of the sexual origin of hysteria, and of the efficacy of the confessional; but in this novel confessional the doctor is the priest, listening to subjects of extremest privacy, while the penitent does not even know what he is about to confess, and his confession (a sceptic might add) may be purely the result of imagination or of suggestion received from without. The authors give a most interesting and suggestive exposition of their hypothesis; but we fail to see the proof that hysteria is always due to some past sexual shock, and we suspect that the treatment by inquisition and confession might sometimes do more harm than good.

SYMPTOMS.—The symptoms of hysteria may be usefully divided into those which are paroxysmal and those which are not. Many of the "inter-paroxysmal" symptoms, however, are prone to fluctuations, which shew that the distinction is not an absolute one.

A. **Hysterical Paroxysms.**—1. *Mild Form of Hysterical Fit.*—Paroxysmal symptoms are best exemplified by hysterical "fits." These may be represented by three types. The mildest form, popularly known as a fit of hysterics, is much as follows:—Either suddenly, or after a preliminary stage of malaise and disquietude, the patient is attacked by disagreeable sensations in the abdomen or epigastrium, by palpitation or "globus"—that is, the sensation of a lump rising to the throat and causing strangulation, or perhaps by dizziness and faintness; as her sufferings intensify, she sinks on a chair, pants for breath, tosses her hands in agitation and distress, till the attack ends, it may be, with sobs and weeping, or with meaningless laughter; or perhaps with eructation of wind. The fit is often followed by a copious evacuation of pale watery urine. Even these mild attacks may occur in a fragmentary form; thus there may be palpitation and globus without an overt fit, or the attack of agitation may occur without the aura. In such fits there is very evident loss of self-control, but no loss of consciousness; for the patient is not oblivious of her surroundings, may express her feelings by words or gesture, and afterwards remember all that has happened. But sometimes the fit takes the form of a "swoon," in which the patient simply sinks to the ground in apparent unconsciousness.

2. *Hysteroid Fits.*—Such swoons form the transition to the second class of fits, for which the name "hysteroid" has been proposed. These exhibit two additional phenomena, namely, loss of consciousness and convulsions; though either of these factors may vary much in severity. There may or may not be an "aura." A complete "aura hysterica" consists of the following stages:—First, pain in the abdomen, either in the iliac region or epigastrium; secondly, globus with attendant feelings of suffocation, of inability to swallow, and cardiac palpitations; thirdly, according to Charcot, cephalic sensations, such as dizziness, tinnitus aurium, dimness of vision, or pain on one side of the head, as if from a blow. Sometimes sensations may begin in the limbs, starting from the feet and passing upwards; or there may be a motor aura, such as twitchings of eyelids, face, or limbs, usually bilateral, but in some rare instances unilateral, so as to suggest Jacksonian epilepsy. Thus there may be considerable variety as regards an aura; though doubtless abdominal sensations and globus are the most characteristic. Some patients do not have an aura.

In the fit the patient falls, apparently unconscious; but her fall is not so heedless or sudden as to result in serious injury. She may utter a cry of pain or terror, but not the inarticulate laryngeal noise that constitutes the epileptic cry. The face is not livid or contorted, nor the eyes staring; commonly the lids are closed, tremulous, and resist efforts to raise them; the corneal and pupillary reflexes are present. She may lie limp and inert, but more commonly she is rigid, the hands clenched, the limbs stiff and extended, the back arched. Such rigidity may last much longer than the tonic spasm of epilepsy. Convulsive movements may now set in, but these differ from epileptic convulsions. For whereas the clonic stage of epilepsy exhibits rapid alternation of muscular spasm and relaxation, producing movements of the limbs, head, and jaws which are mechanical and aimless, and rarely displace the patient's body as a whole, the hysterical fit consists not so much of muscular spasm as of co-ordinated purposive acts. These vary in character; but frequently a scene of violence is enacted, the patient dashes herself about, tears at her hair and clothes, struggles when held, bites, and screams. She does not bite her tongue severely, nor foam at the mouth, nor pass her excreta beneath her. The convulsions of a single epileptic fit rarely last more than a few minutes, whereas an hysterical fit may last even for hours. Moreover, it is peculiarly apt to relapse, so that a status hystericus is commoner than a status epilepticus. Between these two last-mentioned conditions there is, according to Charcot, the following difference: that in the status epilepticus the bodily temperature rises, and the general condition becomes grave from pulmonary embarrassment and the like; in hysteria this is not the case. Again, recovery from an hysteroid fit is more prompt and complete than in epilepsy; there is not the prolonged stupor, nor even the dazed and confused mental state. Indeed, during the fit itself it would seem that consciousness is less deeply affected; for whereas an epileptic is wholly out of relation with his surroundings, cannot be roused by any stimulus, and afterwards does not remember anything that happened, the hysterical patient rarely suffers injury, can be brought round by appropriate measures, and can sometimes recall the circumstances of the fit. Mental excitement, of whatever kind, is the commonest determining cause of hysteroid fits: and more commonly perhaps than in epilepsy does the fit follow immediately upon the action of such a cause. The night-time and the early morning, times of predilection for epilepsy, are not the common hours of hysteroid attacks.

Thus there are many points of difference between hysteroid and epileptic fits; nevertheless difficulties of diagnosis often arise: first, because the physician has often to depend on the account of friends for the characters of a fit; secondly, because both kinds of fit may occur in incomplete and aberrant forms; thirdly, because there is a form of hysterical fit in which the features of hysteria and epilepsy appear to be combined (*vide* also p. 509).

3. *Hystero-epileptic Fit, or* "*Grande Hystérie.*"—This, the third and severest type of hysterical fit, is elaborately described, by Charcot and Richer, under the title of "hystero-epilepsy," or "grande hystérie," as follows. The attack exhibits four periods:—

The first (epileptoid) period closely simulates a true epileptic attack, and begins with a phase of tonic muscular spasm, upturned eyes, distorted features, pallor followed by congestion of the face, and interference with respiration; upon these follow clonic spasms, and then a phase of muscular relaxation with noisy or stertorous breathing. There may be foaming at the mouth; it is apparently doubtful if tongue-biting and involuntary escape of urine ever occur. The distinctions from epilepsy are so far very slight; the neck is said to swell more, the tonic spasm is apt to take the form of opisthotonos, and may be preceded by large, slow movements of the limbs; but the main distinction is said to be that the attack can be stopped by faradisation, or by pressure upon the inguinal region.

After a short interval follows the second period, that of contortions or "clownism." The body is thrown into various strange positions—a favourite one being the *arc de cercle*, in which the back and neck are curved backwards till the patient rests on her head and the soles of her feet; or that of "crucifixion," namely, body and legs straight, arms and hands outstretched at right angles to the trunk. After such posturing, some violent and extraordinary movement of the trunk or limbs begins, and may be repeated many times; or there may be simply struggling, violence, and screaming.

In the third period (that of "attitudes passionelles") the movements are no longer meaningless, but dominated by some idea. The patient enacts some drama of her past life which has taken lively hold on her imagination—incidents of terror, pleasure, or love, not infrequently those which led up to the first fit. She does not recognise her real surroundings, and reacts to her hallucinations only. This period varies much according to the natural powers and drift of the patient's imagination.

The fourth period, that of delirium, marks the decline of the paroxysm. The patient has now more or less regained sense of external objects, but may mistake them for the creatures of her imagination; and about these she rambles disconnectedly, though she no longer acts her fancies. While in this condition she may suddenly become the prey of visual hallucinations, of rats, snakes, beetles, and the like; this has been called "zoopsia." Lastly, as the fit passes off it may leave painful contractions of the limbs, or muscular startings, or hiccups, or borborygmi; and these may be followed by the well-known flow of watery urine, or, more rarely, by secretion of saliva, or of vulvo-vaginal mucus.

The epileptoid period lasts from one to three minutes; the whole cycle may occupy from a quarter to half an hour, but this varies, particularly when attacks are subintrant. The patient remembers nothing of the epileptoid period, if roused from it; but she may retain more or less memory of her hallucinations.

Complete and characteristic fits of this kind are so extremely rare in this country that it is difficult to criticise the French descriptions. Perhaps we see enough to justify them in a general sort of way. Richer himself says that the events of each period may be emphasised, modified, or suppressed in different cases; so that the general aspect and tenor of the paroxysm may be variable. He maintains, nevertheless, that hystero-epilepsy is the full-blown manifestation of the hysterical paroxysm, of which the other forms contain only the germs. But if this be so, the abortive forms are far the commonest. Perhaps the most important point is the true meaning of the epileptoid period. Many physicians would hold that it is a manifestation of true epilepsy, and that the succeeding phenomena should rank as post-epileptic hysteria (*vide* p. 503). To this the French authors object that the epileptoid stage may be preceded by an hysterical aura, that it may be cut short by artificial means, and that the recurrence of hystero-epilepsy does not entail the mental degeneration which accompanies chronic epilepsy. Consequently, while recognising that epilepsy may complicate hysteria, and hysteria epilepsy, in the alternation of distinct and separate fits, they maintain that hystero-epilepsy is hysteria pure and simple.

4. *Incomplete Attacks.*—I have said that there may be incomplete hysterical attacks in which the aura, or part of it, occurs alone. The same thing happens in epilepsy. Paroxysmal vertigo in hysterical patients has been called hysterical *petit mal.* But *petit mal* proper, that is, lapse of consciousness so transitory as scarcely to interrupt the patient in what he is doing, and only manifested to the bystanders by change of colour, strangeness of look, and so forth, does not occur in hysteria. Epileptic automatism suggests analogy to the dramatic stage of the hystero-epileptic fit. The contrasts, however, are that in epilepsy automatic actions usually follow slight fits, whilst the corresponding hysterical condition forms part of a severe fit. The hysterical performances, at least in their most perfect form, are dictated by some delusion or emotional reminiscence; those of epilepsy usually consist of the trivial acts of daily life; or else, which is happily rare, appear to be the outcome of some blind and unreasoning fury. The epileptic never remembers his acts or feelings afterwards; the hysterical patient sometimes does.

5. *Other Varieties.*—Other kinds of paroxysmal attacks may occur in hysteria, though they are comparatively rare. Sometimes the relation of these to the fits we have described may be made evident; either by the occurrence of an hysterical aura, or by the way in which the attack can be induced or controlled, or when it can be construed as representing some particular stage of hystero-epilepsy. Thus, patients may have paroxysmal attacks of delirium, or of violence suggesting demoniacal possession; of prolonged sleep, lethargy, or apparent coma; of catalepsy, ecstasy, somnambulism, or double consciousness. Or motor phenomena of unusual order may occur without affection of consciousness; thus, there may be spasms limited to the ocular movements, to the movements of the head and neck, and so forth; or abnormal visceral and reflex movements, such as paroxysmal hurry of the heart, or of respiration,—sneezings, yawnings, or hiccups; or sometimes a series of co-ordinated gestures and movements constitute a regular terpsichorean display; or there may be paroxysmal pains, simulating angina pectoris, tic douloureux, and so forth. With some of these, however, it will be more convenient to deal later.

B. **Inter-paroxysmal Symptoms.**—We pass now to the permanent symptoms of hysteria. Since some of these are generally to be discovered in the intervals of the fits, they may be called inter-paroxysmal; yet we must remember that hysteria is frequently manifested by these alone, without any fits. As they are varied, and have no fixed order of appearance, we can do little more than enumerate and describe them, endeavouring at the same time to follow a sort of rough physiological order.

Sensory Phenomena.—1. Anaesthesia.—Among sensory phenomena anaesthesia is extremely common, so much so that anaesthesia of the skin ranks among the "stigmata" of hysteria, and its persistence or disappearance has been used to measure the persistence or the wane of the disease. Doubtless there are hysterical patients who have no anaesthesia; still this cannot be predicated of them without careful examination, for it is a curious fact that such a patient may be as unconscious of his sensory defect as a normal person is of the blind spot on his retina. In organic disease the reverse is usually the case, and there are often subjective feelings of numbness, deadness, and the like, where loss of sensation cannot be demonstrated objectively. In mild cases of hysterical anaesthesia the skin only is affected; all forms of cutaneous sensation —namely, for touch, pain, or temperature—may be dulled or abolished together; there may, however, be "dissociation," one form of sensation being much more affected than the others, and the peculiar dissociation characteristic of syringomyelia, in which there is early and marked thermal anaesthesia, has sometimes, though not often, been noted in hysteria. To loss of cutaneous sensation may be added anaesthesia of the deeper parts. Sense of position (so-called muscular sense) may be abolished, so that the patient, with eyes closed, cannot tell the position of the affected limb, nor copy its position with the corresponding sound limb; presumably because the cutaneous anaesthesia has spread to the muscles, fasciae, and joints. Similarly the deep structures may be analgesic, so that muscles may be pinched or faradised, or the bones jarred, without pain. The mucous membranes, where these can be tested, may also be found anaesthetic; and some further symptoms, such as anorexia, or retention of urine, may possibly be adduced as evidence of anaesthesia in the visceral mucous membranes. Certain modes of distribution of anaesthesia are highly suggestive of an hysterical basis. A well-marked definite hemianaesthesia of face, trunk, and limbs, particularly if it involve the special senses, is commoner in hysteria than in organic disease; and the left side is more often attacked than the right. Secondly, when confined to the limbs, the anaesthesia is often distributed by segments. Thus, in the upper limb it may involve the fingers only; or the hand to just above the wrist; or the forearm to just above the bend of the elbow; or the upper arm to the limits of the scapula: in the lower limb there may be a similar distribution. Such a segmentary anaesthesia could not be produced by lesion of either the cord, nerve-roots, or nerve-trunks. When unilateral, it is indeed comparable with the anaesthesia ascribed to some lesions of the cortex of the Rolandic area; but this is usually of slight intensity, the hysterical is more marked. When bilateral, again, confusion may easily arise between this and the "stocking" anaesthesia or "glove" anaesthesia of peripheral neuritis; and the diagnosis would have to be made on other grounds, unless it should happen that the anaesthesia involve a proximal segment, leaving a distal one free (say the forearm without the hand), which would suggest hysteria rather than peripheral neuritis. Thirdly, hysterical anaesthesia may be patchy, scattered about in irregular islets and areas, particularly upon the trunk. Lastly, in rare cases general anaesthesia of the whole body has been observed. Among all these varieties it may nevertheless be affirmed that anaesthesia, which definitely follows the distribution of a particular nerve-trunk, is not likely to be hysterical.

We shall speak of anaesthesia of the special senses (sensorial anaesthesia) presently: it may be mentioned here that this often accompanies anaesthesia of the skin; thus, in what may be called a perfect hysterical hemianaesthesia the skin is anaesthetic, say on the left half of the body, face, and limbs; and the patient cannot smell with the left nostril nor taste with the left half of the tongue; the hearing power is deficient in the left ear, and the left eye sees less well than its fellow. Sensorial anaesthesia has, however, been observed sometimes on the side opposite to the cutaneous hemianaesthesia. In other cases, in which there is affection of special sense without any hemianaesthesia, a relation may still be traced between the special sense and the superficial structures of its neighbourhood. Thus, in hysterical deafness there may be anaesthesia of the pinna, meatus, or membrana tympani; and, in blindness, of the lids or cornea. It is true that this relation is said not to be constant; but when present it may aid in the diagnosis of hysterical sensory defect.

Hysterical anaesthesia may appear suddenly after a fit, excitement or emotion; or may come on gradually; or may exist, as I have said, unknown to the patient. It may vary much from week to week. It is common to find hysterical paralysis of movement and of sensation combined. Briquet regards anaesthesia as an index of somewhat severe and chronic hysteria; Charcot, as one of the common marks of the disease. The following curious observation may sometimes be made in cases of hysterical anaesthesia; and it is used by Janet to shew that the patient really retains sensation, though she does not consciously perceive; and is analogous to the prism experiment on vision which is described on p. 707. Say that a patient is completely hemianaesthetic, feeling on one side neither touch nor prick. Now let the mode of testing be varied a little, and instead of asking her "Do you feel it?" when each touch is made, ask her to say "yes" when touched, and "no" when not touched. Some patients will then as regularly reply "no" when touched on the anaesthetic side as they do "yes" when touched on the sound side. One is tempted to think they are shamming, but against this, among other objections, may be urged that the anaesthesia may sometimes persist during sleep. Thus, a hemianaesthetic girl, who answered in this seemingly ridiculous way, could not be waked at night by a prick on the hemianaesthetic side of the face, whereas prick on the sound side roused her.[1] Still, from many patients no such sign of recognition can be obtained; and some, of course, would see at once the contradictory character of the proposed answer.

---

[1] But three other patients, examined for me by Dr. Edwin Bramwell, did wake when pricked on an anaesthetic part. Janet, too, says that such anaesthesia often disappears during sleep.

KING'S COLLEGE HOSPITAL
MEDICAL SCHOOL.

2. Hyperaesthesia.—(*a*) Pain.—It may be doubted if hyperaesthesia, in the sense of exaltation of sensory function, occurs in hysteria. Hyperalgesia is common, whether it be in the form of spontaneous pain or in that of tenderness. Frequent as hysterical pain is, I cannot profess to give any precise canons for its diagnosis other than the general hints upon hysterical symptoms already made. Common forms are—pain in the head, of which the well-known clavus is an example, namely, a fixed boring pain in a limited spot, commonly one temple; pain in the back; in the side of the chest; at the epigastrium, and in the abdomen. In the hysterical affections of the breast and of the joints, which will be referred to later, pain is the leading symptom. With many of these pains cutaneous tenderness is associated. Pains about the head, in the teeth, and so on, may often be called neuralgic; a true neuralgia—pain, that is, limited strictly to a given nerve district—is rare, according to Briquet; though French authors describe such cases.

(*β*) Tenderness.—Tenderness is even commoner than spontaneous pain. It may be extreme, so as to be provoked by the mere contact of the dress, or the slightest unusual touch; or it may be discovered first during medical examination. Such tenderness may be either cutaneous, or subcutaneous (perhaps muscular), requiring in this latter case somewhat firmer pressure, or movements of the parts underlying the skin, to bring it out; or, thirdly, it may require quite deep pressure. It has several points of predilection; the commonest are the inguinal region, the epigastric, the inframammary, and the vertebral column, or points closely adjacent to it. Tender points about the head are rather less common, and upon the limbs very infrequent. Except when mesially situated, as upon the epigastrium and vertebral spines, they are commoner or generally better marked on the left side of the body than on the right. In certain cases (more frequently, it would appear, upon the continent of Europe than here) these tender points acquire an increased importance from the fact that it is possible by pressure upon them to produce or to control an hysterical fit. This seems to shew their essential connexion with the hysteria (though it is not to be denied that areas of cutaneous tenderness may arise from other causes). They are then called hystero-genetic zones. The best-marked hystero-genetic zone is to be found in the left inguinal region, and was at one time supposed to be connected with the ovary; but this can hardly be so, for similar results can sometimes be obtained by pressure on the same area in hysterical men.

For hysterical affections of the organs of special sense see p. 706.

Motor Phenomena.—Varieties of paralysis.—Motor affections are also common in hysteria; and if not more frequent than the sensory, at any rate force themselves more upon the notice of the patient and physician. Under the rough heading of paralysis we may distinguish several varieties. First, though the limbs retain their muscular power, the patient may be unable to use them for such functions as standing or walking. This may be due either to a simple disability, of which no further account can be given, or to the occurrence, when the attempt is made, of pain or of tremors or of irregular ataxic movements. Inability to stand or walk properly, coupled with preservation of muscular power, may suggest the diagnosis of tabes; but the presence of knee-jerks and of normal pupillary action will generally negative this. Secondly, there may be the symptoms of true paralysis. Such paralysis may be called simple when the patient appears incapable of exercising proper muscular force, and the limb is therefore either feeble or motionless, but in other respects normal—that is, there is neither contracture, nor wasting, nor alteration of tendon-reactions, nor of electrical irritability; a condition perhaps most comparable to that which obtains in a hemiplegia of organic origin when quite recent. But sometimes (not commonly, I think) there may be wasting of muscle. When this occurs, especially if accompanied (as may happen) with blueness and coldness of the skin, it is important to distinguish such paralysis from that due to poliomyelitis, or to neuritis. Hysterical muscular wasting does not pick out particular muscles, but involves the limb as a whole, or by segments; there is no fibrillar twitching; the tendon-reactions are not annulled; electrically there is no reaction of degeneration, though there may be a simple reduction of irritability. The wasting is usually not extreme; it may come on (and disappear) with comparative rapidity.

Contracture.—Lastly, hysterical paralysis may be of a spastic kind. Contracture or rigidity is indeed common in hysteria, so common that the liability to its occurrence under slight provocation has been regarded as one of the distinctive marks of the disease. Sometimes this is manifested in the mild form of increased tendon-reactions; the knee-jerks are exaggerated, and a single tap on the tendon makes the patient start all over; or repeated taps throw the leg into more or less permanent extension. An attempt at ankle-clonus—a few short irregular jerks—is often to be obtained, but whether definite ankle-clonus occurs in hysteria is a moot point. Sometimes there is permanent contracture, resembling in many respects that which accompanies degeneration of the pyramidal tracts in the cord. The limbs may be fixed in various positions, but the most common (as in contracture from organic disease) are—for the upper limb, flexion of the fingers, wrist, and elbow, with possibly adduction of the humerus (but, according to Gilles de la Tourette, these statements are liable to exceptions); for the lower limb, extension of the hip and knee with adduction of the hip: the foot is often in the position of equino-varus. The contracture affects all the muscles, flexors and extensors alike, but preponderates in one of these groups so as to produce the given posture. Paraplegic rigidity of both lower limbs is a common form, or contracture of one upper limb; or sometimes of individual segments—toes, ankle, or fingers. Multiple contractures accompanied with much pain are described. Less commonly, perhaps, parts other than the extremities are affected; for example, the muscles of the neck, causing torticollis: or the trunk muscles, causing spinal curvature; or the masticatory muscles, causing trismus, and so forth. Charcot suggests that certain features distinguish hysterical contracture from that of organic disease, as follows:—(i.) Its onset is much more rapid, it may also disappear rapidly, and recur again, and its appearance may be determined by causes (for example, mental shock, slight injury to the limb itself) which could hardly have produced cerebrospinal degeneration; (ii.) the contracture may be extreme, so that the nails may be driven into the palm, or the leg may feel like an iron bar, whilst attempts to overcome it cause the patient much pain and alarm; (iii.) it does not amend during and after natural sleep; (iv.) anaesthetics, unless they are pressed far, do not produce resolution. With respect to this last point it may be noticed that in some old-standing cases of hysterical contracture, even when muscular relaxation has been obtained by chloroform, there may be a further obstacle to mobility of the joint in the shape of fibrous adhesions, which must be forcibly broken down before movement is possible.

The distribution of hysterical paralysis may be varied. There may be a monoplegia—paralysis of one limb, or part of it. This but rarely results from cerebral disease; whilst from the monoplegia of spinal or neural origin the hysterical form differs in the absence of electrical reaction of degeneration, and in the distribution of the anaesthesia which may accompany the paralysis. Hysterical hemiplegia is a well-recognised form. The distinction from hemiplegia of organic origin rests on several points. Thus, there may be an absence of all conditions which lead up to cerebral disease, such as lesions of the heart or vessels, and of the signs of cerebral tumour; whilst other hysterical symptoms may be present, and among these notably hemianaesthesia. The more marked and definite the hemianaesthesia in comparison with the paralysis, the more likely is the case to be hysterical; and when the hemianaesthesia affects the special senses in the manner already described the probabilities in favour of hysteria are very great. A difference in gait was pointed out by Todd; the hysterical hemiplegic drags the leg after her like a lifeless appendage, in organic disease it is circumducted with the object of clearing the toes from the ground. He also stated that in hysterical hemiplegia the face and tongue are not paralysed. This is the general rule, but one to which there may be some real, as there certainly are some apparent exceptions: by apparent exceptions, I mean cases in which the face is distorted and the tongue deviates, but in which such deviation or distortion is due not to paralysis, but to unilateral spasm. Hysterical paraplegia is common and may give rise to very great difficulty of diagnosis. Paraplegia of the flaccid type, due to organic disease, whereof peripheral neuritis affords the commonest example, is characterised by absence of knee-jerks and usually by alteration of electrical reactions which easily distinguish it from hysteria. Much less easy is the distinction from those forms of organic disease which give rise to paraplegia of the spastic type; say from a transverse dorsal myelitis, or still more from disseminated sclerosis. Where dorsal myelitis is in question, the following points should receive careful attention:—(i.) The limits of anaesthesia, if this is well defined. In myelitis the upper limit corresponds to the level of the cord involved, and the anaesthesia affects all the skin below this level; in hysteria the anaesthesia is more likely to be distributed on the plans we have already described. (ii.) The condition of the sphincters. Retention of urine is of course common enough in hysteria, but incontinence either of urine or faeces is exceptional. (iii.) The presence of bed-sores or cystitis, which point to myelitis. (iv.) The condition of the reflexes. Increase of knee-jerk helps us but little: the presence of a definite ankle-clonus, especially if this have been observed to increase gradually and steadily, militates, I think, against hysteria. Dr. T. Buzzard points out that a certain combination of reflexes is highly suggestive of hysteria; namely, when the knee-jerks are increased, and the plantar reflexes are absent, or are at any rate so sluggish that they can only be obtained by prolonged tickling of the soles. In cases of functional paralysis the plantar response is flexor; in some cases, as described by Dr. Buzzard, the toe movement does not occur, but the contraction of the tensor fasciae can still be made out. But where there is any organic lesion involving the pyramidal tract, the type of response is apt to be different; *extension of the toes*, of the great toe specially, Babinski's sign (*vide* Vol. VII. p. 312), is the prominent feature. And although with a normal (flexor) reflex we cannot altogether exclude organic disease, yet where the abnormal (extensor) reflex persistently occurs we may confidently affirm that organic disease exists. (v.) The mode of onset. An acute transverse myelitis in which there is a sudden appearance of characteristic symptoms offers little difficulty; neither, perhaps, does the subacute meningo-myelitis, commonly due to syphilis, which is marked by the incompleteness of the motor paraplegia, with disproportionate exaggeration of tendon-reflexes, trivial sensory affection, and distinct affection of the sphincters. In the more chronic forms of myelitis a gradual but steady advance of the paraplegia, painful cramps in the legs, jumping and drawing up of the legs at night, tell against hysteria and for organic disease. In some instances the diagnosis between hysterical paraplegia and that of disseminated sclerosis may be almost impossible; for the characters of the paraplegia itself may be such as to give little aid, and the history of the disease and the general condition of the patient may be misleading, unless some symptom can be discovered which definitely points to organic disease.

The course of hysterical paralysis is by no means uniform. It may appear suddenly, as is the case when it follows an hysterical fit. If the nature of the fit be clear, this throws much light on the nature of the paralysis; but the nature of the original fit may be obscure; thus, an hysterical hemiplegia may succeed an attack of hysterical coma, and so the sequence of events suggests organic disease. Shocks of all kinds, whether mental or bodily, are a potent influence in producing hysterical paralysis, which may either follow at once or after a period of latency. Thus, when an injury to a limb determines an hysterical paralysis of the part, there may be a few days' interval, marked perhaps by a sense of discomfort or numbness, before the paralysis appears. Or the onset of hysterical paralysis may be gradual, and its exciting cause uncertain. The paralysis may also disappear suddenly, and under influences similar to those which provoked it—fits, emotions, and the like. Recurrences may take place. Or, again, improvement may come slowly, possibly only after much persistent treatment. Paraplegia is apt to be the most obstinate form. Or, lastly, hysterical paralysis may endure for years, a reproach to treatment and a disappointment to prognosis.

Reflex phenomena.—The condition of the various reflexes is a subject which naturally follows that of hysterical paralysis. Simple matter of observation as this may seem, yet it appears to be one on which opinions differ. As to the tendon-reactions, I hold that total absence of knee-jerk is never caused by hysteria pure and simple. Exaggeration of knee-jerks (or of any tendon-reactions) gives us no such diagnostic criterion, for this may be found both in hysteria and organic disease. (It is not uncommon for hysterical and neurasthenic patients, when the patellar tendon is tapped, to jump and start as if frightened or hurt.) As regards ankle-clonus, I think that where this is persistent and well established the fact tells against hysteria; but I acknowledge that imperfect ankle-clonus and spurious clonus, which may be difficult to distinguish from the true, are not uncommon; there is no reason at first sight why true ankle-clonus should not occur in functional disease.

Next, as to the superficial reflexes. I have referred to Dr. T. Buzzard's observation that in hysterical paraplegia the plantar reflexes are often absent, and this, as it would appear, whether there be anaesthesia of the feet or not. Again, it is common knowledge that the fauces in hysterical patients often shew great lack of irritability. Does hysterical anaesthesia of the skin affect the condition of the skin reflexes? If, as Janet holds, such anaesthesia is psychical, the skin reflexes should remain; and he affirms that they do. Other authorities, equally distinguished, maintain the reverse. Dr. Head distinguishes two groups—the "psychical" cases, wherein the reflexes remain, despite anaesthesia, the "cerebrospinal" cases, in which they disappear. Further observations, therefore, are desirable, and particularly of those cases in which an anaesthetic area on one side of the body can be contrasted with a corresponding normal area on the other. On the other hand, it is generally allowed that anaesthesia does not interfere with such processes as the raising of a blister, the reddening of the skin by irritants, or the reaction on stimulation of erectile tissues. The reflex action of the pupil to light remains unaltered in hysteria.

As to the functions of micturition and defecation, retention of urine is well known to be fairly common in hysteria. This symptom may stand alone, or it may be associated with paraplegia. It is apt to appear and disappear irregularly. It does not, as a rule, cause distress, or entail serious consequences, albeit Brodie narrates a case in which fatal cystitis followed. In extreme distension the catheter may be necessary; but if there be no urgent need for this the patient should be induced, either by dint of some local application, or by insistence, to empty the bladder voluntarily. One explanation of this symptom is that the retention is due to anaesthesia of the bladder which prevents the patient feeling the need for micturition; but there may be other factors, such, for instance, as spasm of the sphincter. The condition known as irritable bladder consists in suprapubic pain and distress with frequency of micturition; the symptoms differing little from those of cystitis, except that the character of the urine is normal. This may be ascribed to hyperaesthesia of the bladder. True incontinence of urine is admitted on all hands to be rare. It is not denied that involuntary evacuations may occur from time to time; but persistent incontinence, like that of organic paraplegia, is not a symptom of hysteria.

Similar statements may be made about defecation. Constipation is common, either as a constitutional condition, or, in severe cases, from anaesthesia or inactivity of the bowel, or in association with spasm of the sphincter. "Irritability" of the rectum, resulting in small frequent stools, has been described by Weir Mitchell; but paralytic incontinence of faeces is not known.

Involuntary Movements.—A. Tremors.—Though paralysis and contraction are the commonest forms of hysterical motor disability there may be others. Of hysterical tremors there are several varieties; the commonest, in my opinion, consists in a rapid, fine vibratory movement, usually seen in the hands, and most noticeable when the patient stretches them out for examination. The head and tongue may also be tremulous. This resembles the tremor of general paralysis, alcoholism, and Graves' disease. Another form is a coarser, slower, rhythmical movement, commonly of the wrist or forearm, on both sides or on one, or affecting the legs as well; and present both when the limb is at rest and when it is in use. The flapping movement of the hands thus produced may recall an advanced stage of paralysis agitans; but there is not the finer "cigarette-rolling" movement of the fingers, and voluntary movement increases rather than checks the tremor. French authors describe a third variety in which the limb is more or less steady when at rest, but on attempts at use becomes subject to coarse, irregular movements resembling those of insular sclerosis or mercurial palsy: this I have never seen. The following aids to the diagnosis of hysterical tremors may sometimes be invoked; such tremors may appear (or disappear) directly after an hysterical fit: they may intermit or disappear without known cause; they may be associated with hysterical contractions.

B. Rhythmical spasms.—Another class of involuntary movements has received the name of rhythmical hysterical spasm; these are of a more elaborate and purposive character. Thus, a patient may be constantly making bowing movements (salutation spasm); or hammering (malleation); or when placed on her feet may be forced to progress by a series of springing movements (saltatory spasm). Such rhythmical spasms are not infrequent in the domain of the respiratory and phonatory muscles, as we shall presently see. The general characters of these rhythmical hysterical spasms are thus stated by Pitres: they are commonest in children or young adults; they generally appear suddenly after a distinct exciting cause, probably traumatic or emotional, or it may be after an hysterical fit; they keep to the same type throughout their course, the same form of spasm recurring with monotonous iteration, and at regular intervals; they cease during sleep, and may sometimes be stopped by pressure on hysterical "zones," by occupying the patient's attention, or by employing the muscles concerned in the spasm in some other way. They are apt to be contagious by imitation. Yet the same author warns us that they are only to be distinguished from non-hysterical spasms by careful examination of the patient and of all the circumstances.

C. Relations of hysteria to chorea.—The rhythmical monotonous character of such movements is enough to distinguish them from genuine chorea. Yet that genuine chorea should sometimes be seen in hysterical subjects is not surprising, when we consider the close connexion that both diseases have with nervous heredity and emotional influences. The two affections would thus either simply coincide, or at most own a common origin. Still it must be admitted that from time to time cases do occur in which the development of ordinary choreic movements is so closely bound up with hysterical manifestations as to justify the diagnosis of a true hysterical chorea. But the term "hysterical chorea" is more often, and perhaps with more etymological propriety, applied to a species of fit, in which the patient goes through some elaborate performance of dancing, leaping, gesticulation, and so forth. This is the so-called chorea major, as distinguished from Sydenham's chorea or chorea minor (see p. 541).

Ocular Symptoms.—1. *Motor.*—Eye symptoms, of both a positive and negative kind, are of importance with reference to hysteria.

There may be a sort of ptosis, in which the eyelids (for the affection is usually bilateral) spontaneously close, and the patient is unable to reopen them. Although this condition may strongly resemble a paralysis of the levator palpebrae superioris, it is probably due in most instances to a spasm of the orbicularis (blepharospasm). Obvious blepharospasm is often seen; either persistent, in which case it is often associated with photophobia, or, still more frequently, during the hysterical fit. The eyes are then shut, or semi-closed, there is often a nictitation of the lids, and attempts to raise the lid with the finger produce increased spasm. As to the other external ocular muscles, I incline to say that there is never a pure and simple paralysis either of single muscles or of muscles supplied by a single nerve-trunk; if this be true, a paralytic squint excludes hysteria. But a squint may be caused by spasm affecting in unequal proportion the muscles engaged in a conjugate movement; such conjugate spasm is, of course, seen in the facial contortions of an hystero-epileptic fit, and may perhaps occur independently of an overt fit. As to paralysis of conjugate movement, possibly this may occur, but certainly not often. Monocular diplopia, that is, the seeing of two or even of more (polyopia) images with one eye, is sometimes complained of by hysterical patients; and this Charcot ascribes to a spasm or paralysis of the ciliary muscle, coupled with irregularities in the refracting power of the lens. These latter give rise to secondary images, which in health can be neutralised by the action of the accommodation, but which appear when the ciliary muscle is no longer under control. He finds support for this supposition in the fact that micropsia, or macropsia, may be associated with this monocular diplopia.

To return, however, to facts more common and easier of observation. I think that nystagmus proper does not occur in hysteria; neither is the reflex action of the pupil to light abolished; and an Argyll Robertson pupil is evidence of organic disease.

2. *Visual.*—Next as to vision itself. Acuity of vision is not increased, though abnormal sensitiveness to light (photophobia) is common enough. Subjective visual sensations may occur as auras in hysterical fits, or as visual hallucinations in the later stages of the hystero-epileptic fit. Attacks resembling ophthalmic migraine—that is, paroxysmal pain in the eye or temple, associated with phosphenes, scotomas, or even, it is said, transitory hemianopsia—are described by French physicians. But hysterical defects of vision are more common and important. Complete blindness may occur, though it is not common; perhaps it is more usual in one eye than in both. Such unilateral blindness has been made the subject of some interesting observations by means of prisms and other apparatus. It has been found that a patient who avers that she can see nothing with one eye, nevertheless when a prism is put before either eye, sees two images, which (like the experiment on anaesthesia mentioned on p. 699) appears to indicate that she really sees without recognising the fact. More often the loss of vision is partial, and the most characteristic form of this is "crossed amblyopia"; that is to say, one eye—usually the one corresponding to the side on which there are other paralytic phenomena, and particularly hemianaesthesia—sees very badly, while the vision of the other eye, though imperfect, is much less affected. Or, again, there may be loss of vision for colours; or imperfect distinction of colours, distributed in a similar way. All these cases of blindness or imperfect central vision may be regarded as the exaggeration of a visual defect which is very common in hysteria, namely, reduction in the extent of the fields of vision. Perimetric observations shew that frequently the fields of vision are narrowed, generally for both eyes, but sometimes more for one than the other. This narrowing is concentric, that is, the fields preserve their normal shape, their size only being altered. The colour-fields are reduced proportionately to those for white, though whether this proportion holds throughout is uncertain, for Charcot regards it as almost characteristic of hysteria that the colour-field for red remains disproportionately large. Concentric reduction of the visual fields he regards as so common in hysteria that it may be ranked as one of the "stigmata" of the disease. Another point with respect to the visual fields, upon which much stress has been laid, is the effect of fatiguing the vision. If

perimetric observations be repeated, within short intervals of time, they disagree with each other, in the sense that the first observation gives a wider field than the second; but if a sufficient interval be allowed before a third observation, the field will be found to have regained its original size. It is argued from this that peripheral vision is more easily tired out in hysterical than in normal individuals. There are, however, certain defects of the visual fields which are extremely rare in hysteria, and, except as transient symptoms, almost unknown; to wit, central scotoma and hemianopsia;[1] at any rate, the discovery of them should lead us carefully to reconsider the possibility of organic disease.

Auditory symptoms have been less completely studied. Noises in the head or ear, and vertigo (if this latter may be counted as an auditory symptom), may occur as the aura of an attack, and hallucinations of hearing as well as of vision may occur in the course of an hystero-epileptic fit. Hysterical deafness has been described. Apparently it may be complete, sudden in onset, and transitory; and, in paroxysmal deafness of this kind, patients have been known not only to lose their hearing, but also to become deaf-mute. Another and perhaps a more common form is that in which the deafness is incomplete, and either unilateral or more pronounced on one side than on the other. This may form part of a sensory and sensorial hemianaesthesia; or there may be a limited anaesthesia of the auditory meatus and membrana tympani; or there may not be even this. (For the diagnosis of hysterical deafness see Vol. IV. Part II. pp. 533-4.) In view of the frequency of middle-ear disease, care should be taken to ascertain the condition of this part before definitely pronouncing a deafness to be hysterical.

Organs of Phonation, Speech, Respiration.—1. *Aphonia.*—The functions of speech and of respiration are liable to frequent disturbance from hysteria. The best known of these aberrations is hysterical aphonia. The patient cannot speak above a whisper; but there is no interference with articulation, nor any sort of aphasia; simply tonelessness of voice. The cough, however, retains its natural tone. Laryngoscopically the structure and colour of the larynx are normal, but the vocal cords are incompletely approximated during phonation; either from imperfect action of their tensors, in which case the edge of each cord remains slightly crescentic in shape and a chink is left between them, or from an additional inaction of the adductors, in which case a small triangular aperture is left at the back of the larynx. Such aphonia may appear and disappear suddenly, often from some emotional cause, or, it may be, in connexion with the appearance of the menses; and it can be quickly, though temporarily, dissipated by the application of sharp faradism or of static electricity to the larynx. Such cases are often extremely easy to examine with the laryngoscope, owing to a lack of irritability in the fauces, which is common in hysteria (*vide* also Vol. IV. Part II. p. 270).

2. *Mutism.*—Far less common is hysterical mutism. There is here an additional disability, for the patient cannot speak at all, and is thus

---

[1] Hemianopsia has been observed by Janet and by Dr. Wilfred Harris.

aphasic as well as aphonic. Indeed the inability to speak is even more complete than in aphasia from organic disease; for in the latter case the patient can generally utter a few words, syllables, or noises, whereas the hysteric may be completely mute. Another difference is usually (it would appear not invariably) to be noticed, namely, that in organic disease agraphia commonly accompanies the aphasia, whereas the hysterical mute expresses himself in writing easily and correctly. Like other hysterical paralyses, the attacks of mutism are apt to be capricious in appearance and disappearance, or dependent on psychical influences and variable in duration; speech may return suddenly, or during recovery stammering may take the place of complete mutism.

3. *Cough, etc.*—There are irritative affections of the phonatory and respiratory apparatus which strictly fall under the head of rhythmical hysterical spasms (Pitres). Foremost among these stands hysterical cough; this is a harsh, obtrusive cough, absolutely monotonous in its pitch and character, which goes on with a regular and wearisome iteration during the day, but ceases during sleep. There is no expectoration, and examination of the lungs and larynx furnishes no clue to the cause. It is commonest in young people. Other laryngeal noises are sometimes produced, which may recall the cries of the lower animals—such as squeaking like a guinea-pig, barking like a dog, bellowing like cattle, mewing, and so on. Epidemics of these strange performances have been witnessed. Again, such normal respiratory acts as sneezing, yawning, hiccuping, may occur in paroxysms lasting for days or longer. There is an hysterical dyspnoea, or rather tachypnoea; the respirations are hurried, even to an extreme degree, and short and laboured, so as to suggest pneumonia or some other acute lung affection; but there is no rise of temperature, no cyanosis, no morbid physical sign in the chest, and the respiration becomes tranquil during sleep.

Organs of Digestion.—*Dysphagia, vomiting, anorexia, etc.*—In the district of the digestive organs there are several affections to be noticed. There may be a spasm of the oesophagus, causing difficulty in deglutition sufficiently severe to simulate an organic stricture, and requiring the passage of a bougie for purposes of diagnosis. The ordinary hysterical globus is sometimes explained as being due to paroxysmal spasm of the pharyngeal muscles; if so, it is by far the commonest hysterical affection of the throat. Hysterical vomiting is not very uncommon, and has been explained variously. In the best-marked form of it, the food gives no pain, but is rejected at once, without much effort, unchanged in quality, and in such quantity that it would seem none had been retained. Probably this is not really so, for a patient may go on vomiting copiously, and yet retain the appearance of good health and nutrition. Yet the use of the scales has shewn that weight is actually lost; and it should be remembered that such vomiting has been known to lead up to grave and even fatal nutritional disturbances. So too with derangements of appetite—capriciousness, perversions, or diminution; these should not be treated too lightly, for they may advance so far as to constitute a serious disease.

In this, the "anorexia nervosa" described by Lasègue and by Gull, the patient feels less and less inclination for food, and possibly vomits what she does take; at first she exhibits an abnormally restless activity of mind and body; but eventually languor and emaciation set in, she takes to bed, and rapidly becomes a mere skeleton, eating nothing, and with all her bodily functions reduced to a minimum. This state is fraught with danger to life, unless judicious and energetic treatment be adopted. Out of such material, where the friends and surroundings supply the elements of fraud or credulity, are made the fasting girls, who from time to time become notorious, and whose exploits have been known to terminate in death (*vide* also Vol. III. p. 397). Other abdominal manifestations of hysteria are flatulence, borborygmi, and eructations of wind; and their nature is indicated by their frequent connexion with hysterical fits. When there is much distension of the abdomen coupled with pain and tenderness, and perhaps vomiting, confusion with acute peritonitis may arise. There is not, however, the wiry pulse or the Hippocratic facies; the tenderness is more superficial, and the distension will disappear under chloroform. Mucous colic and hysteria are very commonly combined.

So-called "Local Hysteria."—Sometimes hysteria appears to concentrate itself upon organs—such as a joint, the spine, the mamma—which come more under the notice of surgeons than of physicians. The local nature of the disease is more apparent than real; still such affections have been particularly described by surgeons first (Brodie, Teale, Skey, Paget), and more lately by the physicians of the Salpêtrière school, and in America by Weir Mitchell.

1. *The Joints.*—So-called hysterical joint disease is mainly characterised by pain which is intensified by any kind of movement, tenderness to touch, and contraction of the surrounding muscles which has the effect of immobilising the joint. Commonly a single large joint, such as the knee or hip, is affected. Apart from general conditions, such as the constitution and history of the patient, the disproportion between the local distress and the general health, the mode of onset and the like, the following points may help us to a diagnosis. There is no shortening of the limb by measure in hysterical hip disease, although the position assumed by the patient, if she can stand, may suggest such shortening; the tenderness is largely cutaneous and spreads beyond the region of the joint itself, for example, on the abdomen, producing "ovarian" tenderness; there is no painful jumping of the limb during sleep, and when chloroform is administered in sufficient quantity to relax the contracture, no organic joint-disease is discoverable. On the other hand, we must remember that certain physical alterations in the surrounding tissues do not necessarily preclude hysteria; thus there may be some muscular wasting of the kind already described, some vasomotor disturbances, as coldness, blueness, oedema of the limb (*vide* p. 712), perhaps paroxysmal heat and tension of the joint, particularly at night, or even a brawny thickening of the connective tissues around it (Weir Mitchell).

2. *The Spine.*—Pain in the back and, still more, tenderness over and

by the side of the vertebrae, are common in hysterical patients. They may constitute the leading symptom and give rise to the suspicion of vertebral disease. The very intensity of these symptoms, and particularly of the tenderness, is an index of their nervous origin; for, with the notable exception of malignant disease, vertebral disease is not acutely painful. The tenderness is often superficial; there is neither distortion nor loss of mobility of the vertebrae, nor any swelling characteristic of abscess or tumour.

3. *The Breast.*—The hysterical breast is not common. Here, too, there is a concentration of pain and tenderness upon one of the mammae. A local examination may reveal nothing to account for these symptoms; but sometimes small movable swellings can be felt in the gland substance, which do not necessarily indicate organic disease; and sometimes, it is said, the whole breast may be swollen, and the skin covering it reddened or discoloured, while yet there is no more than a neurotic basis for the symptoms. A local injury not infrequently starts such hysterical affection of the breast.

I have already stated that hysterical contracture of muscle may produce club-foot, torticollis, and apparent curvature of the spine.

Circulatory System.—1. *Palpitation, Cardiac Pain.*—In the sphere of the circulation palpitation is the commonest symptom; the heart's action is increased in frequency and becomes at the same time painfully evident to the patient. This, like the "globus," may form part of the aura of a fit, or may occur without any fit upon slight exciting causes; or again there may be paroxysmal attacks of palpitation and rapid pulse, even of considerable duration, for which no cause can be assigned. Attacks of pain, spreading from the region of the heart, particularly if accompanied by feelings of faintness or suffocation, may arouse suspicions of angina pectoris; but we shall usually be guided to a diagnosis by looking to the patient's age and sex, the physical condition of the heart and vessels, and the circumstances in which the attacks arise.

2. *Dilatation of Arteries.*—Vasomotor phenomena—namely, abnormal throbbing with dilatation—are also to be seen in the arteries of neurotic subjects, and these may be so localised as to arouse the suspicion of aneurysm. This condition is oftenest seen in the abdominal aorta; but Paget remarks that it is not rare in other arteries, such as the carotid, subclavian, innominate. He also points out certain facts which are useful in the diagnosis of a "nervous artery" as against an aneurysm; namely, the absence of lateral expansion; the preservation of its tubular form; the diminution in the pulsation when the soft parts covering it are relaxed (for example, when a patient with abdominal pulsation is made to sit up); the softness and compressibility of the vessel; the absence of paroxysmal pain; the stationary size of the tumour during months or years.

3. *Ischaemia and Haemorrhage.*—Hysterical patients may present curious symptoms in the domain of the arterioles and capillaries. Limbs that are paralysed or anaesthetic may exhibit also "ischaemia"; that is to say, when cut or pricked, they bleed less freely than normally, or not

at all. This is commonly ascribed to a vasomotor spasm. The opposite condition, spontaneous haemorrhage, is less easy to explain; yet apparently this may happen, even after we have made all deductions for possible fraud and cultivation of the marvellous. That subcutaneous haemorrhage may be caused by a purely nervous disease is shewn by certain rare instances of tabes dorsalis, in which minute or even massive subcutaneous ecchymoses have been known to follow the bouts of lightning pains. The stigmata of the medieval saints may possibly have had a neurotic origin; and, at any rate, modern examples of such subcutaneous haemorrhage have been recorded, without the religious element. Bleeding from the mucous surfaces has been described, as from the nose and from the ear; haemoptysis too (commonly thought to proceed from the throat or mouth rather than from the lungs), and haematemesis. Obviously the mere possibility of haemorrhage owning no organic cause may give rise to great difficulty in diagnosis; but for practical purposes we must remember that such occurrences are very rare, and not to be diagnosed as hysterical unless other causes can be certainly excluded.

4. *Oedema.*—Hysterical oedema, again, would appear to be an unlikely occurrence. Yet the description of it dates from Sydenham. Hysterical swelling of the ankle differs, he says, from dropsical swelling thus—it is greatest in the morning, not in the evening; it does not pit on pressure; it commonly attacks one ankle only. Brodie refers more than once to the swelling which may follow severe pain, and specially mentions a diffuse swelling of the hand and forearm, without redness of skin, which accompanied prolonged hysterical pain in these parts. Under the title of "blue oedema in hysterical patients" Charcot described the swollen parts as cyanosed, that is, blue in colour, sometimes shewing scattered spots of red, and cold to the touch. Higier, again, maintains that some hysterical swellings exhibit a red colour and a raised temperature. In addition to this chronic oedema, which may persist for months or even for one to two years, a paroxysmal form of oedema in hysteria has been described (Edgeworth). Hysterical swelling is no doubt rare, and usually there are other hysterical manifestations in the parts attacked by it, such as pain, anaesthesia, or paralysis; and this may aid in the diagnosis from certain other forms of swelling which may come on without obvious cause.

Nutrition and Secretion.—1. *Statements as to general and local nutrition.*—The consideration of vascular phenomena leads us to the subjects of nutrition and secretion. Under the head of local nutritional disturbance stands the wasting of muscle, to which I have already alluded. Certain other phenomena, such as decoloration of the hair, falling out of the hair and of the teeth, and skin eruptions, such as pemphigus, herpes, eczema, and even cutaneous ulcerations and gangrenes, have been described. Cases of this kind need minute individual study before they can be pronounced hysterical. There is no one general type of nutrition which invariably accompanies the disease. Laycock, indeed, emphasises the *embonpoint* of hysterical women, which he apparently connects with some supposed abnormality in the sexual sphere. Yet we have seen that anaemia, or the exhaustion and emaciation which follow some acute or accompany some chronic disorder, may determine an outbreak of hysteria; and, again, that hysterical anorexia may be the starting-point of a severe and even fatal marasmus.

Certain rare disorders of secretion have been described, such as excessive secretion of sweat, of saliva, of urine, or of milk—this latter even in the virgin. As the commonest example, I may point to the copious flow of limpid watery urine which may follow an hysterical fit.

2. *Composition of Urine after Fits.*—In connexion with this last fact I must mention the researches of Gilles de la Tourette and Cathelineau upon the composition of the urine in hysteria. They state that whereas the urine of hysterical patients in their normal condition, that is, apart from any sort of fit, does not differ from that of a healthy person, yet the occurrence of a fit, or of any paroxysmal phenomenon which is the equivalent of a fit, produces distinct changes in its composition. Thus, if the urine of twenty-four hours, reckoned from the beginning of a fit, be collected, it is found that the total fixed residue, and with this the total amount of urea and of phosphates, is small in amount, and that the ratio of the earthy to the alkaline phosphates is increased. This ratio is in normal urine about 1 : 3, but in the post-paroxysmal urine of hysteria the proportion of earthy phosphates rises, so that the ratio may even become 1 : 1. After epileptic fits, on the other hand, the fixed residue of the urine is increased, and there is no alteration in the "formula of the phosphates." These statements have not been verified in this country, so far as I know; but if correct they are of importance, both because they direct our attention towards the corporeal and chemical side of hysterical fits, and because they would supply important aid to the diagnosis of obscure kinds of paroxysm.

3. *Hysterical Anuria.*—Suppression of urine—"hysterical anuria"—is a truly strange phenomenon, the reality of which has not unnaturally been called in question, but is attested by such observers as Laycock and Charcot. From Charcot's case it would appear that there may be complete anuria for as long a period as eleven days, and a very scanty excretion of urine for still longer periods, without deterioration of the general health, or any consequence other than vomiting. Such vomiting appears to be compensatory, since urea has been found in the vomit. It has been suggested that minor degrees of such anuria may be more common than is generally supposed, seeing that, unless complete and prolonged, the condition might pass unnoticed.

Hysterical Fever.—It may be taken as a general rule that in hysteria the bodily temperature is not raised. And to this we must always have regard in the diagnosis of doubtful cases; particularly where arthritis, meningitis, peritonitis, or deep-seated inflammations of any kind come into question. Again, in persistently recurrent convulsions (*état de mal*) the temperature may constitute a distinction (not perhaps an invariable one) between the status epilepticus and the status hystericus. Nevertheless, from time to time instances present themselves which seem to shew that the temperature of an hysterical patient may rise without assignable organic cause. And, indeed, if we remember how the temperature of sick children and of convalescents may rise from slight causes and even mere emotional stimuli, the occurrence of such "nervous fever" in hysteria will appear less improbable. By fever I mean here simply rise of temperature, as measured by clinical thermometry; I do not assert that there are in such cases corresponding alterations in tissue metabolism. Such fever may come on either in the course of severe hysterical disease, or with comparatively slight affections, such as joint pains, abdominal pains, and so forth—in this latter case itself constituting the leading symptom. It may last for days, weeks, or, it is said, even longer; or be paroxysmal. Unlike most other fevers, no definite course or type can be assigned to it. The temperature may run up to an extreme height (for example, 106°, Debove; 109°, Mierzejewski; 111°, Sciamanna; 118° F., Teale), such as would be fatal in other forms of disease, yet without serious affection of the patient's health; oscillations of very wide range and rapidity may take place (11° in twenty-four hours, Mierzejewski); or the temperature as taken in the different parts of the body may differ absolutely (108° in one axilla, 98° in the other! Drummond). As to the concomitants of this temperature, no general rule can be laid down except perhaps the negative one, that they are not in proportion to the temperature itself. Headache, thirst, frequency of pulse, hurried breathing may occur with little or no rise of temperature—this has been called pseudo-fever; or very high temperatures may be recorded without any of these things, and even without sweats or burning heat of skin. Delirium appears to be not uncommon. Further, the temperature, though refractory to ordinary antipyretic measures, may come down spontaneously, or under influences which might be classed as mental rather than physical. Need I add that two precautions should be taken when the temperature rises in an hysterical case? First, since these facts are very rare, to exclude all possible organic disease; secondly, since they sometimes border on the physiologically marvellous, to exclude malingering and manipulation of the thermometer by the patient.

Mental Symptoms in Hysteria.—1. *General Mental State.*—I have discussed the theoretical importance of the mental state in hysteria, and need here only mention the more obvious mental perversions. No doubt most hysterical patients are unduly impressionable, both as to feelings—in that they react with abnormal readiness and in abnormal ways to emotional stimuli, and intellectually—in that they too readily adopt ideas forced on them from without. This must contribute to instability of character, and hinder a patient, persevering effort. Nevertheless, I doubt whether defects of this kind are universal, and whether every hysterical patient is to be stamped as "varium et mutabile." This, however, is the least part of the indictment brought against them: it is often added that they are vain, egotistical, prone to deceit; and that their complaints are more or less assumed for the purpose of attracting attention. We may dismiss the supposition that hysteria is "humbug." There may be malingering here as among any other class of patients; sometimes perhaps the shamming and hysteria may have developed side by side, as is shewn by the "confessions" of hysterical women, published by Weir Mitchell; but for the bulk of cases this is no explanation. How can a patient sham symptoms of which she has never heard, with groupings and limitations which would be unknown to any malingerer; or assume contractions which may persist during sleep, or anaesthesia of which she is totally unaware? And to go farther, we think that hysterical patients are credited with a far greater power of voluntary control over their symptoms than they actually possess. It does not follow that because a paralysis, for instance, disappears suddenly under some powerful incentive to action, that it was previously under voluntary control; and when we are released from the necessity of finding a motive for the appearance and disappearance of symptoms, we may fairly doubt if facts warrant us in ascribing vanity, egotism, or deceitfulness to hysterical people more than to others.

2. *Amnesia.*—Passing to conditions more distinctly pathological, I may mention two which appear to be rare. Hysterical amnesia, described chiefly by French authors, occurs usually after a fit or some emotional disturbance. A whole section of the patient's past life may be blotted out, so that she may forget, say, the beginning of her illness and all subsequent events, even for an interval of many months. Sometimes this mental deficiency continues, so that she cannot appropriate or remember events as they pass before her in the present. Or, again, the lack of memory centres not round a period of time, but round some person or set of circumstances, so that she forgets them, remembering everything else.

3. *Double Consciousness.*—Double consciousness is another rare condition, no doubt allied to amnesia. The patient appears to possess two personalities between which she alternates, being at one time her natural self, at others apparently thinking and acting like some second being. In each state she remembers only the events of the life which corresponds to her present state, and forgets those of the other.

4. *Insanity.*—Is there an hysterical insanity? That hysterical patients should sometimes become insane is no more than we might expect, considering the absolute frequency both of hysteria and insanity; and, more than this, the fact that both diseases own a common origin, namely, hereditary nervous taint. Nor would it be surprising if insanity, complicating hysteria, were tinged with the colours of the previously existing neurosis. But apart from this, we do not know that there exists any form of permanent insanity, which by its mode of origin or special characteristics can be justly described as insanity "ab hysteria." Subject, therefore, to the correction which further study of this subject may bring, we are disposed to adopt the French view, that the only true hysterical mania is delirium, transitory in duration, sudden in onset and disappearance, and essentially the same as the hallucinatory stage of the hystero-epileptic fit. It is nevertheless right to remind the reader that "simple hysterical

mania" is recognised by eminent authorities on mental diseases (*vide* p. 893); who presumably regard such mania as a manifestation of hysteria. It is difficult to decide what symptoms are to be included under the heading of hysteria until we have a satisfactory definition and hypothesis of the disease.

General Remarks on Diagnosis.—It would evidently be impossible to discuss all the diagnostic problems to which hysteria may give occasion : we must limit ourselves to some general remarks, and to a mere reference to the conditions which are likely to give most trouble. Hysteria may be mistaken for organic disease, or conversely ; or, again, both states may coexist, and the one may mask the other. To overlook organic disease may do grave injury to the patient and bring discredit on the physician ; to treat hysteria as organic disease may prolong and intensify the hysteria. To avoid the first, and more serious, of these errors we should look carefully for any of those symptoms which tell—some absolutely, some less strongly—in favour of organic disease. Such, to recapitulate, are :—in the sphere of the eye—optic neuritis, optic atrophy, palsies of individual muscles or of muscles supplied by individual nerve-trunks, nystagmus, palsies of the pupil, persistent hemianopsia, or central scotoma ; in the sensory sphere—anaesthesia in the district of special nerve-trunks ; in the motor—paralysis and atrophy of individual muscles, or of muscles falling under special nerve districts, and well-defined electrical reaction of degeneration ; in the reflex sphere—absence of knee-jerks, well-defined and persistent ankle-clonus, and above all an extensor plantar response, and incontinence of urine or faeces ; in the sphere of nutrition—bed-sores. If all these signs are absent, still the case may or may not be hysterical ; the former diagnosis is rendered probable by the discovery of any hysterical "stigmata," such as anaesthesia of the segmentary or hemiplegic form, hystero-genetic zones or tender points, visual troubles of hysterical character, fits, globus, and the like ; and, if we can establish a connexion between such admittedly hysterical symptoms and those which are under discussion, this points to the conclusion, that hysteria is responsible for them all. If positive indications in either direction fail us, we can only look to the course and development of the symptoms—have they progressed steadily and after the manner of any known organic disease? or do they fluctuate in response to mental and moral influences or without apparent cause? Lastly, we must take into account the vaguer characters suggested either by the general grouping of symptoms or by the temperament of the patient.

It is obviously in its beginning, when symptoms are few and ill-developed, that organic disease is most likely to be mistaken for hysteria ; and in diseases other than those of the nervous system, pain and tenderness are the symptoms which will probably come under discussion. The mere fact that we can discover no cause for a pain must not tempt us to call it hysterical ; such possibilities as deep-seated inflammation, aneurysm, tumour, or bone disease ought never to be forgotten. The reaction which these, or maybe even the pain arising from them, cause in the general condition of the patient, as contrasted with the well-being of many hysterical subjects, is perhaps the best criterion, if we cannot afford to wait and to trust to time for diagnosis. The same remark applies to cases in which hysteria simulates disease of internal viscera (by pain, functional disturbances, or perhaps even haemorrhages); yet not universally, for hysterical disturbances of digestion may produce deplorable and even fatal malnutrition. The difficulties in diagnosis from many nervous diseases are still greater. It is here that wide knowledge and experience are indispensable; and these qualities, if they cannot always ensure correctness in judgment, will at least ensure caution. We may enumerate among these diseases:—epilepsy, when the fits are mild or aberrant, or known to us only in the descriptions of friends; meningitis, when simulated by headache, photophobia, or cerebral irritation; intracranial growths in an early or quiescent stage; peculiarities of gait such as may arise in cerebellar disease, in ilio-psoas palsy, or sporadic cases of Friedreich's ataxia; some forms of tremor and of chronic spasm; paraplegia of all kinds, and particularly the paraplegia of disseminated sclerosis. In disseminated sclerosis there are many opportunities for error. First, because the manner and general appearance are often such as is commonly called "hysterical"; next, because, as Dr. T. Buzzard has shewn, disseminated sclerosis, as seen in practice, often varies widely from the abstract type of the textbooks. The characteristic tremor and the staccato speech may be absent, and the symptoms may be reduced to mere paraplegia with increased tendon-reactions. And, what is still more important, the advance of this disease may be characterised by transient paralysis, transient loss of sight, first in one eye, then in the other, and other indeterminate and fluctuating symptoms which are sure to suggest hysteria. Add to this that the outbreak sometimes follows a mental shock, and it will be apparent how great the difficulty of diagnosis from hysteria may be. The plantar reflex, however, is in most instances very helpful. In pure hysteria it is either absent or normal; in disseminated sclerosis it is generally of the extensor type (*vide* also Vol. VII. p. 841).

There are other conditions, due to general nervous break-down rather than to organic disease, which it may be difficult to distinguish from hysteria. Here, perhaps, it is more a question of distinction than of practical diagnosis. Some such conditions are rare, such as the "akinesia algera" of Möbius and Erb, and paramyoclonus multiplex.

"Neurasthenia" is a name of comparatively recent origin, which is now extending its denotation so as to cover all functional neuroses. If that be permitted, then hysteria is but a subdivision of neurasthenia. But neurasthenia in its original and more limited sense describes a state which older physicians might have called hypochondriasis, and which presents the following contrasts with hysteria:—It is commonest in men; it usually begins in middle life or later, and often owes its origin to overwork, anxieties of business, and consequent intellectual strain; the symptoms are less varied in character, and more purely subjective—vertigo, insomnia, dyspeptic feelings, and cerebral and spinal "sensations" being predominant—yet they are more persistent and more refractory to treatment, and reduce the patient to a state of mental depression rarely seen in hysteria. Still in some cases the line cannot be drawn; and even Charcot, who sharply distinguishes the two conditions, admits that they may coexist. And one cause, namely, injury with nervous shock, is capable of eliciting either of them, so that a "traumatic neurosis," as it is sometimes called, may be either hysterical or neurasthenic in its mode of manifestation. However, for a full discussion of neurasthenia, and of its differential diagnosis, the reader is referred to the article on the subject (pp. 727, 767).

General Remarks on Course and Prognosis.—We can scarcely speak of a definite course for hysterical symptoms, so irregular are they in themselves, and so largely dominated by external circumstances. This much we may say (and the mere possibility of truthfully and persistently asserting it is an aid to treatment), that however grave and persistent, they may always eventually get well. But the diathesis is in bad cases very permanent; that is to say, where there is a marked nervous heredity, or where, from adversity of circumstances, hysterical tendencies have become deeply ingrained, symptoms will be hard to treat, and subsequent relapses likely. The likelihood of relapse is all the more evident, when "stigmata" such as anaesthesia, "ovarian" tenderness, and so forth, persist. Yet it would appear that the advance of old age may sometimes ameliorate even a bad hysterical diathesis. In mild hysteria, such as frequently occurs in young people, we need not take so gloomy a view; many women, who have had fits and other symptoms in earlier life, subsequently attain to perfect health.

The disease is said to present different features in different classes of patients. Thus, in grown men hysteria is said to depend less on emotional causes than in women, and more often on trauma; and its symptoms to be less manifold and fluctuating, but more difficult of cure. There is also a tendency to mental depression rather than to the exaltation and vivacity which we are apt to associate with hysteria. Men of effeminate or sedentary habits are not more liable to it than the workingman. In children hysterical symptoms are commonly less persistent and more easily treated than in adults. Charcot thinks, too, that in children the usual stigmata are often absent, and the disease is "monosymptomatic"; some striking symptom, such as rhythmical movements, paralysis with contracture, a false arthritis, or mutism, standing perhaps alone. In the hysteria both of children and adult men it is said that nervous heredity is generally a prominent feature. Perhaps the race and country may introduce modifications from the type of the disease. Thus, severe hystero-epileptic fits appear to be less common in this country than in France, and the hysterical stigmata work out with less regularity here; similar observations have been made by American authors.

Does hysteria ever cause death? Strange as it may appear, we must answer, yes. Sometimes, as I have already said, death is due to exhaustion and inanition,—this may happen in cases of hysterical anorexia, or of oesophageal spasm; sometimes again to violent convulsive seizures, especially, as it would appear, to seizures in which dyspnoea is a striking feature; sometimes in thrombosis of the cerebral sinuses or other abrupt ways. Briquet gives a list of some 12 fatal cases; Charcot mentions that he knew of 4; Weir Mitchell had seen 3. Still, a fatal result is so rare that, except perhaps in cases of severe progressive inanition, it need not materially overshadow our prognosis. Such prognosis, to recapitulate, is good in the main (for recovery from any hysterical condition is always possible); but should be tempered by the remembrance that some symptoms are extremely obstinate, may last, in fact, for years; and that, unless the diathesis be mild, or the circumstances uniformly favourable, relapses are probable after apparent cure.

TREATMENT.—A. **Preventive.**—With regard to the prevention of hysteria, one principal factor, namely nervous heredity, is at present beyond the control of the physician. So long as lunatics, epileptics, and drunkards can propagate their kind without restraint, so long will the broad basis of this and of other neuroses remain. But no doubt the neurotic tendency may be minimised by a wise education and upbringing, since the direct antithesis of hysteria is the "mens sana in corpore sano." The hygiene of youth has been dealt with already in the first volume of this work; nevertheless, I must here emphasise the importance, for children of a nervous disposition, of proper feeding from infancy upwards, of country air, outdoor occupations, regular and early hours, and abstinence from luxuries and unwholesome stimulants of whatever kind. In a happy and well-regulated nursery or schoolroom, with healthy companions, the seeds of hysteria do not grow apace; but where the parents themselves supply the source or the example of nervous instability, the conditions of health must be sought elsewhere than at home. The mental education must be prosecuted, but must on no account be allowed to outrun the physical. It should be calculated to provide intelligent interests for the present and the future, and to necessitate active attention and application of the mind; and, without being dull, should be austere rather than sentimental. Long hours of mechanical fingerwork, or of occupations that leave room for solitary reverie, novel-reading, or even the exclusive study of poetry or art, are not advisable. And, to touch a higher sphere, we may say that few things are more opposed to hysteria than the trustful, patient, altruistic spirit inculcated by Christianity, and few things more conducive to it than the terrorism, revivalism, mysticism, or self-concentration which sometimes pose as religion. Certain periods of life need particular care: namely, those of the first and second dentition, when eclampsia, night-terrors, sleep-walking and other nervous manifestations are apt to occur; that of puberty, when the nervous system is being modified to meet the bodily changes of sexual development, and the period which follows it, when (as Janet well says) the correlative mental development is being carried out, and the individual has first to face the great problems of sexual, social, and religious life.

B. **General Treatment.**—In dealing with declared hysteria, the diathesis or general condition will be our principal object of attack, though we may have also to reckon with particular symptoms. Further, the treatment, like the disease itself, presents both a psychical and a physical side, which it is difficult to separate. Nearly all that I have said concerning preventive hygiene may be transferred, *mutatis mutandis*, to treatment. Not only must healthy conditions of bodily life, proper food, good air, employment, and regular hours be secured, but the inner life must also be regulated, and such exciting causes as fears, anxiety, misery, ennui, and depressing emotions of all kinds must be removed. Obviously this may be sometimes impossible; we may, in fact, be unable to fathom, much less to remove them. The restoration of the general health, when this is impaired, is a matter of great importance. Indeed, it may be said that when there is some bodily ailment, which can be efficiently and speedily removed by simple methods, the prognosis of the hysteria is better than when we have no such tangible thing to treat. Anaemia is the best example of this; difficulties and irregularities of menstruation another. Sometimes hysteria occurs in connexion with symptoms of local disease, generally pelvic disease; and we have to face the question which is the predominant factor, and whether local treatment will do good by removing a source of irritation, or harm by encouraging a chronic invalidism. The general physician would then be wise to obtain an authoritative opinion of some well-known (and not too special) specialist, remembering that what can be done quickly and thoroughly will probably be beneficial, and prolonged tinkering the reverse.

The question of marriage, which is likely to be mooted in some cases, really falls under the head of general conditions. Marriage is no specific cure for hysteria; indeed, some of the worst cases occur in married women. It is of advantage when it brings beneficial changes in the physical and mental environment, and the reverse when it entails anxieties and adversities.

The relation of the physician to his patient is important. First, she must believe in his superior knowledge and skill, and that he possesses the clue to the puzzle of her symptoms. We know the advantage of this faith in the treatment of any disease; but in hysteria it is essential that there should be no wavering assertions, no vacillating directions. What, then, is to be done in those difficult cases in which certain diagnosis between hysteria and organic disease is impossible? Something must here depend upon the character of the patient; there are those whose mental needs can be met by nothing less than vigorous assertion (wherein we take the risk of error); others to whom we can truly and sufficiently say, that we can discover no reason why they should not recover. Doubts must be expressed to the friends only, or kept to ourselves. Of course, should the diagnosis lie between hysteria and some disease that calls for immediate and special treatment, such as a surgical operation, the case is different, and we must make up our minds at any cost; but luckily this is not often the case.

Secondly, however sure we may be that symptoms are hysterical, that they rest on no organic basis, may at any time clear up, and are worse in proportion as attention is directed to them, we must not treat them as mere nonsense or matter for ridicule. This would merely put us out of touch with the patient; we must enter into her point of view in order to convert her to ours. And it is safest not to attempt too sudden a conversion; for though there may be exceptional physicians who can tell a paralysed hysteric to arise and walk, and exceptional patients who will obey the command, yet a failure only makes future treatment more difficult. Time and steady pressure are more certain agents, and, indeed, there are patients, as Weir Mitchell says, who must be "urged and scolded, teased, bribed, and decoyed along the road to health." Another matter of prime importance is the influence upon the patient of home and friends. In mild and recent hysteria, when the conditions of life are rational and happy, and when the relatives are capable of exercising firm and wise guidance, things may well be left alone; but circumstances are often not so favourable, friends, in particular, are apt to be over-anxious or weak, or in some cases the best friends may be thoroughly baffled by the persistence of the disease. In such instances, the patient must be removed from home, friends, and the conditions wherein the complaint has grown and flourished, and be placed under some wise and competent surveillance. This alone may do much; but it may be necessary to carry out a more complete isolation, namely, to keep her in solitude with her attendants, shielded from disturbing influences, and accessible only to those which we calculate will aid recovery. Such treatment is best carried out in a recognised "home" or hospital, and is usually combined with some drastic form of physical treatment, such as I shall presently describe. Evidently, then, the general treatment of hysteria may be no easy matter: it may imply considerable command of circumstances; on the part of the physician and his coadjutors it requires penetration, tact, firmness, and kindness; on the part of the patient a confidence and self-surrender peculiarly hard for her to exercise.

Special Modes of Treatment.—1. *Plan of Weir Mitchell and Playfair.*—Special modes of treatment have often to be adopted. Foremost among them is the plan devised by Weir Mitchell, and introduced by Playfair to this country; the essential points of it stand thus:—The patient is removed from home and friends, and placed in seclusion with a suitable nurse. At first she is absolutely confined to bed, and put on milk diet. Next, passive muscular exercise by means of massage and faradism is begun; and, as increased metabolism is thus ensured, the diet is also increased till the patient takes a large quantity of mixed food as well as milk. In about six weeks the patient may begin to sit up, and then be gradually restored to normal conditions of life. To travel for a time, with her nurse as companion, may form a good prelude to her return into home life. It may be useful to give details of this treatment, which I take from an article by Playfair. It is better carried out in a medical home or hospital than in lodgings. The choice of a nurse is most important; she must be kind, sensible, and firm, and in education and manners a suitable companion for the patient; probably she will have enough to do without undertaking the massage and electrical treatment. Further, if the patient, rightly or wrongly, is unable to get on with a particular nurse, it is best to make a change. In severe cases confinement to bed, without any physical exertion whatever, is required, say, till the fifth or sixth week of treatment, when the patient may begin by sitting up for an hour or two, next to go out for a short walk or drive, and so onwards. Massage is begun on the second or third day, and given twice a day; each administration lasts a quarter of an hour to twenty minutes at first, and is gradually increased to one hour or one and a half; by the time this maximum of two or three hours a day is reached the patient should also be taking the full diet.[1] In fact the capacity for food is the test of the massage; if in a week or ten days the patient is unable to assimilate the food given her, the massage is ineffective, and the operator should be changed. About ten days is the time in which full diet and full massage are reached, but with feeble delicate patients a longer time may be required. After each massage an hour's complete rest in the blanket, in a darkened room, is advised. When, towards the end of treatment, the patient begins to sit up, the afternoon massage is stopped, and thus by degrees it is discontinued altogether. Electricity, in the form of faradism to the principal muscles, in applications lasting from twenty minutes to half an hour daily, is sometimes used as an adjuvant to the massage; this may be begun when the patient has been about two weeks under treatment. At first the nurse must feed the patient. Three ounces of fresh milk every three hours are then given; in a day or two 5 ounces, and then 10 ounces. She will now be having a quarter to a half-hour's massage twice daily. Next, solid food is begun; at first some breakfast, then a fish dinner, then a minced chop, till by degrees the full diet is reached (in favourable cases this is attained in about ten days). If any dyspeptic troubles result from the over-feeding, solid food must be stopped for twenty-four hours, and then resumed. The surest test of progress is the weight; an emaciated patient may gain at first 5 or 6 lbs.

---

[1] Playfair gives the following specimen of full diet:—

Breakfast.—Plate of porridge with a gill of cream, fish or bacon, cocoa or *café au lait.*

At 11 *a.m.*—Cup of beef tea, with two teaspoonfuls of beef peptonoids.

Luncheon at 1.30.—Fish, cutlets or joint, with a sweet such as stewed fruit or a milky pudding.

At 5 *p.m.*—Beef tea and peptonoids as at 11 a.m.

Dinner at 7 *p.m.*—Soup or fish; joint or poultry; sweet.

It must be remembered that in addition not less than 80 ounces of milk (10 ounces every three hours), sometimes 100 or 110 ounces, are being taken.

per week, and afterwards 2½ lbs. per week; and if a patient do not gain at least 2 lbs. per week, it may be assumed that she is not doing well. This method of treatment succeeds best with patients who, by dint of long-continued hysterical paralysis or anorexia, have fallen into a state of chronic helplessness and emaciation; and its success depends partly on the moral influences involved in removal from home—in the steady help and encouragement given by doctor and nurse, in the demonstration of daily improvement—partly on the tissue renovation effected by the passive exercises and over-feeding. The results are less favourable in those who are not emaciated, but unduly fat;[1] in those who appear in good bodily health; and in those whose complaints are not so much of helplessness and paralysis as of pains, headaches, insomnia, and other symptoms of a subjective type.

2. *Electrical treatment* may often be employed with advantage, both for the relief of special symptoms and for the general condition. Static electricity is convenient and efficacious: the patient is charged from one pole of a Wimshurst machine, and the charge is either allowed to dissipate itself gradually, or may be taken off from any part of the body, in the form of sparks if a brusque effect be desired, or as an electrical douche (obtained by breaking up the spark by a suitable electrode) if a milder stimulation be required. Or general faradism may be given, either in the form of a bath or in the usual way. The high-frequency current may be used as an alternative.

3. *Hydro-therapeutics* are recommended by the French physicians; and the essential part of such treatment appears to consist in the bracing effects of cold water followed by a good reaction. This is most effectively secured, according to Gilles de la Tourette (in whose work detailed directions will be found), by applying a douche of cold (say about 50° F.) or chilled water for five, fifteen, or twenty-five seconds, according to the temperature, to the trunk and limbs, avoiding the head, and avoiding any hyper-sensitive zones. The douche is to be followed by brisk friction. Or in delicate or sensitive patients the douche may be begun with warm water, which is rapidly cooled down. Other forms of cold-water bathing may be used, provided always that reaction be obtained.

4. *Drugs.*—Unlimited physicking is to be deprecated; still drugs are often beneficial, and it has yet to be shewn that their effect is purely moral. Tonics are most generally useful, particularly metallic tonics, iron above all; also zinc, which may well be given as the valerianate, and arsenic. Narcotics, such as opium, morphine, chloral, sulphonal, and the like, are best avoided, but may be required to meet emergencies. Briquet,

---

[1] Abnormally fat patients must be subjected to a preliminary process of thinning as follows (details here also from Playfair):—Confine patient strictly to bed; diet with skim milk, at first 2 quarts per diem, given in small quantities every two hours; after a day or two lessen this gradually, till not more than a pint per diem is taken. If the patient's strength fail unduly under this, some beef tea or soup may be temporarily substituted for the milk. Under the minimum diet the weight gradually lessens about ½ lb. per diem (the patient should be weighed daily), and when some 14 or 20 lbs. have been taken off, pure milk may be substituted for skim, and the treatment as detailed in the text pursued.

it is true, recommends a course of opium for some cases; but we must remember the great danger of producing a drug habit. Bromides, however, may be given in small doses (gr. x. ter die) for a considerable time, and may be combined with hyoscyamus or valerian; or with tonics, such as iron, arsenic, or nux vomica. A combination of bromide and digitalis (pot. bromid. gr. x., tinct. digitalis ℳ x.) is often particularly useful in reducing the number of hysteroid attacks; but larger doses of bromide have not nearly the same potency in this direction as they have in epilepsy.

5. *Hypnotism.*—As to the treatment by hypnotism, I have very serious doubts. There are so many similarities between the hypnotic and hysterical states, that in hypnotising for the cure of hysteria we may be said to favour the very condition we want to eradicate. At any rate we produce a dreamy, semi-conscious state, in which the subject's mental activities are removed from his own control; not a good training for an hysterical patient. I admit that this is an a priori objection, subject to correction by experience, and further, that some symptoms may be so urgent as to require removal at any cost; still, I notice that the tide of treatment by hypnotism, which in some quarters has flowed so high, shews signs of ebbing; and eminent authorities declare that useful therapeutic suggestions may be made quite as well without hypnotism as with it.

Treatment of Symptoms.—The treatment of hysterical cases must be mainly directed against the diathesis; by attacking each symptom as it arises we run the risk of perpetuating the series. Yet the control of individual symptoms may be advisable or even necessary, both for the relief and encouragement of friends and patient. It would appear, too, that in some chronic cases a symptom may persist, as it were by habit, after the hysterical diathesis has largely subsided.

1. *Of Fits.*—Mild hysterical fits are best left alone; if restraint be really necessary, it should be applied in some quiet but effective way; against a mere show of force the patient will struggle all the more. There are many devices for cutting a fit short, which mostly consist in the sudden production of some painful impression. Cold effusion to the face is often effective; one way of applying this without drenching the patient is to pour a small well-directed stream from a little height upon the face, and to direct it, when the mouth opens, back upon the fauces; or the face and neck may be smartly slapped with a wet towel; or the supraorbital nerve may be compressed where it emerges from the notch; or best of all, if it be available, a sharp faradic current may be applied with a wire brush. Other plans are to stop the mouth and nose till the "besoin de respirer" becomes insupportable; or to exercise firm pressure on some tender area, particularly the so-called "ovarian" region. As soon as the patient begins to recognise her surroundings she should be encouraged, and commanded to wake up and gulp down some cold water. But it may often be best simply to watch the patient through the fit, or to let her severely alone; and in any case, alarm or fussiness on the part

of the bystanders tends only to make matters worse. To terminate such paroxysmal states as trances, catalepsy, and the like, the faradic wire-brush is probably the best means, unless it be possible to control them by pressure upon some hystero-genetic zone.

2. *Of Pain, Anaesthesia, Paralysis, etc.*—I doubt whether local treatment of painful and hyperaesthetic parts is generally advisable: still it may become a necessity sometimes. For tender joints and spines Paget recommended sponging with water as hot as can be borne; cold he believed to be mischievous. Yet Briquet recommends cold application for hysterical pains; or, better still, counter-irritants of various grades—friction, mustard, iodine, blisters. Perhaps cutaneous faradisation is the best counter-irritant, as it can be repeated at will. There can be little harm in giving drugs of the antipyrin class for temporary treatment of headaches and neuralgic pains. For anaesthesia, especially if it be limited to the surface, faradism with the wire brush is an efficient remedy. So is faradism of the muscles for many of the milder forms of paralysis; and the instantaneous effect of faradism applied to the larynx is particularly striking in hysterical aphonia. But there are obstinate and inveterate palsies, of which hysterical paraplegia most often affords an illustration, whereof we can hardly expect a rapid cure. Here massage, passive movements, and faradism must be applied persistently; and when the least voluntary movement returns, the patient must be encouraged towards daily progress, however slight, and each advance demonstrated to her as an earnest of complete recovery. For many such cases, indeed, it is best to adopt the Weir Mitchell method in its entirety from the beginning.

3. *Of Contracture.*—Contracture may be difficult to deal with. In the earlier stages, and more limited forms thereof, we should avoid surgical treatment, remembering that such contracture is in fact often produced by splints and immobilising apparatus. We shall limit ourselves to general treatment, coupled with massage of the limb and frequent passive extensions, painful though these may be. With inveterate and severe contractures the case is different. It may be necessary then to administer an anaesthetic to determine the condition of the muscles and of the joints; and, if adhesions be found, to break them down, or to perform tenotomy, with the object of rectifying or preventing dislocations of the articular surfaces; and to follow up these measures by mechanical extension, either by way of splints or weighted lines. Weir Mitchell maintains that the pain which sometimes exists in the lower limbs, where these are rigidly flexed at the hips, is due to pressure on the sciatic nerves, and can be relieved by tenotomy. But surgery has indeed a very limited scope in the treatment of hysteria; for evidently hysterical disease of the joints, of the spine, of the breast, etc., concerns the surgeon only as regards the diagnosis.

4. *Of Digestive Symptoms.*—Hysteria and dyspepsia are often hopelessly intertwined. The ordinary stomachic drugs, such as bismuth, bicarbonate of sodium, rhubarb, and the like, are then useful; nux vomica and iron as

soon as they can be given. Asafetida, once the supreme antispasmodic, finds its best applications for eructations and flatulence. In some cases of hysterical vomiting it seems probable that the regurgitation comes from the oesophagus, and it may be well to try feeding by a nasal tube. In severe hysterical vomiting it may be necessary to feed per rectum; but, as a rule, some food is retained by the stomach, and it is wisest to insist upon feeding by the mouth. Nevertheless in dieting all cases of hysterical disorders of digestion we must ensure that a sufficiency of really nutritious food be taken (impairment of nutrition being certain to prove the beginning of a downhill course), and avoid therein two extremes, that of insisting on a full diet which may be acting injuriously on a really weak stomach, and of restricting (or allowing the patient to restrict) the diet till she rejects everything. The latter course may be the beginning of the grave disturbance of digestion and nutrition called anorexia nervosa, which should be promptly met with isolation and enforced feeding under the care of skilled nurses (*vide* Vol. III. p. 403).

J. A. ORMEROD.

REFERENCES

1. BABINSKI. "Du phénomène des orteils et de sa valeur sémiologique," *Semaine méd.*, Paris, 1898, xviii. 321.—1*a*. *Idem.* *Ma conception de l'hysteria et de l'hypnotisme* (*Pithiatisme*) 1906.—1*b*. *Idem.* *Démembrement de l'hystérie traditionelle—Pithiatisme*, Paris, 1909.—2. BASTIAN. *Hysterical or Functional Paralysis*, Lond., 1893.—3. BREUER und FREUD. *Neurol. Centralbl.*, Leipz., 1893, xii. 4, 43; *Studien über Hysterie*, 1909, Leipzig u. Wien.—4. BRIQUET. *Traité clinique et thérapeutique de l'hystérie*, Paris, 1859.—5. BRODIE. Vol. iii. of Brodie's works as arranged by Hawkins, *Lectures on Local Nervous Affections*, Lond., 1865.—6. BRUNS. Apud *Neurol. Centralbl.*, 1895, xiv. 522.—7. BUZZARD, T. *Clinical Lectures on Diseases of the Nervous System*, Lond., 1882.—8. *Idem.* *Simulation of Hysteria by Organic Nervous Disease*, Lond., 1891.—9. CHARCOT. *Leçons sur les maladies du système nerveux*, Paris, 1877-87, vols. i. iii.—10. *Idem.* *Leçons de Mardi*, Paris, 1892-3.—11. CLARKE, MICHELL. "Critical Digests upon the Subjects of Hysteria, Neurasthenia, etc.," *Brain*, vols. xv.-xvii. xix (References).—12. *Idem.* *Hysteria and Neurasthenia*, 1905, John Lane.—13. COLLIER, JAMES. *Brain*, 1899, xxii. 71.—14. DONKIN. Article "Hysteria," Tuke's *Dictionary of Psychological Medicine*, 1892.—15. EDGEWORTH. "Hysterical Paroxysmal Oedema," *Quart. Journ. Med.*, Oxford, 1908-9, ii. 135.—16. FREUD. Apud *Neurol. Centralbl.*, 1896, xv. 434, and 1899, xviii. 408.—17. GILLES DE LA TOURETTE. *Traité clinique et thérapeutique de l'hystérie*, Paris, 1891-95 (References).—18. GORDON. "Troubles vasomoteurs et trophiques de l'hystérie," *Rev. neurol.*, Paris, 1908, xvi. 945.—19. GULL. Collected writings (ed. by Acland), p. 305, New Sydenham Society's publications, 1894.—20. HARRIS, W. *Brain*, Lond., 1897, xx. 308.—21. HEAD, HENRY. *Brain*, 1893, xvi. 114 foll.—22. HIGIER. Apud *Neurol. Centralbl.*, 1894, xiii. 735.—23. JANET, PIERRE. *Arch. de la neurol.*, xxiii., xxiv., xxv.; *L'état mentale des hystériques*, Paris, 1892; *The Major Symptoms of Hysteria*, 1907, New York and London.—24. LAYCOCK. *Treatise on the Nervous Diseases of Women*, Lond., 1840.—25. LÖWENFELD. *Pathologie und Therapie der Neurasthenie und Hysterie*, Wiesbaden, 1894.—26. MEIGE, H. *Presse méd.*, Paris, 1908, 425.—27. MITCHELL, WEIR. *Lectures on Diseases of the Nervous System, especially in Women*, Philadelphia, 1885.—28. *Idem.* *Clinical Lessons on Nervous Diseases*, Phila. and New York, 1897.—29. MÖBIUS. *Neurol. Beiträge*, Leipzig, 1894.—30. PAGET, J. Clinical Lectures and Essays, ed. Howard Marsh, *Lectures on Nervous Mimicry*, Lond., 1875.—31. PITRES. *Leçons cliniques sur l'hystérie et l'hypnotisme*, Paris, 1891.—32. PLAYFAIR. Article "Hysteria," Tuke's *Dictionary of Psychological Medicine*, 1892.—33. RICHER. *Études cliniques sur la grande hystérie*, Paris, 1891.—34. SKEY. *Lectures on Hysteria*, Lond., 1867.—35. SYDENHAM. *Epistolary Dissertations*, 58 foll., and *Processus Integri*, 1-21.

Hysterical Hyperpyrexia.—36. Debove. *Bull. Soc. méd. des hôp. de Paris*, 1886, 3. s., iii. 209.—37. Drummond. *Brit. Med. Journ.*, 1888, ii. 1397.—38. Mierzejewski. *Neurol. Centralbl.*, 1890, ix. 573. (Abstract).—39. Sciamanna. *Neurol. Centralbl.*, 1890, ix. 574. (Abstract).—40. Teale, John W. "Excessive and long-maintained High Temperature, after Spinal Injuries, with Recovery," *Trans. Clin. Soc.*, 1875, viii. 98. Sequel, 1889, xxii. 357 (duration of the raised temperature was seven weeks, maximum temperature over 122° F. ; rapid alterations of temperature in a few hours were noted without apparent alteration in the condition of the patient).

J. A. O.

NEURASTHENIA

By Sir Clifford Allbutt, K.C.B., M.D., F.R.S.

We can hardly begin to discuss or to describe neurasthenia without being met by the fundamental question, if there be such a thing? Yet unless we can agree upon the subject of our argument we argue in vain. Are there then among the elusive varieties of disease, especially of nervous disease, persons suffering after the manner we are about to describe, who in their symptoms are not only superficially alike, but so far alike also in more intimate experience and analysis as to make it convenient for us, as clinical observers, to put them in a category together?

If, then, a critic demurs that in his opinion the phenomena to be enrolled under neurasthenia are too diffuse, too scattered, too distributed, too evanescent, to be classified together; and that the abstract conception, or the type, is too hazy for recognition and comparison, the demur, whatever its validity, will be at least a reasonable one. But if he imports into the group, as too often he does, alien phenomena, of whatever nature and origin, merely because they are vague and equivocal—such, for example, as those of hypochondriasis, of hysteria, of bad temper, of incipient hyperpiesis, of infective states intrinsic or incidental, of incipient general paralysis of the insane, or other insanity, of incipient Graves' disease or tuberculosis, of convalescences, of transient overwork and worry in persons fundamentally sound, and so forth, his demur will fail of justification. To say that the cases grouped as neurasthenia are of too elusive and indeterminate a character for classification is one thing, to use the category as a rag-bag for heterogeneous sets of symptoms, elusive and indeterminate not so much of their own nature as of the stage of their development or of the slipperiness of their diagnosis, is another. Furthermore we are told that in the absence of "objective" signs there can be no disease; and that neurasthenia has no criterions. Now, without quibbling about the validity of the term "objective," we may surely ask what are the "objective" signs, let us say, of delusional insanity? But, as we shall find when we come to the investigations of Kräpelin and Rivers, in neurasthenia the defect of objectivity depends less on the inefficiency of the disease than on that of us its observers. For my part I am still of opinion that there is in this respect a frequently recurrent series of morbid phenomena which, even if they be vagrant, protean, and elusive, have a large common-denominator, and may conveniently be grouped together and called by a name; and the name of Neurasthenia happens to have prevailed. But the more indecisive the criterions the more vigilant should be our diagnosis: to make neurasthenia everything is indeed to make it nothing.

The convenience and reasonable significance of the class and name of neurasthenia are overtly or tacitly attested by general adoption; they are adopted for a special chapter in all systematic works on Medicine, home and foreign, and in treatises on Nervous Diseases (as, for instance, in one of the latest, that edited by Curschmann).

Space does not permit me to reject one by one the attribution to neurasthenia of the many sorts of toxic consequences which false diagnoses have thrust under this name; I venture to think that Savill's interesting contributions to the subject were impaired by this kind of confusion; I must pass on to the objection that we are proceeding upon "a false analogy" when we accept certain results of "trauma" as a variety of neurasthenia; that it is as it were "to call coma a disease." Surely not; coma is a single symptom or term, neurasthenia is the name not of one term but of a series: moreover, to assume the analogy to be "false" is to beg the question; the reply is that if we are proceeding upon analogy, we are proceeding upon homology also. Those who have patiently laboured at the analysis of both these morbid series recognise that, as is the case with other maladies, "traumatic neurasthenia" is a variety which combines terms of two series, of the hysterical as well as of the neurasthenic series; as we shall discover hereafter.

The reasonableness of the name and category of neurasthenia lies in the undesigned coincidence of the medical testimony which we have to consider; thus compared, the agreement of causes, features, and points in the class proves to be remarkably uniform. The overwhelming majority, if we omit the pathologists we may almost say the unanimity, of clinical physicians, recognises in man a characteristic series of symptoms, recurring with fair uniformity, to which the name of neurasthenia has been given. It is true that the more intimate natural form, the processes of molecular perversion and the static modifications, if any, which underlie this series are unknown; but no more true than in asthma for instance, or epilepsy. A malady which consists chiefly—as neurasthenia confessedly does—in a complexity of subjective symptoms, must offer great difficulties to the positive methods. These we have to carry into the dim and intricate recesses of the psychosensory and psychomotor receptions, associations and determinations by which work is framed and liberated; and in these recesses of function dominant, subdominant, and contingent factors are so complex and elusive as to have baffled research almost to this day—*almost* to this day, for at last some glimmer is shed on the way by the investigations of the schools of Kräpelin in Germany, and in England of Rivers, M'Dougall, and others. German wits have been turned to this investigation by the system of insurance against accidents which has recently been established in their country—a system which affords convincing evidence of the existence of large groups of cases of the kind labelled as neurasthenia. This malady, as a clinical category, every considerable German physician, so far as I am aware, accepts as distinct; yet in the circumstances of insurance particular cases are involved in a tangle of equivocation, guile and artifice most difficult to unravel and expose. The shrewd physicians who are continually engaged in adjusting the defence of the national fund on the one hand with the proper claims of the injured on the other, may indeed by practice attain to a sagacious insight and to rules of thumb whereby, on the history and clinical picture, fair approximations to justice are to be made; but to demonstrate before the law and the litigant parties the steps by which definite conclusions are reached is another matter. Were such litigants divisible into sheep and goats, had we to deal only with rascals on the one hand and honest patients on the other, the always delicate task would be less fallacious; but matters are more tangled than this. Dismissing the rogues for a moment, a patient of common honesty cannot shake himself clear of the bias of self-interest. An upright patient is apt to resent cold-blooded appraisements of his health, and unless in fear of death would prefer a little indulgence to the indifferent eye of precise justice. When ailing a man takes a sad view of things; he is reduced more or less to the child's incapacity of seeing through the murk of instant sorrow; he sees himself, and it may be a wife and children, deprived of his right hand; in justice to himself or to them he must not take for granted a tittle of the lassitude which besets him, nor a shade of the pain which dogs his work. The physician did not seem to listen to every complaint; he did not put his finger exactly on the very spot of the pain. *Sollicitae mentes speque metuque pavent.* To appreciate the evidences, therefore, the appellant must exaggerate them, as an artist heightens his picture by accentuating the shadows. Moreover, his nerves are quivering, he feels he is not coherent, not eloquent; his mind wanders; he must bore and reiterate where he cannot logically persuade. Now the liberal or illiberal settlement of a claim at any rate dissipates this atmosphere of uncertainty, these quasi-conscientious appeals; the patient gets out of his querulousness, and by making the best of the consequences, unhappy as they may be, finds that things might have been worse. Then he has liberty to recover.

These being the problems, are there any tests adequate to the purpose? What are the conditions under which energy should flow in and out? Well, without complicating this argument with a revision of the many methods with much ingenuity attempted or pursued by Kräpelin and his pupils—of whom Amberg and Specht should especially be mentioned—we may now say that at last the main lines at least of such a method are being set out. The test which ultimately they have fixed upon, and are making use of in practice, is addition of columns of units, a bell being rung by the drawing of a line at the foot of each column. Between columns, or groups of columns, longer or shorter intervals are inserted at the discretion of the observer. To this important condition of interval we shall return. Dr. Rivers and Dr. M'Dougall, after like series of trials, have discarded doing sums for hitting at dots, made to pass a slit at rates determinable by the experimenter; fatigue being betrayed by inaccuracy of aim. Besides other advantages this method, having something of a game about it, is more attractive to the patient.

Fortunately it appears that the quality of the individual's test work may be neglected, and attention concentrated upon its quantity (Rivers and Kräpelin). Now, of course, the first procedure was to put normal and neutral persons, under rigorous regimen, to courses of trial, so that if possible a standard of health might be computed. It is obvious that the complexity of the conditions is such that, if error is to be reduced sufficiently, the series of experiments must be long and manifold; and, already no inadequate train of work is on record. With the seat and mode of fatigue we have not at present to deal; these questions, by the different methods of separate muscle testing as well as testing of the whole working organism, are under investigation, especially in Verworn's laboratory and at Cambridge; but the subject before us now is the measurement of fatigue, however and wheresoever generated or seated.

Let us then suppose a curve to be plotted out by taking as ordinates the times, and as abscissae the work done on the sums (Kräpelin) or on the dots (Rivers); if now the work be set agoing till the patient comes to a standstill, we shall obtain a very complex curve, the many cross components of which have to be analysed before we can interpret it. Comparisons of many curves taken under very various conditions have brought out the nature of these perturbations, the subordinate contingencies by which the deflexions are produced. Fortunately some of these interferences, such as "habituation,"—an underfactor of "practice," and spurt—an underfactor of attack, prove to be comparatively negligible; so that for ordinary use we may be satisfied with three subdominant factors, subordinate to Time and Fatigue, namely Practice, Attack, and Momentum. Concerning Practice it is obvious that by repetition a certain facility is gained, varying with the individual but more easily discerned than might be supposed. Attack is a translation of "Anregung," which I propose as better than "impulse" or "incitation"; it signifies the promptness of starting—the "warming to work" of Rivers—the "getting hold of the beginning," as the oarsman would put it; a faculty which is separable from Practice, and varies in different persons of equal practice. Momentum explains itself, as denoting the rate of running down on cessation of work; it is also a quality in which individuals vary widely: in some persons, after, say, a five minutes' stop off, the engine is still running, so that on beginning again there is no inconsiderable inertia to the good. By divers kinds of test-curve, and by many of them on many various individuals, formulas of these several subdominant faculties have been obtained, so that fatigue curves can be read off with no little discrimination; with so much indeed that simulated fatigue is betrayed at once. Did the curves consist simply in work and time only, simulation might be successful; but the manifold trials have signally proved that even assistants skilled in the conditions of the tests, with time given to think out a plan of simulation, have all failed to imitate the natural curves with their dominant and subdominant characters; and the attempts of outsiders, however wily, are quite clumsy and tell-tale. Space will not allow me to go far in illustration of these most important tests, but a few instances seem desirable: thus facility of Practice alters the curve by making the acme earlier; as in the individual practice improves, it comes earlier and earlier until the acme may shoulder up towards the first ordinate. Dr. Rivers finds that not only does the capacity for practice vary much in different individuals, but so also does the retention of it; some persons of high capacity for practice have a low capacity for retention of the skill, on laying the tests aside it soon wanes; and it is notable for us that these persons are very liable to fatigue—their acquirements are unstable. The fatigue-recovery rate, on the other hand, is little altered by practice. Again, as to the pauses; on the whole the more the fatigue the better the effect of pause, but not regularly so, for here comes in momentum; if the individual machine soon runs down, and so has to be restarted from or near rest, even brief pauses will tell against tale of work. The places and durations of pause therefore count for much; but on the whole momentum is inversely as fatigue, and pause effect is parallel with fatigue. Fatigue in all curves and pauses comes out then as the dominant factor; a multiple of even two or three times capacity is needed to obscure, or rather to postpone, fatigue. Under such conditions the experiment has to be prolonged; but, generally speaking, plus number of experiments—say periods of five minutes with short pauses—is better than plus duration. Some persons present a comparatively high fatigue-rate, but so good a recovery-rate that no abnormal condition is indicated; and I desire especially to emphasise this law—that in liability to fatigue normal individuals differ widely. This physiological law we may compare with the less precise clinical assumption of "latent neurasthenia," made by Jendrássik and others.

When after all these pains standard curves had been obtained, Kräpelin found himself at liberty to apply his method for testing cases of traumatic neurasthenia, especially those in which pecuniary damages were involved; to these uses I shall return hereafter, but while by such methods the factor of "interest" is reduced to lower ratios, investigation is still embarrassed not only by the actions but also by the reactions of mind; in other words the influence of the uppermost webs of the brain cannot yet be discriminated.

Although, then, these methods have advanced into practical use, as yet they are adapted for partial investigations, for research, and for appraisement of compensations, rather than for current clinical service. For current work simpler means are required; and we are not without such alternatives, though of less exactness. Rules of thumb have been formulated by several observers; but I may refer especially to the observations of Dr. Warner on tests of muscular relaxation, and of economy of muscular effort. Dr. Warner first shews us in detail how fatigue is multiplied by unnecessary expenditure; as for example by moving the head along the lines in reading, in using muscles of the forearm and shoulder in driving a bradawl, and so forth. I have found that the occipital (muscular) headache of some neurasthenic students may be relieved by placing a support for the head at the back of the reading chair. All such "waste labour," which makes for exhaustion, Dr. Warner tries to prevent. Among his tests are such as this: after half-an-hour's or an hour's school work he directs the child to stretch out his arm and hand forwards, and notes if, at once or very soon, the thumb droops, then the fingers, then the wrist, and so on. He points out that the orbicularis palpebrarum is one of the first muscles to betray fatigue, when its slackness becomes visible as a puffiness under the eyes. Also, by noting the inability of the child to give continuous attention to the instructor while performing the prescribed muscular actions, his want of spontaneity, and his bias to watching his own limbs and motions, Dr. Warner infers the degree of that introspective habit in which the neurasthenic is only too prone to be enthralled. By these and other similar observations he finds it possible to detect, even in children of quick wit and eager temper, a want of resisting or sustaining power, or a waste of energy, and to recommend the appropriate training and precaution.

Let us now turn to a brief speculation on the nature of fatigue. I suspect that of fatigue there are at least two kinds, kinds which I may call respectively catabolic and atonic fatigue; and that these kinds, though often concurrent, may move independently. For my own part I think that in neurasthenia the prevalent fatigue consists not so much, perhaps comparatively little, in the accumulation of waste in the muscles or blood, but in the atony.

On the right principle of proceeding in thought from the normal position to the abnormal, we may try to grope our way from the margins of health into the obscurities of the alleged malady of neurasthenia. In sound health a weary man after a hard day's work goes to bed and there sinks instantly into a deep sleep, a sleep of eight or nine hours. During this change, peripheral stimulus being reduced, his muscles relax; his head and jaw drop, if his limbs are raised they fall flaccid; their reflexes by their quickness betray their release from inhibition. By personal experience we know that, as they are being thus abandoned, twitches will occur in them, in obedience, no doubt, to casual cutaneous irritation. On rising from bed, as Professor Sherrington has explained to us, tone, and the harmony or associative synergy demonstrated in attention by Flechsig, are restored by flexion and extension movements, a generalised yawning; but buoyancy is scarcely high until with the cold bath and active friction tone is once more poured out freely upon the muscular system from the stores replenished during the night. But if the day's labour had been excessive, on the next day, even after the night's sleep, the man may remain temporarily neurasthenic, languid, and snappish. On awaking, the limbs may still be consciously slack, so that the loins, thighs, and legs, and in a less degree the shoulders and arms, are wrapped in a pall of lassitude—"lassitudo," in the words of Celsus, "quae citra fatigationem sit" (*De Med.* i. 2). The day is tasteless, the morrow is vanity; the grasshopper is a burden. Now tone may consist in a spontaneous stream of energy from centres to periphery; more probably it is an awakened response to incessant tiny bombardments upon the special and general surfaces; but as in sleep the limb-jerks are heightened, and by other analogies, it would seem that the withdrawal from active service is rather on the part of the higher and inhibitory than of the inferior centres; and clinical experience points the same way. We observe that, generally, control centres do weary before the subordinate systems; strong control and intense determinations of attention proverbially fatigue us. We remember in the dozes of the night, how, under loss of uppermost control inferior thoughts career through the ungoverned subordinate offices of the mind. To awake from sleep, then, seems to be a passage from what, in another sphere of science, is termed an "adiabatic" to a "diabatic" state of energy; from storage to release, from abeyance of muscular activity to muscles "on the edge of action" but held in a position of restraint by the total balance of forces.

Within proper limits then of fatigue nature in sleep draws the shutter for what I may venture to call restorage. Indeed rests less complete, an hour or two in an easy-chair, serve the same end, though but imperfectly and provisionally; sleep must be had, sooner or later. For, if sleep be withheld, the patient, for such he then becomes, again and again depletes his stores, and, like the diabetic in the later stage of his malady, has to begin to feed the fire with chippings of the furniture. Bankruptcy is then within measurable distance. It is true that there are neurasthenics who can sleep, but these cases are the few. Sleeplessness may not be a *causa sine qua non*, but it is a malignant abettor. The languor of normal sleepiness thus resembles the languor of neurasthenia, and as an abeyance of tone may be identical with it; but the states behind them are widely apart. By sleep the normal man, fatigued as he may be, retires into potential abundance; the neurasthenic, whether by lack of sleep or by other exhaustion, is in his upper or control centres at least reduced to poverty. For these reasons, after a study of the researches in Verworn's laboratory and at Cambridge (Langley and W. M. Fletcher) as well as of the clinical phenomena, I am not convinced that the neurasthenic state is essentially one in which the vital energy is in no great defect, but is clogged by the accumulation of waste products in the blood, or muscles, or both; there is no particular evidence of this beyond a few alleged cases of indicanuria and the like, cases which, being few, were probably of complex causation, or of false diagnosis. Commonly the excretory organs signify no such intoxication, or only so much as may quickly be dispelled without cardinal change in the malady. All the processes of the body are languid, it is true, but the condition seems to be one of intimate defect of potential energy, perhaps in the large central ganglia of the brain; and the means of radical cure must consist in laying up working capital at the back of all the outward functions. In what vital resistance consists we cannot tell; nor how far potential energy may be used up in molecular frictions and other invisible work. The axis-cylinder itself seems to be more than a mere conducting wire, and to be an energy-containing tissue; but Dr. Waller says by virtue of its sheath it does not undergo fatigue, disintegration and reintegration being synchronous and equivalent. Fatigue seems to extend far, perhaps infinitely far, beyond the sphere of apparent activity, and the general capacity is reduced. Thus the old maxim that rest may be found in a change of work is fallacious, as indeed psychological experiment has demonstrated; and of this conclusion we must take due heed in therapeutics.

If we are agreed that sleeplessness, though a terrible adversity, is not the *causa sine qua non*; and if the fault lie in the exhaustion of the resources of centres of control, what and how is this fault? Are these centres smaller in size, less capacious, than they ought to be? is their molecular constitution reduced or labile? or are their sources of supply—the vascular for instance—temporarily or permanently defective in content? We are apt to forget how little we know of the laws of cell activity. There seems to be some relation between the diffusion rate and the vitality of the cell, and Hugh Ross has shewn how variable is the coefficient of all diffusion, and—as bearing on our problem—how sensitive to the action of certain drugs, especially of morphine. Upon such conditions, among others, diabasis and adiabasis may depend. Alkalis and acids likewise affect this process. Here it is instructive to contemplate the normal man in another aspect; namely, under certain variations of climate. Men vary in their susceptibility to climatic influences, and these variations are so mutable and manifold as to lend themselves ill to study; but, explain it as we may, many men at any rate are soon affected by climate, especially by damp and warm airs. Perhaps no European can defy such an atmosphere in the tropics; though the equally hot but dry air of the desert is more tolerable. Hot dry air seems to dispose to spasmodic outbursts of energy, to flashes which do not endure; moist heat, even in natives, to perpetual indolence. Fales, who describes these cases in Europeans, says that on returning home their symptoms soon pass away. My experience of them is not so sanguine. In Europeans who have long resided in the damper tropics, this acquired neurasthenia with loss of sleep, nervous irritability, incapacity for work, and fatigue, is often most obstinate. To call it a fantasy or a "psychosis" is absurd. Two persons known to me, who are obliged by their position in life to live and work in relaxing climates in our own country, are there soon reduced to a state virtually of invalidism, which passes off quickly on removal to brisk mountain airs. Here they can undertake long excursions and enter into the usual pleasures and occupations of life. But in this change there is no mental interference; they are ill, not idle; all their chief interests lie in the place and sphere of their work, and the frequent change of climate is an inconvenient interruption and expense. Dr. Campbell Smith—the translator of Ballet—who practised for many years in a very relaxing climate, in his preface expresses the same opinion.

In respect of "Psychosis," Eichhorst, like his compatriot Dubois of Berne, is prone to confuse neurasthenia with morbid ideas ("krankhaften Vorstellungen") making it as a corporeal malady factitious. As he attributes to neurasthenics "paraesthesia of all the senses" he also would seem to confuse hysteria, a different, probably an essentially different disorder, with neurasthenia. Many English authors also, aspersing civilisation, declare that a mental twist is at the back of every case—is the one common feature; and yet not untruly, if inconsistently, they interpret the malady as an inadequacy of "vital income" to "vital expenditure." Surely every physician whose practice has carried him into both private and public circles can testify by many particular cases that neurasthenia may not even embrace mental disability, let alone effluxions from it. To assume "psychical" failure or deviation, even in an insidious sense, as a primary cause in such cases, which are of a kind common enough, is without justification. Such an opinion, built upon no data, is a freak. If, with Janet, the name "psychasthenia" be still proposed for the "central cases," we should still deprecate a term which would bring after it cardiasthenia, gastrasthenia, and so forth, names of far more cumber than they are worth. Even in the frequent cases of eccentric causation in which mental application does fall into abeyance,—as in the relatively external condition of "eye-strain" for instance—we must remember how the attention flags, and how in the ordinary tiredness of common life the mind will droop from Shakespeare to novels, from novels to newspapers, and from newspapers to—bridge! Loss of mental grip, mental confusion, "empty head," true absence as opposed to preoccupation of mind, are still more often secondary to mere bodily exhaustion or common tiredness. Moreover, in most cases there is, perhaps in excess, a reflective appreciation of the defect of capacity; and the sense of uselessness, of baffled purpose, of waning hope, and too often of failing resources, conspire to mental gloom.

Dr. Drummond puts forward very ably this "psychotic" point of view; he would fix also the causes of the mental dereliction or perversion in a previous treachery to the higher life, in a refusal of all "large and generous ideals," in starved or wounded affections, in a seclusion of ideas "in a groove," in egotism, in a submission of the mind to bodily sensations, and so forth. It seems to me, as I have said, that in not a few of the cases to which such reasonings may be plausibly applicable causes and consequences are confused together. Every disease has its psychological equivalents or echoes; but the saddest feature of many a neurasthenic is the ardent desire to buckle to work, and yet the baffling exhaustion which follows every attempt to go forward.

E. Müller likewise dwells too much on the psychical origin of neurasthenia, declaring that the mental defect dominates the malady; but he errs more than Drummond and Dana in lack of discrimination between hysteria and hypochondriasis and neurasthenia; distinctions to be delineated hereafter.

As a particular call upon the system becomes more multifarious, and the more elaborate the web concerned, the more does it flow into the intricate paths which we vaguely indicate as psychical. The matter is one of degree; but the moment direct response to impact is suspended, in that moment, whatever the level, inhibition begins, effort begins, and fatigability begins. The victim of prostatic disease has too good reason to discover that the vesical reflex is indefatigable, and that it is the inhibitory suppression of it which is fatigable; and thus it is with the woman who dare not trust her vesical control in church or concert-room. Reaction capacity, as we have seen, varies inversely as the place in the hierarchy, so that we observe anarchical "unrests" in a system of which the government is enfeebled. Thus if in some cases neurasthenia seems to be a diffuse disease, in others it is selective, for, as pathologists know we may have selective disintegrations as we have selective reinforcements. Certain centres and tracts of the nervous system may lose capacity, or be ill-endowed originally, while others remain efficient.

By these considerations we can understand the difficulty of deciding if or how far the child can be properly neurasthenic; his responses are normally far more direct, he stores them less, so that he is subject to the fatigues of inhibition in far less degree; yet energy runs to waste, and the reservoirs run low, and in him also we perceive how profoundly education consists in an edification of controls, in a development of the more complex webs of inhibitory mechanisms. An outbreak of passion is a "let-go"; a convulsion is a let-go; but preoccupations, automatisms, submissions to suggestion are not a let-go: they consist in closures, fixations, or exchanges of the several fields of activity. Of this kind are the manifestations of hysteria.

Once more, we have noted that of millions of impacts upon our receptive surfaces but a very few or none may obtain immediate answer, or even rise into attention. But as in a "highly-strung" person the receptive surfaces are unusually sensitive, the inhibitory management has more work; and for such persons it is all in all that during youth the mechanisms which serve it should be developed to the fullest. If this development fall short, or the receptive surfaces be extraordinarily susceptible, response becomes too "diabatic," too immediate, too unreflective;

moreover, what is inhibited is converted at a greater expense—at a greater fatigue. Inhibition and storage, then, though not identical, are mutually implied; the one means the other. The "highly strung" person then is apt at times to put such a strain upon his resources as to impair his nervous system; but this is not his usual character. Usually he spends his life in alternating periods of ardent activity and comparative torpor, and accordingly is not neurasthenic—except for literary gentlemen on the search for adjectives. His unthrift is that being so intensely impressionable, no little of his energy is wasted, as in children, by overflow, by superfluous muscular fuss. In the neurasthenic it is that these apparatus, either in quality or quantity, are idiopathically defective, at whatsoever levels; and inhibitions are very far from being confined to the cerebral convolutions.

Unquestionable as is the evidence of heredity in the currency of this disease, yet it may appear in persons in whom no such disposition is traceable, and from causes definite in time and nature, without any sign of auto-intoxication or other auxiliary. Such cases lie by the score in the memory of every physician of large experience. And as recovery is often, in the simple cases usually, complete, the symptoms cannot depend upon any substantial lesion. The patient constitutionally indigent, whatever the degree of recovery from an attack, will remain indigent; the man naturally well endowed will, in ordinary circumstances, be made whole again. The more the hereditary proclivity the less need be the accessory causes from without; in those born neurasthenic the common stresses of affairs suffice to keep up a frequently recurrent or even a persistent exhaustion. An elderly man, hale as he may be, free as he may be from "auto-intoxications," begins to have glimpses of "neurasthenia"; a heavy day's work, especially of bodily exertion, may leave him languid and snappish for a day or two.

May we not fairly infer, then, that the theory of neurasthenia will be one consistent with these simpler instances; that it will not consist primarily in vascular or nutritive hypoplasia, nor in toxic states, however accessory these may be as aggravating factors; that it will prove to be a "non-specific" state, one to which, on sufficient disturbance, every nervous system is liable, and of itself liable, without co-operation of vice or defect in any other system, humour, or particular part of the body? Most frequently the malady depends upon a disposition of the nervous mechanism, a disposition often inherited, which is or implies lability or incapacity. The proposition that, by some lapse or reversion, however remote in origin, this defect of the nervous mechanism must in all cases of neurasthenia be primary, lies of course outside contradiction. Jendrássik and other authors postulate the "latent neurasthenia," to which I have alluded, for all cases; experiment however, more precise than clinical observation, indicates rather that the limits of fatigability vary widely amongst individuals apparently of normal constitution, so that in this, as in other self-engendered maladies, there is no strict division between health and vulnerability, no marches occupied by latent or larval forms or spectres of disease, but that neurasthenia in the unstable man differs from fatigue in the vigorous man in degree only, in the degree of reserve, of recovering capacity; or, in other words, in its permanency.

Era.—We are too apt to think that a disease is engendered in mankind about the time of the first description of it. But we must not suppose that the clinical series to which the name of neurasthenia is given is in any exclusive sense a product of our own or of recent times. As insanity, epilepsy, and hysteria, so neurasthenia had no doubt its part in all ages; but, unlike the sufferers from those portentous diseases, which obtained somewhat of a sacred character and thus commanded a certain protection, merely neurasthenic persons would be harshly thrust aside, and fail even of a superstitious regard. Were this the place to enter on such an inquiry, it would not be difficult from ancient records to adduce evidence of morbid states resembling neurasthenia, even if we do not go so far as to recognise in the restoration of sexual capacity to Iphiclus by means of a course of iron rust exhibited by Melampus, a rehabilitation of a "sexual neurasthenic." If instead of searching into past time we travel into present space, we shall find not only that the citizens of the United States, in spite of their claims, are no more neurotic than other people, but that neurasthenia, in its pronounced examples, is common enough in the wage-earning and in the rural classes of England; it is frequent in the West Riding, especially, I think, among colliers. Such patients are grievous to the medical officers of Friendly Societies. It occurs also in peoples—Oriental and other—which are at once mentally undeveloped, and yet of sufficient stability to carry the diseases of peace. We read again that it has been a frequent disease in Finland, even in the remote rural districts where the strenuous life has probably been no common peril. That neurasthenia is chiefly fostered by weight or intensity of business, at any rate in any eminent degree, is not proved, however likely this notion may be. The truth is that neurasthenia is found no more in the market-place than in the rectory or in the workhouse; no more in busy citizens than in idle damsels; development of the higher nervous centres makes for control, while, on the other hand, listlessness, frivolity, and adversity are not confined to any rank of life.

Now let us listen to the sober words of Charcot: "The neurasthenic neurosis does not belong exclusively to the men of the privileged classes, softened by culture, worn out by abuse of pleasure, of business, or of intellectual toil. This is a prejudice that time after time I have set myself to combat, but against which I shall have to protest no doubt long enough yet, for it is far indeed from being eradicated." Have the events of later time contradicted these words? Let us see. Formidable as is the list of experts who are now loudly denouncing civilisation in its very essence, in the very development of the nervous system on which it depends and in which it has its being, and are begetting flimsy platitudes about genius and nerves, yet happily these Jeremiahs have it not all their own way. Strümpell in his *Lehrbuch* declares to the contrary, and says that the malady is as common among the poor as among the well-to-do; Martius, and Dercum in America, give a like testimony, not on a priori prepossessions, but on substantial data, not forgetting negative instances, data drawn not from limited circles but from all conditions of men. Von Hösslin, who distrusts modern culture, writes from a sanatorium for the well-to-do; others write from hospitals which the neurasthenic does not much frequent, for his doctor, and his friends for that matter, assure him that there is nothing the matter with him; so, gradually forsaken, he drifts from the chimney-corner into the workhouse, without even the consolation of figuring among the "degenerates." Moreover, it is the clever ones who know how to talk about themselves, only too well; the sons of the soil are shy, stupid, dumb, and do not count. We are learning then that the disease is not a new one invented by Beard, but, as Arndt says, "ein uraltes Leiden." Our ideals of life may well be amended, but do not let us expect therewith to abolish neurasthenia. In one sense, no doubt, our times do increase recorded neurasthenia, in so far as medical facilities, means of public expression, insurance advantages and so forth encourage not the pretender only, but also encourage many persons to make no unreasonable complaints who in less sympathetic times would have held their tongues. Leubuscher and Bibrowitz, who are concerned for one of the large Berlin Sick Insurance Societies, emphasise this kind of accretion among the working classes. Lubenau, on the basis of a like experience, says that among the working men of the great cities neurasthenia is widespread ("ganz ausserordentlich ausgebreitete"). Terrien finds neurasthenia (and hysteria—which in small communities is infectious) among the peasants of La Vendée; and notes in it much the same features as urban neurasthenia (he had practised in both town and country), though on the whole it is less "mental" and is manifest more as back pains, physical fatigue, or dyspepsia. To a recent Royal Commission certain medical inspectors reported that neurasthenia was very common in the poorer districts of Galway. This aspect of the problem is so important that I must not omit the investigations of Karl Petrén of Upsala whose experience of all classes of society, mostly of peasant farmers or fishermen, in valleys and uplands, a sturdy folk not sophisticated by millionaires or motor cars, is very large. He found neurasthenia in plenty, but not predominant in the upper ranks of the people or the "intellectuals." In four or five years he computed from his notes taken from the simple life of quiet provincial Sweden that, of 2480 patients 285 were cases of neurasthenia. These he subdivided as follows:—

Petrén's Table.	Total Number.	Number of Neurasthenics.	Percentages.	
Men of upper classes	226	30	13·3	14·2
Men of middle classes	159	21	13·2	
Men of lower classes	609	90	14·8	
Women of upper classes	302	20	6·6	9·7
Women of middle classes	244	16	6·6	
Women of lower classes	940	108	11·4	
	2480	285		

So that in Sweden at any rate the working classes are slightly more liable than the upper classes, from whom, as Petrén points out, most authors have with some prepossession drawn their materials. In such walks of life Playfair collected his experience, and from his partial data concluded that the malady was one "of the cultivated and educated classes." Petrén says that authors copy this prepossession as a "Glaubensatz" one from another. Host of Helsingfors, whose facts again are derived from simple folk, agrees with Petrén. Martius likewise finds neurasthenia as frequent among peasants and yeomen, wind-millers and wind-milleresses, stone-breakers and mountain folk, clodhoppers and day labourers, as amongst men and women of the great world. He thinks the nervous system is no more strained by travelling 250 miles in five hours on a modern railway than by bumping for five hours between one little town to another in a rustic wain. Among the women the malady is more frequent, as might be expected, in the peasant and labouring classes than in the upper and middle classes in which the women lead a more protected life.

Causation.—Many of the causes of neurasthenia will be implied in the subsequent paragraphs. The interest of the traumatic cases lies in their dependence on a simple series of antecedents; for, "compensation cases" apart, contingent causes, even psychical, may be few or absent. If it be true that in many such persons the nervous reservoirs seem to have been originally shallow in capacity, it is no less true that a mechanical cause, without any mental stress or suggestion whatever, can induce similar phenomena in persons previously of normal nervous capacity. Perhaps in persons thus originally endowed, a dislocation of synapses, or some sluggishness of the ancillary processes of these systems, may become equivalent to defect of capacity. On the other hand, as rest, massage, and high feeding, which in hysteria are of little service, cure a large number of neurasthenic patients, even cases of traumatic origin, if the pathological condition consist in a solution of continuity, the feeding, peace, and other curative means fill up the gaps.

The attribution of abnormal irritability or "over-excitability" to particular nervous parts in disease is absurd. No nervous matter was ever too excitable: to be excitable is its business; it is in "over-irritability" that a race-horse differs from a jackass. The more excitable our nerves the quicker and higher our life. It is by no means clear to me, as I have said, that keen, highly-strung persons are necessarily more liable to neurasthenia than the vulgar man, though they are more apt to overwork themselves; and at particular moments it may be difficult at first to distinguish between neurasthenia and temporary overwork or fag in highly-strung persons. But the method, rate, character, and degree of recovery soon settle the question. Of nervous matter instability is the peculiar virtue; the fault in neurasthenia is that the vibrations of the sense organs, instead of being suspended by the higher controlling nervous masses, and thus absorbed into the larger harmonies of the nervous functions, fall to "short-circuiting," wherein their energy is wastefully dissipated. Many neurasthenics are quite dull people; the clever ones we admire and lament, the humbler we forget.

In Petrén's tables 62 patients attributed the malady to family troubles; 24 to financial worries; 47 to "overwork"; 29 to the effects of antecedent illness; 21 to various sexual causes.

Heredity.—We have seen that by natural endowments individuals differ widely in reserve, in potential. Some are seaworthy even in storm; some pass a fair voyage of life continually in peril of wreck, but in safety; some who go under seem so frail or ill found that an original frailty is presumed. Nearly all physicians are agreed that such a defect is, often at least, primary and inherited. Löwenfeld estimated congenital defect in four-fifths of his cases; yet he thinks not all are hereditary. Dr. Herman says an unhappy marriage would usually tell for more than a neurotic heredity. This question of degree, I think, I may leave where it is without accumulation of surmises, however authoritative. But I would warn the observer against the too facile assumption of heredity which is current among us. "His aunt died in an asylum"; "her cousin had fits!" what family is there without such poor relations in the background, whether subjects of nervous disease or otherwise, of arteriosclerosis, syphilis, myxoedema, and so forth? It is said that the children of elderly parents are apt to be of low potential; if so, the exceptions are many. Hospital patients know little of their kin, superior persons do not choose to tell.

To attach neurasthenia to particular *callings* is not easy—for instance in his figures Jendrássik found it rife in the scholastic profession; but further consideration shewed that persons would adopt this calling "because they were not very strong." Dr. Michell Clarke in his interesting little book says the malady is not drawn from all classes and from all occupations, but is essentially a disease of those who earn their living by brain work. This opinion, as we have seen, is the prepossession of consultants occupied with the middle and upper classes of society.

Bodily, mental, or emotional shock or strain may produce neurasthenia with the suddenness of a traumatic cause—the "psychical trauma" of current jargon. Young persons fagged by long mental application, by prolonged muscular exercise performed for a time perhaps without a sense of undue fatigue, or, again, by the continual harass of ill fortune, may shew no patent effect of the kind we are contemplating until the condition reveals itself all at once. Dr. Acland has warned us of the dangers of too little sleep, especially in young persons, but not in them only. Or the effects may come about gradually and almost unperceived. If then neurasthenia, often of an obstinate kind, may set in suddenly without any obvious cause, yet in many of these cases there is a strong family bias towards nervous disease. "Ne devient pas neurasthénique qui veut," as Charcot was wont to say.

Sex.—Women, if on the whole less subject to the malady than men, are more prone to break down suddenly; some women never quite get over a long and ambitious term of study, or too long a course of athletic exercises, although the stress was borne for a long time with apparent ease. Those of us whose practice lies in schools and universities have seen too often such breakdowns occurring explosively, and then obstinately resisting the remedial means of nature and the physician. A man under urgency is apt to turn dunce, and thus spares himself; the more docile woman will let herself be exploited till she drops. Such an one, after living a full life in the eye of the world, may disappear all at once into the twilight of invalidism. For the volume of energy may be lower than momentum in work, and if this be not appreciated, exhaustion may be sudden and profound. As a rule athletes are a picked lot—the weaklings take to golf or lawn tennis; yet very strong men may thus be broken down. A friend of my own told me that he was once persuaded, when he had gone out of training, to take a vacant place in his college boat in the May races. Each day's race was a hard one, and the stress so told upon him that since that time, though his general health is good, he has never been able to withstand any hard mental or bodily work. Whatever the heralds of degeneration may say, the bodily frame may give little indication of neurasthenic possibilities. Before the breakdown not a few such patients are of fine physique. It is not easy for the physician to be sure when emulation, vivacity, and self-devotion are consistent with a solid and quickening development and a steady maintenance of the precious structure and functions of nervous matter, or how far the demand is trenching upon exhaustion. The eye may have the brightness of teeming life or the false gleam of failing control. Yet an error in our calculation is almost irremediable, for even when cure is accomplished the convalescent may have lost his youth or his first openings in life.

Toxic Causes.—Under this head we must discriminate between cases of adherent poison, as of lead, influenza, and so forth, and cases of impairment after the poison is dispersed. Those cases, at any rate, are not neurasthenia; and even these, unless the poison has unmasked a neurasthenic habit, are no other than ordinary convalescents. Thus "alcoholism" is not neurasthenia; but the physicians of the Northern races suspect that drunkenness in the parents and ancestry conduces to neurasthenia in the descendants. Still, as they say, drunkenness in cold countries is so widespread a habit that it is difficult to draw any sure conclusion.

Age.—Does neurasthenia proper occur in children (p. 736)? This is a hard question to answer. Children lack endurance, but are very rapid in replenishment. A child between ten and fourteen, or younger, loses interest in games, is tired and inattentive, moody, whimsical, and irritable; he sleeps badly, or too heavily with dreams and terrors; the hands are cold, and the pupils dilated; the face twitches, the appetite is capricious. I need not labour upon the too common picture. In most of these cases faults of organic function are conspicuous; the tongue is coated, the abdomen podgy, the urine scanty and thick, the motions offensive and irregular. The temperature rises occasionally. Doses of calomel benefit these children greatly, and a scrupulously defined plain diet, excluding tea, coffee, and sweets, has its reward. Some of them are overgrown; many precocious and "pathologically pious" children are suffering only from foolish parents or nurses; a few, perhaps, are truly neurasthenic—that is, they are short of storage capacity, but others are defective in quality rather, are just feeble-minded, lethargic, dull. Bad teeth, worms, phimosis, eye-strain must not be forgotten. With the "degenerate" it is not so much a matter of economy of energy as of arrest of development. Frequently in clever youths, often in the middle or later "teens," a curious phase of stupidity intervenes, to the vexation of disappointed parents. For a time intellectual and ethical promise sinks out of sight, but in two or three years the faculties brighten again, as if for the development of the body, or in self-protection against over-pressure, they had retired into a chrysalis state. As Horace Walpole said of certain philosophers, "they may be growing wise but the intermediate stage is dulness." To explain this to a parent may prevent much family anxiety.

Symptoms.—Neurasthenia is, then, no mere hotchpot—no limbo into which odds and ends of unconsidered neurotic trifles are to be thrust away for lack of definite affinities; nor is it a sham, nor a figment of complacent physicians. Yet the varieties of the disease, and the wheels within wheels, are so many, and in apparent diversity so bewildering, that the essential unity of the process long lay hidden. Moreover, its particulars are apt to gather in sub-groups, and to form fairly consistent and uniform series subordinate to the larger type; but the nature of these several lesser and subordinate constellations of symptoms has been worked out by an analysis and a training in exact observation which were scarcely at the disposal of our forefathers in physic. It were scarcely to be expected that the clouds of neurasthenia should always gather over the whole body alike and at once; and they do not. The depletion of potential energy is various in its centres, sub-centres, directions, and degrees; now this region is in low pressure, now that. The nature and significance being uniform, the manifestations break forth here or there according to the seat of inward frailty or the incidence of the external causes in the particular case. Thus, though the therapeutical principles are consistent, their application is various.

Again, if in a part or organ functional disorder has run so high as to have brought about some perversion, or even static change, then on the maxim of the "vicious circle" lately emphasised by Dr. Hurry, recovery may be obstructed until the local trouble is mitigated or removed; and this to such a degree that the general principles of treatment of the malady, as a whole, may have to be set aside temporarily for local remedies. Now it is found by experience that in neurasthenia depressions and storms may prevail in certain areas, and in specifying these we may err on the side of subtlety or on the side of breadth; the following divisions, or perhaps I should rather say points of view, are, I think, of practical service:—A, Cerebral; B, Spinal; C, Cardio-vascular; D, Visceral; E, Urogenital; F, Traumatic. Like neurasthenia itself these subdivisions are far from being water-tight compartments, and have general symptoms in common; yet they serve to distinguish the various aspects of neurasthenia, and, often confused, as in practical experience they are, one with the other, to aid in their recognition. As E. Müller well says, the local symptoms are not the neurasthenia, but they give the colour to the case. For descriptive purposes, then, these divisions will serve us well enough. In their large sanatorium experience Leubuscher and Bibrowitz, who use these divisions, report that their cerebral cases were 31·6 per cent, their cerebrospinal 48·3 per cent, their gastric and cardiac 6·2 per cent and 4·2 per cent respectively. These patients were, however, of the working classes in Berlin. (The remaining cases were traumatic and convalescent.)

A. *The Cerebral Form.*—By "cerebral" I would indicate particularly the functions we attribute to the cortex—the functions of mind. This form of the malady may and often does exist alone, the patient being corporeally robust, active, or even athletic. One of my patients, a man of neurotic family history, who suffered from the cerebral form of neurasthenia, could tire out most of his friends in hunting and shooting. One of the most characteristic features of cerebral neurasthenia is the "weary brain." The sensation is familiar enough to any fagged man, especially if he fall short of sleep. Impressions seem to go half into one's head, and there to sink into a woolly bed and die. Voices sound far off; the lines of a book run one into another, and the meaning of them passes unperceived, or, if for a moment perceived, is dissipated. Doors bang and windows rattle as they never did before; if but a shoe-string break an imprecation is upon the lips; and to the fretful skin the touch of the gentlest hand may be torture. Business matters are in a conspiracy to go wrong; letters are left unopened, partly from want of will, partly from a senseless dread lest they contain worries or ill news. Let us suppose that the poor mortal thus exhausted, instead of sinking into slumber, tosses on a thorny bed beset by all the cares which blacken with the darkness, and lies there restless, or paces his bedroom, till dawn, when some fitful filmy slumber may bring a truce rather than recreation, and the weary day has to be faced anew with no renewal of strength. Now this man is on the brink of neurasthenia, or in it. Either he is undone by some exorbitant mental stress, in which case there is still much hope for him, or he comes of a neurotic family and is entering on his inheritance.

Headache, in its more local and definite forms, is of course consistent with neurasthenia and frequent in it, but is not very characteristic of it. Some of these sufferers complain of a never-ending diffused headache, of which, however, they may cease to complain, unless questioned about it; it is often occipital and not acute enough to assert itself prominently. Still, it is characteristic of neurasthenic pains that the patient emphasises them more at one time than at another; and the times of acuter complaint are not uncommonly marked by vasomotor impressions, acceleration of pulse, cutaneous hyperaesthesia, sweats, or even contracted brow. Such "neuralgias" have been called "local obsessions." "Oh, I always have a headache," is the reply. Or complaint is rather made of strange sensations in the head; it feels leaden or wooden, empty or full; there is a heavy weight upon it, or a tight band or cap about it (casque neurasthénique); or it is too light, it opens like a lid, it hums, or trickles, or little explosions take place in it. When headache is persistent, defects of refraction may be found; yet often such search is vain, and by the aching, probably with some insomnia, the patient, who may or may not be vigorous enough otherwise, is disabled for bookwork. Although as a factor in neurasthenia, and even in migraine, "eye-strain" has been overestimated, yet it is a *vera causa*, and in not a few cases it is the efficient and sufficient cause, especially of the cerebral form, though it is often adequate indeed to exhaust the energies of the whole body, so that before commencing the cure of a case expert investigation of the eyesight is essential. Most frequently hypermetropia, often with astigmatism, is discovered, and after the adaptation of glasses some weeks may elapse before the tormented eye comes to a position of rest, and the cause of the disturbance is abated. Furthermore, under neurasthenia in a normal eye the muscle of accommodation may fatigue like any other muscle. A few hours with a catalogue in a picture gallery will prove this even to the healthy man. It is said that small astigmatisms, such as ¼ diopter, are negligible. No such fraction can be so regarded apart from the patient and his state at the time; the "bottom fact" is the receptive instability. Vertigo may suggest eye or ear disorder. The cavities of and about the nose and the ears must be observed, especially if there be pains in the head or face. The pupils are often dilated; I am scarcely ready to admit that "they may be unequal." Tenderness of the scalp is often present, and not in cases of headache only. I have remarked (p. 732) that the occipital headache is often muscular—a strain on the occipital aponeuroses—and that the simple device of placing a pad at the back of the chair to support the head often relieves it. Headache or confusion on stooping alarms some persons who happen to be temporarily out of tone.

Certain psychical symptoms are characteristic of the malady; and, although they vary in different cases, they preserve much consistency, differing in degree rather than in quality. Of these features loss of memory is eminent, sometimes distressing; in the severer cases it is wider and deeper than mere inattention can explain. In the matter of extent it differs from the losses of memory in hysteria: in neurasthenia the loss of memory, indefinite as it may be, is general; but in hysterical loss of memory a certain interval of time is often cut out as with a knife, whilst things before and after may be remembered well enough; or the defect is definite and particular, not general. Fatigation, the mark of all neurasthenia, may be manifest chiefly in effort of memory, as it may be in the painful lassitude of the hands and arms of a pianist, in the back and legs of a labourer, in the orbital muscles of a miner, and so on. In neurasthenia the loss of memory is due in part to the "malady of not marking withal"; the patient has lost interest in life, is self-centred; and not only so but also inattentive from sheer lack of power of application, of mental attack and endurance. On the first effort of thought the brain flags, and is felt to flag—"it goes empty." With these defects are bound up the loss of energy, of self-control, and of determination so manifest in these cases; it is, indeed, but a different aspect of the same mental failure. It is often difficult, for instance, or indeed impossible, to rouse the patient to the thought and decisions necessary to the treatment of his own case; he cannot face the business arrangements, he cannot set himself to pack his portmanteau. But in others loss of control is seen in the consuming haste with which ideas must be realised in action; the patient may suddenly conceive the idea of seeing some person who lives many miles away, and not rest until he has sought out his friend, often for no very important purpose; or some project or purchase must be carried out instantly, although indeed of no urgency. And when these hasty schemes miscarry he is put about. This ineffectual state is commonly associated with irritable temper, tremor, and vague fears.

In cerebral neurasthenia patients vary in manner and temper. I may hint at three or four types of them: first, the man of healthy, even robust frame, and well-coloured face and lips, one who, but for a certain characteristic look of preoccupation and moodiness, might pass for healthy and vigorous; secondly, the voluble, restless haunter of consulting-rooms, the connoisseur and too often "disillusioned" critic of specialists, "l'homme à petits papiers" of Charcot, spare of frame and sallow of face, petulant rather than sulky, and concealing under a mask of energy and promptitude the same irresolution; thirdly, the man who faces his work and does it valiantly, but is subject, many times a day it may be, to periods of almost ghastly prostration, he sinks into a chair, his eyelids droop, his thoughts evaporate, his utterance flags, and he may break out into a dripping sweat; yet the face does not become very pale, nor does the pulse alter much; and, fourthly, the silent patient, rather blue than sallow, with limp and clammy hand, always brought more or less against his will by doctor or friend, miserable or morose, shifty or mulish rather than unstable in temper, sometimes reticent and shrinking, but sometimes possessed by secret or unreasonable resentments; this last patient may drift into melancholia as the second may end in "paranoia." Indeed, on revision of this article, I do not feel sure that this last condition will prove to belong to neurasthenia. Patients of the three last kinds are usually under weight, but scarcely perhaps to be called emaciated unless food has been fitfully or persistently refused ("anorexia nervosa"), in which case the sufferer, sallow, hollow-eyed, and with dried-up hair, may look like one raised from the dead; such cases occur rather in women. Neurasthenic patients are sometimes, though rarely, suicidal, especially those after the fourth type; for the most part the "taedium vitae" stops short of this depth of despair. Or the soured, pertinaciously argumentative patient may do away with himself in a sort of malicious freak. Women never, I think, commit suicide out of mere neurasthenia.

Although the dangers of insanity in neurasthenia must be indicated, the transition is not one of ordinary anticipation. The insane person is only too often indefatigable. However, insanity and other nervous diseases are frequent in the families of these patients; and in the later years of life they themselves may lapse almost insensibly into incurable delusion or settled melancholy.

Obsessions.—Certain mental perversions, not unhinging the mind as overt insanity, are prone to occur in neurasthenia, are even significant of it: these are peculiar dreads and oppressions which do not tincture the whole being as insanity does; they are more or less arguable, more or less kept in their place by the judgment of the sufferer, who does not lose all sense of relative values. Perhaps no considerable case of neurasthenia is wholly without them. Indeed, we are all of us subject to small tyrannies of whim and observance; we have our little rules, our little hoards, our little prides. And when we flag, these get more hold of us. Obsessions are a sort of short-circuiting; certain ideas, as Prof. Sherrington would express it, get possession of the limited number of public efferent paths. Bunyan, when preaching, was liable occasionally to a temptation to speak blasphemous words, probably when he was tired. But such obsessions occupy but a part of the mental life, and, unlike insanity, do not colour or pervert the ordinary avocations. Human nature is hard reading; a man may seem to mope and to yield to melancholy only because to mix with the world brings home to him the narrowness of the limits within which, at his own pace, he can get along. "Attempted explanations from a man apparently fit, who hitherto had led an athletic life, seem so absurd that I prefer my own society to having to make them, and get looked upon as a crank," is the story of one of such sufferers. The graver dreads, obsessions, besetting thoughts, impulses, or anxieties, have their origin within, and are not reflections of any sufficient external events. Such are false apprehensions of evil, fears of fears, recurring besetting impulses; or, again, peculiar bodily oppressions or disturbances—cardiac, respiratory, pelvic, and so forth—which have not the fixity, and may have some more real organic substratum than the possessions of hypochondriasis; and they are more remediable by ordinary medicine. Mr. Dent noted in some cases that an obsession was the first definite symptom to appear. But of the obsessions which seem to take their origin in the viscera I shall speak in other paragraphs. Amongst those which appear to have a psychical origin, I have already spoken of the sense of brooding evil; and although a dread of formless evil cannot well be described, it is none the less harrowing in that it stretches forth from the unknown. If these vague and illusory fears should stiffen into delusion the case would pass from the sphere of neurasthenia into that of insanity (paranoia); but this is not the usual course. "Paranoia" may begin with a simulation of neurasthenia, but here the ambiguity is one of diagnosis. Some of the besetting apprehensions of neurasthenia, however, come perilously near insanity, especially the dread of committing crime. A fear of doing some wrong returns until it possesses the mind, the besetting thought looms larger and larger until it assumes the garb of a temptation; but the very large majority keep on the hither line of insanity and are thus preserved: still there may be a streak of insanity in the patient's family, and no responsibility of the physician and counsellor is graver or more difficult than his course in such aberrant cases. For instance, a young lady, devotedly attached to an invalid father, dwells on the presages of death, begins to doubt if her strength will endure as long as his sufferings, and to fear that to him his life is rather a curse than a blessing; then perhaps she wakes suddenly in the night, as from a dream, and, solitary and overwrought as she is, shudders to think she has wished for his death. Not daring to betray her horrible sin, she lies under its spell, drawn nearer by its fascination, until at last she accuses herself of a temptation to poison her father; and the poor torn little soul is taken to a physician who has to decide whether all this passion be the temporary and factitious result of stress and solitude, or the grim reality of a homicidal impulse. The physician may have a definite yet not infallible opinion; and while he is quietly extricating her from her bondage cannot avoid an anxious doubt whether the case be indeed one of mere neurasthenia, or of insanity in its most ghastly form. Another very distressing kind of burden is remorse. Remorse is at best but a wasteful passion; repentance bears sin forward into hope—remittimus irremissibile. The neurasthenic who can lift his eyes to the future will recover; he whose thoughts writhe in the past is probably marked down rather for melancholia than for neurasthenia.

Less degrees of obsession are seen in compulsory counting, in phrase or tune reiteration, or again in the petty doubts which beset a man as to whether or not he had wound up his watch, locked his outer door, or omitted to touch a certain post in Fleet Street (*vide* p. 628). These obsessions, however seemingly trivial, often occur without neurasthenia, and may lie nearer to the confines of insanity. Hand-washings, suspiciousness, syphilophobia, voices—these are not characteristic of neurasthenia, but occur in masturbators, or in persons of fair bodily health marked down for insanity. German authors say that the examination bogy embodies a dread which to the overworked and anxious student becomes an obsession; so that the conclusion of the curriculum may be "Ende mit Schrecken," or still worse, "Schrecken ohne Ende."

I must mention two obsessions in particular which are characteristic of neurasthenia, and indeed pertain rather to it than to insanity; namely, "*agoraphobia*" and "*claustrophobia.*" I am not concerned to defend these names; they are accepted, and that which they signify is familiar enough to those who see much of neurasthenia. Claustrophobia is more of an obsession: of agoraphobia the patient's imagination is usually free until, as a wide space opens before his eyes, the dread falls upon him; but of claustrophobia he is always more or less conscious; perhaps its conditions are more continually about him. The dread of being shut up in church, in a theatre, in any closed apartment, even in a friend's drawing-room—especially if full of people—may be intolerable. Some of the obsessions, as I have said, are associated with visceral sensations of a more or less oppressive kind; thus claustrophobia is apt to formulate itself as an oppression at the chest, a panting, or a heart constraint, or a disposition to pass water or motion. Indeed, the obsession usually dates from a real experience of the kind. One patient of mine, who is subject to attacks of violent trembling or shuddering when he finds himself so placed that he could not retire, is so beset by the fear of an attack as to bring on the very discomfort which he dreads. But it is often independent of visceral sensation, a sort of panic, a notion of a flight which might be impeded. Delusions of persecution or hallucinations belong to insanity and are consistent with good bodily health.

Agoraphobia, on the contrary, is a fear of empty space as such. The "giddiness" which possesses some big strong men in high places, or on ladders and the like, is peculiar, and not to be confounded with vertigo or with ordinary "funk." It is incomprehensible even to a timid climber who is free from it. Now agoraphobia, like claustrophobia, is common in neurasthenia, and is, I repeat, a mark of this malady rather than of insanity. A young athletic schoolmaster, suffering from cerebral neurasthenia and sleeplessness, was bewildered and ashamed to discover that he dare not, with whatsoever self-command, cross the market-place of his little town. But agoraphobia is not only a matter of market-places, but also of any unhelpful space—of a space, that is, in which there are no easy posts for the eye. A neurasthenic lady, to whom, during convalescence, I had recommended the bicycle, came to me almost with tears in her eyes, for she had quickly and gladly taken to the exercise, and found great benefit in it, saying that when she turned into a spacious and unoccupied road she was seized with panic, although in narrow or winding lanes, or in broad thoroughfares full of traffic, she was at ease. A sturdy Yorkshire clothier (one of our railway cases) was amazed to find that, after his accident and while suffering from insomnia and other consequences, he could not face the broad road from his house to his office, unless indeed when blocked with traffic. He would turn into a cottage, pretend fatigue, and send for a cab; so that for a time he was constrained to go about his work in a carriage. In consultation with skilful oculists I have often tried to find an explanation of this dread in some disorder of adaptation of the ocular muscles, but hitherto without definite success; still the defect seems to lie in some lack of grasp of space relations, for the touch of a child's hand will often suffice to carry the patient easily forward. Some neurasthenics have alternating obsessions, and during the dominion of either marvel at their subjugation by the other.

The *vertigo* of neurasthenia is quite independent of agoraphobia. It is a very characteristic and common symptom, and much good may be done simply by reassuring the patient who conceives it to mean some grave cerebral peril. Although often associated with tinnitus, and sometimes with detonating noises, especially in worried or overtaxed persons, it is not to be confounded with aural vertigo, there is no kinship between the two affections; the neurasthenic kind is generally independent of any aural symptom, indeed it is not the lurch or gyration of aural disease, but a lightness of the head with a sense of failure of the legs. There is not the reeling gait of cerebellar disorder. It is never very severe, never so severe as in the graver aural kind, and is usually of brief duration. It rather resembles cigarette vertigo. Like other kinds of vertigo it is apt to occur on rising from a chair, stooping to tie a shoe, or other rapid or awkward change of position. It may be a throb of atonic arteries; it has nothing to do with vision.

Insomnia, in its degrees from cat-sleep to "nuits blanches," is a frequent but not a universal feature of cerebral neurasthenia; it is generally one of the links of the vicious circle in which the victim is enchained. But some patients sleep in the heavy unrefreshing way which may precede a megrim. "I sleep, and even doze wearily and late, but I am none the better for it," is no uncommon complaint. A patient whom I have just now seen, who suffers from distressing prostrations, with sweats and vasomotor oscillations, told me he was sleeping well enough. Some on dozing off are startled by a palpitation, some have nightmare, especially of physical helplessness in some peril. Those who sleep in the early night and those who sleep late need some difference of treatment.

With the insomnia is too often the torture of acute hearing ("hyperacusis"). The hater of cocks is a neurasthenic. In spite of cotton-wool or even of wax in the ears, in spite of double windows and thick nightcaps, the accursed sounds, even of a sleeping world, drive the sufferer to distraction. It has happened to me on more than one occasion to be called to the fortress of one of these neurasthenics, for whose unattainable peace a hushed family circle had boarded up the windows, stretched cords across the thickly carpeted passages, awed the children into dreadful silence, tied up the door bell, and banished dog and fowl from the parish. The doctor is implored by the vigilant wife or daughter to enter the room, if enter he must, with velvet foot, and to speak with the still voice that guides were wont to use under the séracs of the Géant. In his turn the patient beckons you to his lips, and confides his tale to you in a feeble and broken whisper. This is not only for fear of noise; in neurasthenia a feebleness of the voice is another characteristic symptom: it enfeebles rapidly, and will drop into undertones.

Boarding up the windows, no unusual freak of these patients, is not done for the ear only; they often shrink as much from light (photophobia) as from sound. Both perception and accommodation soon cause fatigue. As he reads the patient complains that the lines, even of a well-printed book, run into each other; although careful search into optical conditions reveals no abnormality, or none which is not already counteracted. Möbius has pointed out that if the patient be told to converge upon a near object the convergence is quickly exhausted. During the test for Romberg's sign the patient does not, as in ataxy, shut the eye firmly, but he half shuts the eyes, blinks, and opens them again. The pupils, in my experience, are sluggish or disposed to dilate, but otherwise normal; some authors have recorded inequalities of the pupils, a sign possibly of transient importance, but surely not one to be readily accepted as such; it might well, for instance, be an early indication of general paralysis of the insane.

The sensori-motor functions and capacities of the cortex may also be affected; if so, usually with paraesthesia (unreceptive surfaces).

Such are the features of cerebral neurasthenia; we will now pass on to the other subdivisions of the disease.

B. *The Spinal Form.*—The chief features here are weariness of the legs and pains in the back; it is these patients especially who are weak and good for nothing in body. The pains in the back are a dull, diffused ache, or an acuter pain with tenderness, especially at certain spots; namely, in the seventh cervical or upper dorsal spine, between the shoulders, and across the pelvis, the tenderness then being in the lower lumbar spine—Charcot's plaque sacrée—or in the sacro-iliac synchondroses; this site, I think, is more frequent in traumatic cases, but usually without any apparent connexion with the seat of the injury. I have argued (p. 733) that this weary ache is not a chemical irritation of the receptive end-organs of the muscles, but a drag upon them by sagging of their atonic masses, and by stress of body weight upon ligamentous and aponeurotic strands. The coccyx, again, in these patients, especially in women, is apt to be the seat of acute and stubborn tenderness.

Under this subdivision are to be described the various perverted sensations of which these patients complain—of feelings in the legs, such as numbness, tingling, formication, and remarkably of coldness, even of icy coldness, as if water were poured or trickled down the back, or as if the legs were standing in cold water. The limbs die or "go to sleep" very readily, when overlaid or otherwise pressed upon. Even the tongue is sometimes the seat of numb or strange sensations, whereby arise fears of cancer and so forth. The gait may be, and often is, slow, feeble, and creeping, but unless in the hemiplegic form or in association with hysteria, is never paralytic or in any definite sense ataxic; this I venture to say, well knowing that such symptoms have been attributed to neurasthenia.

I have never seen in pure neurasthenia the definite and often latent anaesthesia of hysteria in tracts or patches. Petrén submitted 16 typical neurasthenics to a minute examination in this respect. He used the pinhead and the double points. In 12 the results were negative; in 4 there was hyperaesthesia about the epigastrium (3 towards the left, 1 towards the right), in 3 of the 4 others hyperaesthetic spots were present, in the form of Head's girdle zone.

In neurasthenia the "reflexes" are never absent, on the contrary both superficial and deep are generally in well-marked excess; the knee-tap may make the whole body bounce; wrist-taps generally produce quick responses; a triceps and a supinator reflex are often very ready. The toe bend is always flexor, but in doubtful cases *both* feet must be tested. A transient ankle-clonus, which appears only to vanish, may be obtained in many healthy persons. If it pass beyond, let us say, three vibrations of small excursion we must suspect organic disease. In hysteria the rule is that these reflexes, especially the cutaneous, are abated. Dr. Risien Russell says in some few cases of neurasthenia the reflexes are diminished, and that enhanced reflexes in hysteria are not unknown. In hysteria Dr. Thomas Buzzard's negative plantar test and faucial insensibility are common but not unvariable. In a case of hysterical paresis recently seen the reflexes were excessive in the forearms, knees, and soles.

The cerebrospinal form, common as it is, needs no formal description, being a compound of some or many of the symptoms already described as cerebral and spinal neurasthenia respectively.

C. *The Cardio-Vascular Form.*—In this form the heart's action, on mental or bodily movement, is usually accelerated, or at any rate very easily and continuously quickened. Very rarely is it slower than normal. The patient is too conscious of its action and of the pulsation of the arteries, even of the small vessels of the extremities; although to the observer the impulse may seem feeble and the sounds short. The submammary area is often hyperaesthetic. In this form of neurasthenia, as contrasted with hysteria, the objective cardiac disorders and the patient's complaints are more in accordance. The pulses of the chest and neck may be as visible to the physician as they are perturbing to the patient, who cannot get rid of the apprehension of heart disease. In some cases the arteries are evidently relaxed, and pulsation is visible in the episternal notch, in the carotids, and in the abdominal aorta; or they may be relaxed and constricted in turn or in parts; the ears may glow, and blushes of the face, neck, or bosom, often in separate patches of sharp contour, may be conspicuous; or the extremities may be the seat of these instabilities, as "local syncope" and the like; and the urine may run short, or flow in profusion. Sometimes vasomotor disturbance is seen in profuse sweats, either by night or on the least exertion or excitement. These sweats, often on cold hands and feet, are difficult to get rid of. "Dermatography" may not infrequently be obtained. It is difficult to conceive of exhaustion in respect of the vasomotor system, and we may attribute its vagaries to exhaustion of cortical control. But these phenomena are not by any means confined to neurasthenics. In some cases there is also great instability of the vascular compensations for gravity; thus, in a young man of about thirty years of age with a sense of great exhaustion, pallor of face, and general debility, I found the pulse when he was standing to be 116 and thereabout; but on ordering the patient to recline at full length, the pulse, after three or four well-marked oscillations, dropped decisively to 76. This observation was frequently repeated. After a long and beneficial sea-voyage the patient was put through the same tests with normal results. To make of submammary pain, sense of apnoea, and irregular palpitation with extra-systoles,—a common disturbance in many maladies—a symptomatic sub-group under the name of "phrenocardia," and coarsely to attribute it to sexual lust, seems to me to be very perverse clinics.

I have alluded to a remarkable variety of vascular disorder, often hereditary, in which the patient becomes cold and torpid; the hands, the feet, the nose, and ears are cold and blue, or "dead." He is benumbed, dull, lethargic, almost reptilian; though alternatively low-spirited and fretful. The pulse is usually accelerated but impalpable; the temperatures are subnormal, and the bowels also torpid. In some cases there is air hunger. Such persons are liable to the vaso-vagal attacks of Sir W. Gowers, when the pupils dilate, the pulse vanishes, and faintness, or restlessness with slight delirium or bewilderment, but not complete loss of consciousness may set in. Such attacks may last even a quarter of an hour, but they are not dangerous to life. In some of these cases when we are told that the heart stops, or is "gone," the finger on the pulse may detect no change; at other times or in other persons I have felt the radial to contract to half its volume, and as the attack passes off to expand again. Speaking generally, the blood-pressure is normal. Although it is convenient to describe these cases here, I have expressed a doubt if they are of the nature of neurasthenia. Thyroid extract or calcium is of little or no avail in them; but if the patient can be sent for treatment into a warm dry upland climate the condition is not very incurable. In many such cases we are tempted to jump to the conclusion that they depend upon splanchnic dilatation, active or passive. In part it may be so, but the prime source of the trouble must lie deeper; recumbency brings no definite alleviation, and heavy pressure on the abdomen is ill tolerated.

In neurasthenia in its uncomplicated forms there are no cardiac murmurs, haemic or other. Palpitation is of course very common, and often awakens the patient from sleep, or prevents sleep; it may shake the whole body, and even the bed; and oppression of breathing—"suffocation"—may amount to orthopnoea. Unfortunately it is very apt to appear on retiring to bed. Great irregularities or intermittencies, or both—"a turning over"—are also far from uncommon; though any contingent cause, such as tea or tobacco, should be carefully considered, or an incidental disorder such as dyspepsia or gout. It is too often forgotten, that there are many "nervous hearts" without neurasthenia (*vide* "Functional Diseases of the Heart," Vol. VI. p. 509). The arterial blood-pressure is nearly always about normal; if above it some other disorder or disease is to be suspected. At the same time, I find that impressions, apparently slight, often produce in these patients a "nervousness" which sends up the readings to enormous heights—even to 200 mm.; indeed it may thus be impossible to get a trustworthy record, when the physician must be guided by other evidence. In the sphygmographic curves the respiratory waves are usually larger.

Some attention has been given of late years to "movable heart" in neurasthenia (Vol. VI. p. 508). The apex-beat in normal persons on a full turn over to the left side will vary about an inch. In neurasthenics this point may fall away to the anterior axillary line or beyond—say 6 7 cm. This abnormal mobility may be attributable in part to slackness of the thoracic aorta, in part to pericardial and abdominal emaciation, or on occasion to diaphragmatic movements; but Dr. Christopher Addison also points out that in neurasthenia, the elevator muscles of the thorax being in atony, the first ribs and cervical fascia and attachments of the pericardium sink, and "the normally taut organ" becomes pendulous. The attacks unhappily called "pseudangina pectoris" (Vol. VI. p. 161) are apt to occur in neurasthenic as in other states of nervous perturbation. I have never seen a case in which the diagnosis between these storms and angina pectoris was long in doubt. In the false cases there are such signs as cardiac accelerations and irregularities independent of bodily exertion, submammary hyperaesthesia, windy abdomen, and so forth. The guess of Nothnagel and others at spasm of the coronary arteries in these cases need not alarm us; the blood-pressure does not fall.

The panting spurious asthma, too often attributed to "weak heart," is more common in hysteria than in neurasthenia.

D. *The Visceral Form.—Visceroptosis.*—From the date of my Goulstonian Lectures (1884) the problems of visceral pain have occupied my attention, of late more especially as regards angina pectoris. And I may venture to remind the reader of my contention, since supported by Lennander's and Hugh Anderson's observations, that visceral pain lies not in the parenchyma of a viscus—be it glandular, muscular, or other—but in its investment. It is by dragging on the mesentery, on the pelvic strands, on the pleura, on the pericardium, on the investment of the thoracic aorta, and so on, that pain arises. To elicit it, there is in chronic cases a summation of stimuli, a sum in inverse proportion to the susceptibility of the patient; so that a slight mesenteric or other drag, imperceptible to a person whose centres are in equipoise, may become agony to one in whom such inhibitory integration is shattered. And on some shift of the balance of inhibitory energies, however slight, as in abeyance of counteracting psychical centres, the balance of functions may lie awry till some rousing influence on the counteracting centres —hope, distraction, and so forth—re-establishes the normal adjustment of the reciprocating parts, when, even in some continuance of the stress, its mastery over the "mind" will be disputed. Thus in not a few cases a surgical operation mechanically effecting nothing, by throwing back the psychical activities and determination of blood towards a more normal equilibrium, banishes the notion of pain. A very lopsided balance of reciprocating centres constitutes hysteria; but minor degrees of eccentricity prevail among us all. He is a happy man whose faculties and tempers all quire in spherical harmony. It is astonishing, as cases of cure of the bedridden, or of religious conversions, illustrate, how long centres thus closed to reception, in abeyance by some suspense of animation, can almost instantaneously be thrown back into gear, when a more integral position of equilibrium, what one may call organic common sense, is re-established and maintained. From day to day in minor degrees—as the saints have known but too well—our own mental counterpoint is wont to range now on higher now on lower harmonies. We cannot tell what act, for better or worse, may shake us from one position of balance to another, from centre to metacentre or conversely. For hysteria is rotation about a metacentre, a list to starboard or port; but neurasthenia is the lack of a breeze.

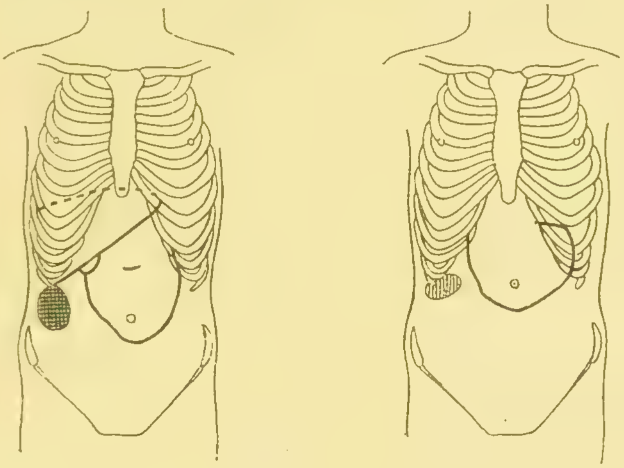

FIG. 25.—Nephroptosis in Neurasthenia.

Now on these principles, which have their application especially in the sphere of treatment, we may interpret the symptoms of visceroptosis; but no doubt, if the fall and drag of parts be great, it will perturb the sturdiest of dispositions. In neurasthenics a local ill, acting and reacting thus, establishes a short circuit and a vicious circle; and the local error must at once be readjusted. In many cases, then, the slackness is at once the cause and the effect of the general malady; it is the weakly fibre which yields; in others by some mischance the bonds of a sound part are stretched so forcibly asunder that surgical repair is indispensable. With these cases we have not here to deal (*vide* "Visceroptosis," Vol. III. p. 860), with those we have some concern. The figures given on p. 755 are from two of the stages of a neurasthenic lady aged thirty-nine, in whom, during the early state of laxity, the right kidney slipped down as there delineated. The second diagram was taken on her recovery after a Weir Mitchell cure by Symons Eccles. In this case the organ had dropped; but in most neurasthenics, although the parts may not be taut, the laxity does not amount to a fall, but yet may swing so far as in sensitive persons to induce vague discomforts. In these the joints likewise—the ankles, for instance—may be slack and "weak."

Of the bearing of pelvic deviations or disorders, or alleged disorders, on the general and mental health of women, the views I urged in 1884 have become, I believe I may say, the accepted doctrines of gynaecologists. The time is past when, as in a story of the late Dr. Playfair, a woman would exclaim, "For God's sake tell me, am I displaced?" Utero-sacral atony means as much or as little as atony of other organs, and is to be dealt with in the same way. The subject of abdominal sagging is discussed elsewhere (Vol. III. p. 860); yet these conditions play so important a part in neurasthenic states that they cannot be passed over in silence here. The physics of "abdominal equilibrium" has received a new attention and revision at the hands of Professor A. Keith, of Wenckebach, and many others, and the value of tone of the muscles of the walls —lateral and fundamental—has received a new recognition. We are seeing more and more clearly that not only is the liveliness of the several viscera forwarded by a normal tautness of the whole, but that of the portal venous system also; so that abdominal tone—in the absence of lesion —is not only a condition but also an expression of activity. Successful therapeutics, then, must partly consist in restoring the nutrition and tone of the muscular and ligamentous braces of this region which, in man, is exposed to the stresses of the upright position. Bourcart has insisted on the peculiarities of palpation presented by an atonic abdomen—atonic as a whole and in its several organs; that it lacks sonority, and on palpation has a "cottony" feel, and accordingly the arterial pulse is ill-filled. With the feeble and divergent recti of such persons we are all familiar. That a slack abdomen, with extensions increasing as gravitation tells upon pendulous parts whose normal and mutual embraces are enfeebled, may be a result of a general fatigue—of neurasthenia, or of any other exhausting malady is a priori probable; or again of emaciation of the cushions of fat which pack and pad up the various organs. In many of these states, as in convalescence from acute disease, for instance, recumbency and cautious exercise are almost compulsory until much restoration of tone and fat has taken place; but the neurasthenic may be incessantly restless. To this point we shall return under Treatment. Thus in imperfect convalescence from illness too early a release may lead to abdominal dislocation, and in a predisposed person may co-operate in causing the malady of neurasthenia; but it is more than probable that primary neurasthenia, consisting as it does in atony, may issue in abdominal laxity. For successful treatment it is important to analyse and to estimate the degrees of these several ingredients. To a possible cause of dysuria in this condition I shall refer (p. 762). That, as urged by Stiller, the "costa fluctuans decima" is not an anatomical accident but a criterion of visceroptosis, I am not prepared to admit; but I admit also that I have no definite notes on the subject. We must bear in mind, then, that although dropped viscera or distended stomach may and often do exist without affecting the general consciousness, yet a background of instability, primary or secondary, of the nervous system may multiply its effects enormously. In a well-balanced system, a considerable dislocation of the kidney may set up no discomfort until Dietl's crisis or nephroptosis compels the patient's attention; in a neurasthenic on the other hand, who has no reserve of tone, a moderate sag may keep up headache, backache, sickliness, dyspepsia, and all the rest of it; an ailing state which, until the local vexation is counteracted, resists every remedy.

For descriptions of gastralgia and neurotic dyspepsia the reader is referred to other parts of this work; but the proportions of gastric disorder as a cause and as a manifestation of neurasthenia, are often very difficult to measure in particular cases. Elderly persons, persons in whom carcinoma of the stomach may be latent, may present almost insoluble problems of this kind. In visceral neurasthenia pains, sinkings, and acidities of the stomach are frequent; and even in patients who recover on general tonic treatment the tongue is often coated for a while, the epigastrium tumid, and the faeces scybalous. Digestion is usually slow, often occupying seven hours; in other cases it is asserted that the food leaves the stomach too quickly. The gastric juice may be normal, perhaps usually is so, the commoner defect being in the motor function; or it may be deficient in hydrochloric acid, or, again, this acid may be in excess. I have had the advantage of expert aid in not a few such cases, but from a practical point of view I have not found these distinctions to be of so much importance as clinical observation; we know too little as yet of the fluctuations of free hydrochloric acid in healthy people to rely on chemical estimates in neurasthenia. Much depends on passing fancies of taste and appetite. Tinkering at the digestion alone never cures neurasthenia, although it may help to cut a vicious circle. The bowels are often capricious, sometimes loose, more frequently constipated; not rarely, however, they are irritable, and diarrhoea goes hand in hand or alternates with constipation. Cases of auto-intoxication from the intact bowels, continually alleged but with scanty proof, are not neurasthenia (p. 758). The diarrhoea of such patients—"church diarrhoea," as we might call it—often coexists, for various reasons, with a dread of closed apartments (*vide* p. 749), and is allied to the nervous diarrhoea of Graves' disease, and that of candidates under examination. It is not lienteric nor slimy, but consists of slack faeces. Probably it is to be explained as one of Pawlow's organic reflexes. The curious alliance of membranous colitis with neurasthenia—not with the mere exhaustion of chronic and enfeebling disease, but with neurasthenia proper—is unexplained; there appears to be some kinship between the two disorders, but the problem has been discussed already. Aschaffenburg regards it as a further stage of "examination diarrhoea," a purely nervous malady dependent upon psychical influences, an opinion which seems rather extreme. Still there are many degrees of transition between these states, phases of exaggerated peristalsis, especially during the night or, in a woman, near a catamenial period, with frequent pellet or ribboned stools and crises of pain. The stools may be associated with mucus, or coated with its coagulations, and the step from this to skins and casts does not seem very abrupt. There may be meteorism, or the abdomen may be retracted and intolerant of palpation. Lebeaupin's essay on the subject is worth reading. And I would draw attention to partial spasms of the colon (enterospasm), distressing to the patient and often palpable to the physician. We find in the right iliac fossa, it may be, or in the splenic or in the sigmoid flexure of the colon, a sausage-shaped or cord-like tumour. Sometimes it is unmistakable, even conspicuous; often it is somewhat elusive, so that the observer may palpate the part again and again before deciding that something abnormal is under the hand; and after all, perhaps, records the observation with some doubt. At some visits he is sure there is "something there," at others he is doubtful, or changes his mind. In two of my cases, both of the right iliac fossa, the appendix was explored, with negative results in respect of static mischief, but happily with the effect of the complete and inexplicable cure that often follows surgical operations for phantom pains (p. 755). In these spasmodic cases the motions have some of the characters already alluded to; in spite of blue pill, colocynth, and the like, they are persistently scybalous: no sooner is the gut emptied, or appears so, than these faecal balls begin to form again. In size they vary from that of a hazel nut to that of a walnut, and when broken up are dry like soft chalk, and in texture friable. Such patients complain dolefully of intestinal malaise and of depression of spirits. They are sallow and weak, but often not much emaciated. Of the host of abdominal and pelvic discomforts of which these patients complain it would be tedious to treat at any length; but the reader is referred to the articles on "Visceroptosis" (Vol. III. p. 860) and "Nephroptosis" (Vol. IV. Part I. p. 633).

Extreme emaciation and refusal of food are seen in those cases which approach the type called "anorexia nervosa," or even coincide with them; they are described elsewhere (Vol. III. p. 397).

Patients of another class, with equivocal neurasthenia, but with substantial gastro-intestinal disorder, present problems of peculiar pathological interest, for they raise the debated question of "auto-intoxication," as a factor in neurasthenia. In trying to answer it, we must suspect that a torpid intestine and an imperfect digestion may produce a secondary and consequential auto-intoxication. On a broad view of the case of a certain learned scientist in whose urine indoxyl had appeared, I opined that although intestinal intoxication was probable, it was secondary to a neurasthenia, and not the primary cause of the illness, seeing that for some months he had been trying by diet and other precautions to eliminate all possible constituents of such poisons. I advised a return to a moderate, or indeed to a rather generous, mixed dietary, with long rest of mind and body, and a treatment directed vigorously against neurasthenia; in these circumstances the indican in the urine rather promptly disappeared. As he himself remarked in a letter to me, "the mind seemed after a time to be able to buckle together the several assimilative processes."

Finally, the dyspepsia alleged in many, perhaps in almost all, cases of neurasthenia is very frequently imaginary; a false inference or suggested idea, too often a doctor's suggestion. In all cases of alleged "dyspepsia," the patient should be questioned precisely as to the time and other relations of discomfort to various foods. Very commonly the evidence of anything more than tired stomach is vague or negative, when it is better to stamp out the dyspepsia notion at once.

E. *The Uro-genital Form.*—These cases are as full of vexation for the physician as they are of wretchedness for the patient; and in many of them the physical and moral tangle is bewildering, or even inexplicable. In many of the patients the suffering is genuine, and quite independent of moral degradation; sometimes it has been intensified or even engendered by the indiscretion of a medical adviser, or by the arts of a quack. My reluctance to speak plainly on this part of the subject is overborne by the appearance of Munsterberg's Psychotherapy, in which this eminent man seems to me to shew some indulgence to a certain side of it. To me, possibly from personal prejudice, the recent introspective and confessional methods are odious; if so, this prejudice may disqualify me from equitable judgment, as it certainly repels me from exact perusal, of certain modern books. It is true that our fundamental appetites, whether of sex, of eating and drinking, and so forth, do through all the gradual transformation into higher powers penetrate these; yet in health and sanity they are not carried up in the gross, but minister subconsciously to higher ends; and it is true that if, by any mutilation, the organs of such an instinct be removed, the mutilation will be felt in some lapse of the superstructure; yet to endeavour to express the whole in terms of the foundations is a false debasement. We are hearing enough and to spare of suggestion—but what about the doctor's suggestions to his marionettes, these morbid women and effeminate men, enticed to heat up sophisticated autobiographies, egotisms, and fictions? The weaker the understanding the more it sinks upon the earthier strata of personality; but to appraise even these men and women by such standards, by analysis to reduce them to these lowest terms, is, as it were, to argue of the living blood from the clots in the barber's basin. For these secret introspective dramas, inflamed by reminiscent curiosity, are more than half factitious; and thirst for experience is confused with frailty of morals. If the human heart is deceitful above all things, if before the Father of Spirits we all dissemble, what conceits will not these disconsolates, craving even for scientific sympathy, imagine before the self-ordained confessor of the flesh? Is this by precept and example to develop self-command, to regenerate the faded self-respect? The emphasis laid by many authors on the sexual passion beyond, and even to the neglect of other emotions, and their curious pursuit into remotest recesses of its inventions and lusts, seem to me in method false, and to engender sophistry and pruriency. Should it be argued that in science all values are indifferent, the answer is that medicine is not science, or not science only, but is the application of science to life; and that in the calling of medicine especially, moral values have always held their pre-eminence. If, then, medical influences are to be kept sweet and wholesome, we must consider the wiles of the human heart, conscious and half-conscious, with a decent reserve and in general terms, and in general terms brace the patient to rise superior to them; but this is not to be done by dabbling in them. The sickly person who analyses his sentiments for exhibition to his doctor begins inevitably to pose. Thus the vague hunger of a poor girl for love and a baby is translated into terms of concupiscence, and conversations are recorded, or implied, which seem to me shameless. In my experience the sexual character of these women has more often been that of coldness, or even of aversion. It is notable that these medical penitents are mostly women; in a recent work on Psychoneuroses the self-revelations of two morbid women occupy one-third of its wearisome pages. Let us beware lest the resentment of the laity against "spiritual direction," against the curious inquisitions of the priest into the casuistical paths of naughtiness, may be turned against ourselves.

Von Hösslin has well observed that neurasthenic patients of either sex, men more especially, are apt to lose shame in the description of their symptoms; they talk of secret disorders or perversions without even a show of reticence; or a gastric neurasthenic and "aerophagist" treats the physician to a specimen of his eructations. Now in sexual neurosis this merging of decency in egotism, which differs from the argumentative or blatant obscenity which usually signifies insanity, is a part of the very loss of self-control which we seek to repair. And it is not to be assumed that all patients who complain of discomforts about the genital organs are masturbators or debauched; complaints of weight or dragging in the pelvis, of coldness or numbness about the scrotum or buttocks, of aching in the rectum, of impotence real or imagined, of pollutions, of faulty coitus are often made by actual or former abandoned masturbators, but such symptoms occur also in patients of continent habits, and indeed of cold temperament. Many happily married men find with disgust that when under an attack of neurasthenia they are weakened by pollutions unknown to them in better health. Such sufferers may fall into a melancholy, and are in some peril of suicide, especially if under engagement of marriage. The masturbator is in my experience more prone to delusional mania. In some old abusers of themselves the penis and spinal centres become so irritable that a thought, or a touch, even of the clothing, will produce an emission; when such a one turns coldly from his wife, impotent awake potent only in dreams, that man is on the borders of lunacy, if not within its domain. I need scarcely warn the young physician never to allow himself to advise marriage to an abject of this kind, lest two people be made wretched instead of one; but on the other hand, many good fellows fall into sexual dreads, and are beset by obsessions—as of impotence and the like—which perhaps by their very importunity and stress may set up more or less neurasthenia, and may even create in them a false aversion from women. Patients of this class may be cured promptly if a discreet medical adviser, clearly discriminating the mere self-tormentor from the debased, encourages the former to marry some kindly woman. Indeed, we may encourage him to pour out his sorrows; while to the dirty confidences of the other we turn a deaf ear, lest, as I have said, by listening to these confessions we breed in him a morbid vanity in his own degradation. Now and then, uxorious men, or men unaware of the proper limits to this kind of indulgence, bring themselves into a somewhat similar state of debility which in persons of otherwise healthy constitution is soon cured by due abstinence. These persons, often robust enough in appearance, may complain of attacks of exhaustion, associated, as the doctor may discover, with a retardation of the pulse rate. No sensible man objects to a business-like allusion by the physician to his sexual habits; and most men discover for themselves that if good work is to be done coitus must be economised. The drain is more continual, of course, in childless marriage.

The urine should always be examined carefully; not especially for albumin or sugar, but for less radical changes. It is often alkaline or neutral; or phosphaturia, oxaluria, or azoturia may throw some light on the case (*vide* Vol. III. p. 226, and Vol. IV. Part I. p. 553). Dr. Maguire has recently noted the occurrence of oxaluria with certain nervous and dyspeptic symptoms in persons subject to mental distress, and that these people should be fed generously, including even wine in the dietary. It is commonly supposed, I think, that the urine of neurasthenia is of high density and, by the hypobromide and uranium tests, shews an excess of urea and of phosphates, especially of phosphates, and that these excesses run parallel with the degrees of the symptoms; but others (Horsley, etc.) say that although the urine is copious the specific gravity is correspondingly low. It is said that sugar may appear in mere neurasthenia; I should scarcely have inserted this were it not alleged by Aschaffenburg, and had I not twice found sugar in the urine of candidates for life insurance, in both on the eve of marriage with its moments of crisis and anxiety. Both were married, nevertheless, and, so far as appearances go, have enjoyed good health since. The last drops of urine, in man, may be examined for spermatozoa, and spun for casts: in the absence of cardio-vascular changes a few hyaline casts, even with minute traces of albumin, are not usually of much consequence; apart from "postural albuminuria," such traces of albumin are often found in anaemic, slinking youths in whom masturbation may be suspected. Irritable bladder is a common symptom, especially in men; micturition is smarting, attended with shiver, and may end in a jet or two of mucus, often followed by a sense of exhaustion which is difficult to explain, unless it be due to the dread of "spermatorrhoea." Symons Eccles attributed a distressing kind of dysuria—frequency and straining—to sigmoid prolapse due to slackening of the pelvic loop (*vide* p. 757). This prolapse, often with faecal lumps inside it, he said, may be felt per rectum, as the patient bears down to the finger, and by arresting faecal carriage, "auto-intoxication" might be set up. Or in women there may be a drag on the broad ligament. Appendix disease might be suspected in such cases.

The catamenia offer no peculiar feature; in any debilitated woman, of course, they may be scanty or absent. Ovarian tenderness is not a characteristic feature of neurasthenia; it has been described with the nervous symptoms of some other pelvic disorders in the *System of Gynaecology.*

F. **Traumatic Neurasthenia.**—It would seem illogical to divide the disease into varieties for reasons of causation only; on this basis, indeed, the division would be unnecessary. However, this division, the name of which we owe to Oppenheim, takes its place somewhat aside from the others because the traumatic series is not identical with the "spontaneous"; and it may be that in not a few cases the variety does not depend altogether upon mechanical injury, but upon injury or shock attended with alarm or stupefaction (Schreckneurosis). For this reason in the first edition of this work traumatic neurasthenia was dealt with, by Sir Victor Horsley, in a separate chapter. On reconsideration, however, so trenchant a division seemed to Sir Victor and the editors to be inconvenient; for, to use the language of our distinguished contributor in 1899, the symptoms of traumatic must be regarded on the whole as forming with those of simple neurasthenia a pathological series which can be recognised clinically with great ease. Notwithstanding, the two series of events do on comparison present certain considerable differences; briefly, the chief difference lies in some intrusion of various degrees of hysteria into the process. In the following paragraphs I have availed myself freely of Sir Victor Horsley's description.

Traumatic cases may be divided into Acute and Chronic. In the *acute* form the patient may be dazed and inattentive to what is going on, but perfectly capable of answering clearly when spoken to, and of giving a connected account of what has happened, shewing that at no time had there been any enduring loss of consciousness; or he may be in a state of excitement and restlessness, alternately laughing and crying as in an hysterical attack, but equally capable of recall to himself and of recollection. Or he may pass in a few hours from the dazed to the excited condition, and so remain for several days, or a week, during which time other symptoms may arise (Horsley). In most respects, however, the symptoms of the acute and of the chronic form differ only in degree. Fatigue is, of course, an early and a salient symptom.

If any structural injury is done the case will be complicated by changes of other kinds which will be discriminated without difficulty.

In the acute form, beside the headache, inattention, and forgetfulness, the sleep will certainly be affected, and often broken up by nightmare and dreams in which animals commonly figure, phases identical with the "zoopsia" of hystero-epilepsy. The intolerance of light and sound are well marked, and a sensation of cold in the skin (vaso-constriction ?) may be felt acutely. Movements may be so impulsive as to simulate incoordination, and a tremor may simulate "intention tremor" (Horsley). The small pulse of vaso-constriction is often mistaken for a pulse of cardiac failure. In this acute stage there is no drop in nutrition, though the pyrexia of the remarkable kind known in hysteria as "neurotic fever" may appear; it presents invariably a rapid rise, an exceedingly brief fastigium, and a very rapid fall. Its speedy and certain amelioration by cold sponging, and the absence of wasting, shew it to be a nervous phenomenon. As a rule it does not rise above 101°, but it may fly up to incredible heights; in one case (Horsley) to 111°; in the well-known and most vigilantly controlled case of Mr. J. W. Teale—attested by Mr. Pridgin Teale, myself, and other expert witnesses, to at least 118° F. again and again.

As at first there may be grave doubt whether structural injury had been sustained or not, it is fortunate that in acute neurasthenia also the first stage of the treatment is absolute rest in bed in a quiet chamber with subdued light.

In *chronic traumatic neurasthenia*, besides the injury, subordinate causes, proximate and remote, have more effect; especially heredity, previous overwork, or illness with possibly adherent toxins, as in the sequel of the infectious fevers. Among the commonest causes are those which give a jar to the whole body, as a fall from a horse or even the bucking of a horse, railway collisions, and other accidents.

A notable and not infrequent feature of the chronic form is the incubation-period—first perhaps specified by Charcot—between the shock and the first serious manifestation of symptoms, or even of the first symptom; so that the interval, instead of being a character of falsity, is rather a note of genuineness. The interval is usually a week; it may be longer, even several weeks (Horsley). Mr. Dent says the interval may be one of rather unnatural calm; especially in post-operative cases, a note which brings back vividly a case or two of my own. A frequent first symptom is that the patient finds a special effort is needed for actions previously almost automatic—to write a letter, for instance, or to perceive the meaning of a document. And the cerebral activity is hindered by "an oppressive fog," which may lift quite suddenly. At the same time he becomes irritable and hypersensitive. The sleep is dreamy and broken. The headache is of two or three forms: a dull vertical pain with heat or pain in the posterior division of the second cervical nerve even so far as the vertebra prominens, with some stiffness and even puffiness of the neck, and clicks, with pain, on rotation. Less commonly the headache is seated in the temples.

The sensory symptoms, general and special, as described by Sir Victor Horsley, do not differ importantly from those of the ordinary kind of neurasthenia. He has not found the strange suspensions of cutaneous sensibility characteristic of hysteria. But in the motor field the facts are more complicated. We are reminded of hysteria by the spasms and contractures, which may come on independently of voluntary movement; they are common in the limbs, and occasionally may be detected in "a heaping up of the tongue to one side" (V. Horsley). In the limbs the digits are most frequently flexed, and the foot inverted with plantar flexion; in the wrist it is usually fixation between flexion and extension. The elbow and knee are frequently extended, but the shoulder and arm generally escape.

Hystero-epilepsy occurs in a few cases of exciting accident (Horsley). I witnessed many such attacks in a young woman shaken and much alarmed in a railway accident; the seizures corresponded with the worst of those I had seen in Charcot's wards, and with the sketches made by his pupils and other French physicians.

Pareses are more frequent than in spontaneous neurasthenia, so that in this respect again the traumatic form presents resemblances to hysteria. These may be so general as to be consistent with cerebral tumour or other organic lesion, and often extend to the trunk and abdominal muscles. Local palsies are generally monoplegic, the lower limbs being most frequently affected; the arms less frequently, the face very rarely. As with the anaesthesias, the left side is affected three times more often than the right.

The rule is that the *reflexes* are exaggerated; but in very severe cases, or on the establishment of analgesia, the superficial may be reduced or even abolished. The deep reflexes are constantly exaggerated in the first stage, but in the second stage of severe cases they may be abolished, though probably even then the knee-jerk may be obtained by Jendrássik's reinforcement. Ankle-clonus is now recognised as an occasional accompaniment of the functional disorder.

The disorders of the several organic systems differ in no important way from those of ordinary neurasthenia.

Furthermore, there is an aspect of traumatic neurasthenia which, since its description by Mr. Herbert Page, has scarcely received due emphasis; namely, that at the moment of jar the muscles, ligaments, and articulations of the spine are instinctively thrown into action and stress, and without any blow may be stretched and strained, chiefly in the lumbar and dorso-lumbar region. There is no external sign, but the patient cannot stoop quickly; he holds his back stiffly, and rises cautiously from his seat. He complains of continual ache in the part, especially—as we should expect in these fibres—on change of weather; and they are tender to deep pressure. A civil servant, whom I have seen on sundry occasions for some years, was thrown heavily upon the upper dorsal spine now nineteen years ago. Ever since, although he has done his duty loyally, he has been subject to returns of neurasthenic symptoms, and especially of weary aches in this region, extending to the occiput, shoulders, and arms, and to a sense of "contraction" referred to the scapular and thoracic muscles on the left side. All this is worse in damp weather. There have been no hysterical features in this case.

From these reflections it will be seen that, as Page, Oppenheim (in his *Lehrbuch*), Strümpell, and Raymond also say, traumatic neurasthenia is a mixed category, with many variable constituent degrees of neurasthenical, hysterical, and hypochondriacal symptoms. An overmastering idea of physical disability, general or partial, may suppress the innervation required for action; in such cases the treatment must be more directly "psychical" than in ordinary neurasthenia. Considerable light is thrown on traumatic neurosis by descriptions of cases such as those of police constables, narrated by Mr. Clinton Dent, and by similar cases of other authors, in which the "trauma" was slight, nominal, or even escaped. Such cases, many and diverse, during the earthquake of Messina are recorded by d'Abundo, but, the wounded apart, seemed almost all to be of the nature of hysteria. On the whole, perversion of inhibitory function seems to have been more notable than exhaustion—there was more hysteria than neurasthenia. And the more preponderant the hysterical ingredient, the more is the liability to "subconscious" simulation. Knopp, in a study of 200 cases of this kind, ranged 70 on the side of hysteria, 50 as neurasthenic. He did not observe any great difference, among the genuine cases, between those subject to litigation and the non-litigated. In some of his cases hysterical attacks occurred at the time of the accident; in others such seizures followed at various intervals, often with subsequent pareses, contractures or anaesthesias: others ran rather on the neurasthenic lines of lassitude, inattention, and so forth. There were some vomiting cases in his series. Dickens, in his railway adventure, was not hurt, though the carriage in which he was travelling hung over the edge of a chasm. He worked bravely in assisting the injured and then collected his own packages and papers, but he never wholly got over the mental shock; his daughter writes that on the least occasion, when travelling by rail, "he would often suddenly fall into a paroxysm of fear, clutch the arms of the railway carriage, large beads of perspiration standing on his face, and suffer agonies of terror." This was pure "suggestion" (hysteria). Mr. Dent's paper merits more attention than I can give to it here; in police cases the patients have everything to gain by a return to work, yet in these there appeared many of the very anomalies which in litigated cases arouse the suspicions of referees. Indeed, Mr. Dent points out—what has not received sufficient attention—that in the policeman on service blows or shocks may set up "traumatic neuroses" which are not set up by collisions or blows, apparently similar, in football or other games, and partake therefore of the nature of hysteria. Speaking generally, a blow on any part of the body may determine the neurotic sequels, but blows on the head or spine or on the testicle are most effectual; yet, blows on the testicle in hunting rarely entail such consequences. In a paper on post-operative neurasthenia, however, Mr. Dent does state that operations about the genital organs are more likely to produce them. The severity of the blow, mechanical injuries of course apart, did not seem to matter much; yet—from the nervous point of view—"on the head no injury is too trivial." Mr. Dent considers that the various effects of blows during games and those received on duty have their difference in the conditions of mental stress and responsibility at the moment. With the police, exciting and perilous encounter may break in suddenly upon periods of solitary watching. Again, even a slight injury to a very important limb may entail grievous anxiety; as in the case, for instance, of a musician whose hand is hurt.

From such considerations we see that some of these plainer cases are scarcely on the borderland, but clearly cross the line into the category of hysteria; that is, they signify a lop-sided equilibrium of the system of forces rather than poverty or depletion of energy. For instance, in a case reported by Mr. Verrall of Brighton, a severe blow on the head in the right parietal region was followed by numbness of the whole left side, sharply limited at the middle line, with some muscular weakness. This passed off in a few weeks, but occasionally had returned rather suddenly for a day or two. Certain recent authors insist indeed that in the main "traumatic neurasthenia" is a misnomer; that these cases are just hysterical, and to be cured promptly on counteracting the morbid by normal suggestion. Indeed, it has been boldly asserted that all cases with an "incubation-period" are hysteria. If this point of view contains much important truth—important both in prognosis and treatment, yet it is not the whole truth. The psychical factor is no doubt more prevalent than we had fully realised, and in cases of trivial injury, or of no material lesion, is pre-eminent; but no less surely in the cases of physical blow or jar the local or the neurasthenic factor is often most difficult to cure, and quite irresponsive to suggestive methods.

In this connexion we may place the "*post-operative*" *cases of neurasthenia*, now well established on the testimony of perhaps all surgeons of experience. On these also Mr. Dent has written. After all toxic symptoms have passed away, and convalescence should be forward, the ordinary symptoms of neurasthenia—insomnia, inattention, irritability, etc.—set in and prevent return to the work or pleasures of life. The duration of this delay bears no relation to the severity of the case; it may follow minor operations, perhaps, as Mr. Corner has suggested, because in these the patient is not kept in bed long enough to recover from the harass, the narcosis, and so forth.

It is said that ovarian operations are specially apt to be followed by neurasthenic sequels. Knowsley Thornton was wont to warn his patients of this chance; telling them that such a debility might last for twelve months after the operation; but Dr. Herman thinks this computation was somewhat too long. In these post-operative cases, although the local disease may be removed and all the direct effects of the interference well surmounted, yet the patient remains weak, and lacks energy and self-control. Mr. C. A. Morris's selection of cases of battle injuries belong to this category, though it is difficult in these again to discriminate between the effects of fear, excitement, exposure, privation, fatigue, and surgical operation. In many cases amputation was added to these distresses; but in others "strapping men without injury but exposed to mental tension, fatigue, and privation, would shrink from the slightest touch and shed tears like children."

Of the **pathology** of neurasthenia—in the sense of morbid anatomy—we know nothing. That it is often accompanied by vasomotor instability is true, but it is a long leap to assume that herein lies the primary pathogeny of the disease. That the arteries of the brain are subject to vasomotor fluctuation seems at present very doubtful; but that the phenomena of cerebral anaemia may throw some light on those of neurasthenia will be admitted by the reader who turns to Dr. Leonard Hill's article, p. 23. H. L. Barnard, working on the same lines with Dr. L. Hill, attributed "many neurasthenic troubles" to a lax abdominal wall in a person with a naturally low blood-pressure. "The blood leaks into the capacious portal system," and the heart is ill filled. To talk of "Paratrophie," "hypotrophische Zustände," "Névrose arthritique," and so forth, seems to me to be mere verbosity. That the weariness depends simply or directly upon accumulation of the "fatigue products" I have given reason to doubt. Clinical experience does not favour this explanation; unless we are to call hypermetropic astigmatism, for instance, an auto-intoxication. Auto-intoxication attributed to some definite disorder in the colon ought to be discernible by other signs; for instance, by indican or aromatic sulphates in the urine.

Diagnosis.—I repeat that, like all other diseases, neurasthenia at its margins melts by infinite gradations into other morbid groups; in its traumatic form it is often mingled with or merges into hysteria; or delusional insanity and melancholia, although usually they pursue other courses, may be entangled with it. Moreover, neurasthenia, bred perhaps of mental distress and disappointment, may supervene upon other diseases, or be grafted into them, especially on diseases of the nervous system. Thus it is that in chronic organic disease, such as tabes or rheumatoid arthritis, for instance, much good may be done by clearing away a neurasthenic accretion as well as by toning up the acquired slackness of unexercised parts, thus reducing the malady to its lowest terms. In distinction from hysteria, neurasthenia does not include contraction of the visual field, nor other anaesthesia; the indefinable "numbnesses" of which the neurasthenic patient is conscious, often vexatiously conscious, do not at all resemble the patches of anaemic anaesthesia which the physician reveals to the hysterical patient, often to her surprise. Hysteria, again, "trauma" apart, in adults is, broadly speaking, a disease of women; but neurasthenia occurs rather more frequently in men.

Dr. Herman says decisively that hysteria is to be distinguished from neurasthenia by its own criterions, and emphasises conjunctival anaesthesia.

The following diagnostic contrasts, which I have modified and enlarged from a table drawn up by von Hösslin (1), may be of service to the practitioner.

HYSTERIA.	NEURASTHENIA.
Chiefly in women; in adult life, and trauma apart, generally.	At least as frequent in men as in women.
Sudden onset, perhaps with some sort of fit, or on annoyance or shock.	Onset, unless traumatic, usually gradual.
Course capricious, irregular, or paroxysmal.	Course more or less monotonous.
Hysterical fits or "scenes."	Absent.
Dispelled, often very rapidly, always mitigated, by enforced effort.	Aggravated, often seriously, by incautious effort.
Intervals or phases of fussy activity.	Uniform incapacity.
Hallucinations.	Absent.
Clavus.	Absent.
Globus, Zones.	Absent.
Definite areas of anaesthesia, especially plantar, faucial, conjunctival.	Absent.
Cutaneous reflexes diminished.	Reflexes usually increased.
Concentric abolition of visual fields.	Absent.
Contractures, etc. Pseudo-sciatica.	Absent.
Neuroses of joints.	Absent.
Palsies or cramps of voluntary muscles.	Absent.
Subject to suggestion.	Not more so than other diseases.
Dual consciousness, etc.	Absent.
Disposition to exaggerate malady and to crave for sympathy.	Depression of spirits, desire to get well.
Mental powers fitful, but in some tracts acute and enduring.	Mental powers uniform, but soon spent.

I would reiterate the maxim, to which Kräpelin has given his support, that whereas in neurasthenia to make impetuous and persistent efforts to fight against the disability is usually injurious, in hysteria it is essential to the cure,[1] but it was of such women that St. Teresa said, "Elles sont d'une faiblesse herculéenne."

Alcoholism and morphinism are to be remembered as possible complications; but drunkenness is not very common among neurasthenics; in most of them alcohol, even in small doses, is ill-borne, it produces flushing and other deterrent discomforts. Tobacco vertigo, and the other nervous consequences of the weed, may resemble those of neurasthenia;

---

[1] In my view of hysteria the "will" seems to be not absent but engaged elsewhere; however, this is not the place to discuss hysteria; the reader is referred to the special article in this *System*, to Briquet's standard work, and to Georg Voss, *Klin. Beiträge z. Hysterie*, Jena, 1909.

as also do more often those of tea and coffee; such sources of error must in all cases be remembered and excluded. The symptoms of tobacco may persist for six weeks of total abstinence from it. The cardio-gastric distress, insomnia, and "shattered" nerves of chronic coffee poisoning may very closely simulate such forms of neurasthenia. Tea may produce severe and persistent gastralgia, or harassing cardiac arrhythmia. Nervous symptoms, including vertigo, are wont to occur in certain cases of high blood-pressure, but these come on gradually in later life, and ought not to be misleading. Stormy functions must be distinguished from shallowness of capacity.

I need not repeat that mere anaemia and mere debility do not constitute neurasthenia; though anaemia may intensify it. Any sign or symptom of incipient rheumatoid arthritis must be suspiciously watched: this is a notable point of diagnosis. The headache and inability to read continuously which belong to disorders of refraction are often attended with much mental anxiety and with some sleeplessness; in this way, and the more as such persons are drawn from the ranks of students liable to be overworked, neurasthenia is not rarely engendered. No case of neurasthenia, whether attended with headache or not, can be fully interpreted or treated until the eyes have been carefully examined by a skilled oculist. Errors of refraction often bring about strange consequences.

The extraordinary and abiding prostration which may follow influenza and other infectious diseases, is to be distinguished from neurasthenia, although in many respects the treatment may follow similar lines. We must consider carefully whether the state be one of toxins still adherent, or only a debility due to bygone causes.

In cardiac cases we have to distinguish between functional cardiac disorder and some occult disease of the heart, especially latent mitral stenosis.

Larval Graves' disease is often an alternative hard to accept or to reject. Many young women have slight enlargements of the thyroid, and a pulse which quickens too readily, at any rate under examination, or on slight effort.

We have seen that it is often difficult to discriminate between a primary dyspepsia and neurasthenia; indeed, a vigilant observation of the results of treatment may be necessary before a final diagnosis can be given. Static dilatation of the stomach, if definitely established, would take the case, in some part, at any rate, out of the diagnosis of neurasthenia; but in this malady the stomach is very frequently in atonic relaxation, a condition I have described already (Vol. III. p. 529), and need not dwell upon here; and the squelchy stomachs of many neurasthenics may seem to be dilated. Some neurasthenics ruminate, but they do not often vomit. The diagnosis of neurotic dyspepsia from duodenal ulcer, chronic gastritis, and so forth, has been fully considered in the third volume. Among the points of more or less significance would be excessive knee-jerks, headache, irritability, fatigability, and palpitations. Recent experience is teaching us that weariness and pains in the abdomen, simulating neuralgia, depend not infrequently upon duodenal ulcer or appendix troubles. In all such cases skilled and assiduous search must be made in the stools for occult blood, or pancreatic disorder. The debility of lingering convalescences, during which, as of influenza, toxic bodies are apt to retain a grip upon the organs, must be discriminated; during epidemics, as of Pfeiffer's influenza, grave sequels of evanescent infections may elude explanation.

Again, I would emphasise the criterion of premature fatigability in diagnosis; every "neurotic" is not a neurasthenic, and many neurotics dissipate abundant energy.

In elderly persons neurasthenia may simulate latent carcinoma so closely as to make positive diagnosis impossible,—a sad dilemma. Nutrition droops, strength fails, the face is worn and sallow. With the appetite free hydrochloric acid diminishes, and often disappears. I speak from particular cases of neurasthenia ending in recovery, in which expert chemical and other assistance was obtained. One of these patients murmured to me when, after a long discussion, my colleagues had departed—"I know what the diagnosis is—it's worry!" And he proved to be right.

In traumatic, or possibly traumatic, cases I would note the initial difficulty of diagnosis between mere hyperaesthesia, jar to the back—even without blow (p. 764), strain, and surgical lesion; conditions obviously calling for different management: and, again, between ' functional" sufferings affecting the head and concussion of severer degree—probably consisting in minute cerebral disruptions, punctiform haemorrhages, slight latent fracture, or meningitis—injuries fully discussed in works on surgery of the head, to which the reader is referred. Dr. Judson Bury divides cases of minute lesions of the cortex into two groups: those resembling ordinary neurasthenia—but with more mental dulness during convalescence; and those in which dulness, apathy, and drowsiness, rather than insomnia, signify minute lacerations or contusions with punctiform haemorrhages. The patients of the first group begin to work but soon tire; those of the second cannot begin, and amendment is generally slow and disappointing.

Insomnia, wrecking to the health as it may be, does not necessarily belong to neurasthenia, or only in a derivative sense; by three or four good nights the effects of insomnia are relieved.

Dysuria may be nervous, micturition being frequent, straining, and with ache at the neck of the bladder. The quantities of urine may be very irregular, scanty or profuse. Oxaluria, baruria, phosphaturia will be distinguished on the principles set forth elsewhere (Vol. III. p. 226). No blood, pus, casts, or bacilli should be present; nor any constant excess of oxalates. If there be albumin it should prove postural ("cyclical"). To glycosuria I have referred already (p. 761); I have seen one case at least of neurasthenia in a middle-aged man, in which it was not easy to discriminate between them.

The warning given by many authors, emphatically by Kräpelin, lest incipient general paralysis of the insane be mistaken for neurasthenia, is a grave one. I have found myself in this perplexity on several occasions, and once I fell into the trap. The patient seemed no more than fretful and weary, and his gait fatigued; his facial twitching, if any, was of the slightest, and, to tell the truth, general paralysis never crossed my mind till a junior partner in his firm telephoned to me that the patient had gone abroad without notice, and was wiring for large remittances. A messenger was despatched to bring him home, and soon after he was placed under restraint. In doubtful cases the pupils and the handwriting should be scrutinised. Specht says that here Kräpelin's tests are very valuable. In another case, in a highly-strung, frail man, I took for neurasthenic a feeble gait which after a long time defined itself as lateral sclerosis with clonus, cramps, etc., which even now, after the lapse of many years, has made but very slow advance. Weariness is an early symptom of both diseases. Incipient tabes again might be ambiguous. The equivocal characters of disseminated sclerosis are already but too well known to all of us; occurring, as it often does, in young persons, especially in young women, its differentiation in early stages may be impossible. In one case, which proved to be sclerosis, three of us were quite baffled by the dilemma. Ankle-clonus is not conclusive, even if fairly well marked; in two cases at least I have seen complete recovery after a period during which the sign was conspicuously present; one was a case of nervous prostration after the fright of a house on fire. For discussion of this diagnosis the reader is referred to the special article (Vol. VII. p. 841).

Dr. Mott states that cerebral syphilis with the symptoms of irritability, insomnia, lassitude, loss of attention, plus jerks, and general hypotonic condition, may simulate neurasthenia. He suggests that, besides the Wassermann test, an examination of the cerebrospinal fluid might be helpful. Kräpelin—who, by the way, while complaining, and with good reason, of the loose application of the name Neurasthenia, yet himself applies it to some cases of retained catabolites—cautions us lest in certain cases in young persons dementia precox, especially in its hebephrenic form, or circular, or "depressive" insanities, be confounded with neurasthenia. As points of distinction he indicates outbreaks of excitement, stuporose, automatic, resistive, or quasi-cataleptic states; and generally an inconstancy of conditions, as contrasted with the monotonous round of neurasthenia. And, I may add that periodicities, religious or pecuniary delusions, objectless wanderings, and radical alterations of character tell for insanity. If a bright and promising boy suddenly becomes hypochondriacal, cannot think, is lack-lustre and lazy, or is anxious, restless and irritable though soon tired, his condition is not to be hastily attributed to masturbation, not even if the youth accuses himself of the practice. This tormenting habit may be the cause, or be substantially contributory; but the confusion usually lies rather in a dislocation of the relative marches of mind and body, and of the world and the awakening self—the sexual, but also the intellectual and imaginative self. The condition has no kinship with neurasthenia, and must not be treated as such; some sharpness of discipline, together with some enlargements of the boy's sphere of wholesome experience, are to be desired. I need not explain that to put big boys to bed, and to massage their limbs, is morally hazardous; even in organic heart diseases such methods should in them be kept watchfully within limits.

The "morphinist" varies between times of capacity for anything and utter prostration; in the brighter phases contraction of the pupils will be noted. Topers are usually fat and often red faced; they, too, vary between extremes of indolence and activity; they retch of a morning, and often are moved in the bowels before breakfast, although, in the female drunkard at any rate, the tongue may be clean.

To mistake arteriosclerotic diseases, of whatever kind, for neurasthenia is a common, and often scarcely a venial, error. Yet occasionally one sees both the error made and the blunder defended. Some of these cases turn out to be early and insidious invasions of granular kidney; others of the malady I have called Hyperpiesis, one which, I think, has no other name; others prove to be premature senility; others, again, to be toxic states of various origin. We shall not forget that cerebral arteriosclerosis may precede that in more accessible areas. It is said the electro-cardiogram may help in this doubt. A short time ago a gentleman under forty came under my notice as a "pronounced neurasthenic," in whom I found the heart's apex an inch outside the mid-clavicular line, the aortic valve ringing, and the blood-pressure up to nearly 200 mm. The kidneys were normal. Not very rarely old people become genuinely neurasthenic, usually under some distress. I have already alluded (p. 770) to such a case in a man considerably over threescore; he presented the ordinary symptoms of the malady. But it is true that the evidence may be equivocal, and that for a while the difficulty of excluding latent, malignant, or senile decay in an insidious form may be insuperable. The patient to whom I refer recovered, to die of an acute disease; at the necropsy the alternative diagnosis of neurasthenia was proved to have been the correct one. On the other hand, Huchard's "fifty-year-old neurasthenics" are a motley collection; on his own statement 25 per cent of them had high blood-pressures. Now persistently excessive arterial pressure—due care being taken to record it in tranquil moments—excludes mere neurasthenia. In hyperpiesis low spirits, morbid apprehensions, and weariness are frequent symptoms; and in neurasthenia Huchard's milk and vegetable diet without further and more proper means does no good.

Hypochondriasis, confused by Müller and others with neurasthenia, is a fixed delusive idea of a particular disease or local suffering; not mere arguable apprehensions now of this and now of the other disease, like those of the young student of medicine ("nosophobia"). One may persuade the neurasthenic that his heart, for instance, is not from the physician's point of view diseased; but not unreasonably he wants it cured all the same. The man who watches his stools or his testicles is usually a hypochondriac. These persons, if merely hypochondriacs, are in good, often excellent general health, if they could but believe it. The mere hypochondriac though disheartened is not disabled. Better a dozen neurasthenics than one hypochondriac.

Melancholia is a mental disease often attended with delusions; it may complicate or follow neurasthenia, but usually the conditions are distinct. The melancholic, in the milder degrees, suffers little from bodily disability, unless it be from a torpid colon; the features of the severer degrees will be unmistakable. He is passive and despondent; the neurasthenic clutches at a new promise of relief, his mind is open to argument, and his heart to hope. If suicide may occur in either malady, it is far more frequent in melancholia. Delusional insanity, again, may be engrafted upon neurasthenia; and, as in the case of melancholia, transition cases between the typical states are seen, though not so frequently as is commonly supposed.

One main rule is not to accept the diagnosis of neurasthenia or hypochondriasis, nor to call people fanciful and the like, until we have exhausted all other possible explanations. I have spoken of "neurosis" as "the last ditch" of diagnosis. For, in the memories of us all, how many are the cases in which such judgments proved fallacious; of simulations of neurasthenia by some visceral adhesion, by a latent affection of the appendix, gall-bladder or kidney, some stealthy organic nervous disease and so forth. A young lady languishing under a severe aching pain seated at a small spot corresponding to the last lumbar vertebra, was wholly relieved after some weeks by a violent attack of gall-stone colic, and a single, rather large unfaceted stone appeared in the stool. Again, an unmarried lady of great intelligence, and of no less physical capacity, became the victim of a continual pain in the hypogastrium; after many months of misery she was seized with a violent attack of gout in the great toe, when the old pain disappeared for good and all.

In respect of local disorders, if multiple, attention may exaggerate one to the reduction or concealment of the other; for instance, in a "gastric neurasthenic" a dropped kidney or a pelvic drag may mask a diseased appendix or a duodenal ulcer. Not even by a large experience of men and their ills is the physician enabled to form true judgments in some of these obscurer cases.

In "cardiac cases" we are too prone to hedge, especially as to the older patients, or those who have led self-indulgent or invalid lives, with qualifications about possible "fatty degeneration," and so forth, instead of frankly declaring against disease. In a few cases some suspension of judgment is inevitable, but unless a defect is notable, such perhaps as a clicking first sound at the apex, a reduplicated second, perhaps a suspicious push before the systole, or an equivocal thrill, acquit the heart. Don't kill the patient with doubts.

The mouth should be examined for pyorrhoea, for masticating power, for sources of pain or pressures of crowding teeth (Upson), and every other channel of the body inquired into. One of my "cardiac" cases turned out to depend upon a bad mouth. Nor must we overlook the possibility of taenia in a case of obscure visceral discomfort in butchers, cooks, or children who have been fed on raw meat.

One of the most embarrassing of these problems is the detection of the malingerer; and in these days of insurance against sickness the problem presents itself with more and more urgency, for, as we have seen, the malady is by no means an apanage of the wealthier or more cultivated classes. Experienced observers say that on complaint of pain in any part, if the complaint be genuine the pulse will inevitably respond to rub or pressure on the tender spot. It is usually accelerated; but it may be retarded, diminished in volume by constriction, or even broken in its rhythm. The hands or forehead may sweat. Before such tests are applied the patient should have been kept quiet in bed for a while. It is said that under such a test the blood-pressures also may change, especially upwards; but such appreciations are as yet too refined for practice. (For remarks on the urine *vide* p. 761.) Flora's sign of defective tetanic response to prolonged faradic stimulation of the muscles alleged to be affected, and slow recovery of such capacity after the test, does not seem to have "caught on" in the German tests for "Unfalls-Versicherungsneurose." Dr. Warner's tests are scarcely adapted for the detection of malingerers; but that substantial aid may be found in those of Kräpelin and of Rivers I have already explained (p. 729). Eppinger and Hess propose pharmacological tests: after a comparison of certain phenomena of the vagus and of the sympathetic, contrasting the "vago-tonismus" of youth with the "sympatico-tonismus" of later life, they proceed to discuss the comparative effects of pilocarpine, atropine, adrenalin, and so forth. The tests proposed by Müller, Bechterew, and others have not been so far verified or adapted to ordinary practice as to command a place in this article. The reader who desires to pursue this line of inquiry is referred to their essays.

This is perhaps a convenient place for a protest against the laxity of dignifying by the diagnosis of neurasthenia or of hypochondriasis, the grumbles and discontents of idle, selfish, egotistical, and undisciplined persons, who nurse up factitious griefs. It is true, as we have seen, that as the control members of our nervous mechanism are more comprehensive they are more fatigable, so that as the moralist knows too well, they are so much the more prone to idle, to "have the luck to be out of the way when there is anything to be done." The lower the centre the more constantly busy it is. At best our higher ascendancies seem to work rather by happy leaps from the mean position than by steady marches, and to need social reinforcements for the accomplishment and maintenance of standards. The name of neurasthenic, or even of "neurotic," should not then be attached to those eager, restless, highly sensitive, and impulsive persons who, lavish indeed of their strength, fail to use it with economy. Many men of fervid temperament, keen intelligence, and quick gestures, who do really an excess of work the year round, are too valuable to be "cured"; but we may teach them some economies. It must not be assumed that every "Temperament-Mensch" is liable to neurasthenia; many such "wiry" persons shew remarkable endurance and tenacity, and often even no little power of throwing off fatigue. Indeed, some of them seem to be anaesthetic of fatigue, declaring that they do not know what it is to be tired; this is a misfortune, for they wear themselves out before the world is ready to part with them.

Prognosis.—It must be remembered that in some persons neurasthenia seems rather to be the expression of a permanent and congenital quality than of an intercurrent disease. "Acquired cases" offer every chance of complete cure; ingrained neurasthenia, often mitigated, is never rooted out. Thus a long life may be divided into periods of effort and intervals of exhaustion, apathy, irritability, depression of spirits, and bodily suffering.

Prognosis should be expressed in plain and concise language, not hedged about with possibilities and qualifications. The patient is too faint-hearted for these compensatory excursions. "Das Gute liegt ihm oft so fern." We cannot calculate the phases of neurasthenia as those of an infection; but the disease is not irremediable in the sense that most of the organic diseases are irremediable. Nor can neurasthenia be said to shorten life, though it may empty it of achievement. I repeat, if neurasthenia must be regarded in its active phases as a malady, in its passive intervals it is rather a colour of the constitution or temperament, an hereditary complexion not to be washed out; and when in hereditary cases we speak of cure we are understood to speak only of abatement or relief of the attack on hand. Leubuscher and Bibrowitz, estimating the results of a large number of cases in their sanatorium, where hydropathic and electric methods, massage, graduated exercises, and healthy serene circumstances avail for cure, report that 60 per cent were discharged fit for full work; 37 per cent fairly fit for moderate work; 2·3 per cent uncured. But in respect of these good results the authors admit that under the insurance system many complain who otherwise would not have been heard of. Petrén considers prognosis to be relatively better in acute cases with a definite external cause, such as bodily or "psychical" trauma or surgical operation; presumably because they are open to wholesome suggestion. Nägeli of Zurich, of 200 cases, found at date the results in 138 could be recorded: 80 were fully reoccupied in their own calling; 17 were doing well in a changed employment; 7 said they were well: the rest had not recovered, but 13 were supposed to be shams. To hunt for moles or crooked ears as stigmata of degeneration is bad medicine; the patient is indeed wretched and the physician faithless who cower before "heredity" as under a fate. In children, neuroses—such as headaches, night-terrors, sleep-walking, or defect of self-control, should be noted even more for that which they presage than for themselves. The education of such children, physical and mental, needs exceptional care, but needs courage and hope still more.

Bad sleepers in the difficulty of cure come next to those of strong neurotic inheritance.

Unfortunately in prognosis much depends on the means of the patient by which he is enabled to provide for his cure; or for his support if cure be unattainable. Neurasthenia, like phthisis and not a few other diseases, is a costly mischance, not only in the damage it does but also in its demands. The means of cure often include long absences from home under expensive conditions, and, especially in the cases of young women, such measures are always costly and often impracticable. But the outlook is bad for the chronic cases of "disillusioned" patients, mostly well-to-do folk, who drift about haunting spas, collecting prescriptions, and hardening into whimsical habits.

Complications, as with insanity, hysteria, hypochondriasis, arterio-sclerotic detriment, Graves' disease, add of course to the gravity of the prognosis.

Treatment.—It was said by a witty American that in hereditary cases we must begin by treating the patient's ancestors; it might be said of all the cases that we must begin by treating the physician. For in the cure of neurasthenia the "personality" of the physician counts for much. Indeed without this peculiar advantage the best therapeutical schemes may fail. Playfair had this strong personality, this assurance of comfort and cure. Jendrássik, in his broad and sagacious section on treatment, insists upon this factor; as do Erb and Eulenburg likewise. Yet, conversely—although in hysteria personality may, and probably will, suffice—in neurasthenia the most ascendant personality without adequate means will fail. It is important, therefore, that in the particular case the diagnosis be sure whether it be one of neurasthenia only or one mixed with hysteria. Here E. Müller and others are obscure; they insist on the personal ascendancy, but do not discern this element of difference—the element which Babinski, with the French love for language, calls "Pithiatisme," (persuadibility), a state in which, by the way, he is clear-sighted enough not to include neurasthenia. Some neurasthenics are independent of this sensibility, of a "psychotherapy," and may even be annoyed by an assumption of it, lest it should imply some unreality in their complaints. Thus the brusque style is risky; even with a penetrating insight into the patient's character not rarely it fails, if not at once yet after a while, when it will leave him more disillusioned than ever. So it is also with rare or secret drugs, or pretentious apparatus. For, as Jendrássik says, "Every ineffectual cure throws its shadow over subsequent endeavours." However, there is ample room in the art of medicine for personal influence without descent to unworthy pretences, without bamboozling the patient. To pretend operations of a bogus kind is not consistent with the honour of the profession. The words of diagnosis again must be careful; for if the patient, after the manner of his kind visiting many physicians, receives a diagnosis the same in substance yet various in terms, he loses faith, and will not accept what he regards as accommodations of them. For the same reason the patient should not see his physician too often, because he will be craving for changes of remedies, and in every change a comparative failure is implied. Assistants employed in the cure should be forbidden in discourse to go beyond a conventional "patter."

While then the seers of entities will "treat the disease," the rest of us, who are so thick-sighted as objectively to perceive only multitudes of deranged individuals, have to be content to treat them individually. The first interview with the physician should be arranged to give time for full discussion, and, however obvious the case may appear, for a universal, punctilious, and thorough examination. A patient will often express dissatisfaction because the physician did not examine his rectum, or his spine, or his little finger. After this complete investigation the patient must not be allowed to reopen his questionings, but must submit to the prescribed rules and remedies of the system and detail, which likewise must be well thought out beforehand, and then strictly enforced. With a wary, expert, and "disillusioned" patient care should be taken to avoid any prescription or prominent drug which he might recognise as belonging to one of his failures. He is sure to bring his prescriptions with him.

Treatment must be patiently continued till the patient is really well. In neurasthenia, above all diseases, plans ill-thought-out, inconsistent methods, and harkings backward or forward—"planlose Polypragmasie"—are fatal to success. The physician, having left no corner of his patient unexamined, no relevant fact in his story unnoted, and with all the data deliberately ascertained, will map out the plan of the campaign, with the patient's co-operation, on practicable and suitable lines ; and then carry it forward ruthlessly. A moment of hesitation, by forfeiting that confidence and hope of the patient on which the result depends, may jeopardise the whole cure. Jendrássik shrewdly advises us in certain cases "not to begin the cure too soon" ; for, as an attack will at first have its way, the discerning physician will wait, to intervene vigorously when its first brunt is slackening. I repeat the neurasthenic, until staled by disappointment, earnestly desires to get well ; the hysteric is never sure that she does ; and an old campaigner, who has tyrannised over her household for years, is pretty sure she does not. As Dr. Graham Brown says, in his able therapeutical chapter, the only way with her is to empty the theatre, for without an audience the most hardened actor will flag. Yet only too often a hysteric is the pride not only of her family and of the parson but of the whole parish.

Now, as in hysteria the key of the treatment is active impulsion, in neurasthenia it is rest, rest, rest—in all directions, but under such conditions as to avert a cloying or rusty sluggishness. The difficulties of treatment vary with the case and the individual to be treated, but in the majority the course to be followed is definite and fairly sure.

First of all we shall begin by a close searching for any vicious circle of function, and, if found, of cutting any link of it, whatsoever or wheresoever. In one lady patient of mine the stretching of the sphincter ani restored her to herself after months of restlessness most wearisome both to herself and her friends. Or a floating kidney, a disorder of the uterus, a faulty eye, a chronic rhinitis, a "tea-" or "tobacco-heart," or other local irregularity, trivial enough perhaps in itself, may close and perpetuate the vicious orbit of function. This must be the first step in the treatment; and, this being done, in persons of either sex in whom the nutrition of the body is reduced *the Mitchell-Playfair method* will work wonders, and with no little certainty. In many cases of apparent failure, either the case did not belong to this category or the method was not efficiently carried out—a common fault. With this, as with all successful methods, a boom has been succeeded by a petulant scepticism. For methods harden into routine, and the cure is ordered for all cases in the lump; then the reproaches, which are the due of the undiscerning physician, are thrown at the remedy, whereas the failure is often due to the imperfection of the plan, or the want of address in its ministers. For the severer cases, if we are to give any promises of success, it is to be prescribed in its completeness. Now the three legs of this tripod are isolation, full feeding, and massage. Isolation, the withdrawal of the fretful surfaces from contacts, from friends who may be harsh or friends who may be coddles, is a cardinal condition in most cases. Under the fairest seeming there may be some discord in the household, or some sorrow to disturb the air; in a gentle and happy family there may be a jarring note which the most watchful of family physicians cannot perceive, and which the patient would rather die than confess. When the nerves are quivering, the tenderest and most affectionate friends may cross each other, perhaps by over-zealous solicitudes, perhaps by an untuneful voice, perhaps by a tiresome kind of talk. But isolation also has its difficulties and faults, such as to be shut up in a stifling or a noisy room in a squalid city instead of awaking each morning to light, beauty, and peace. Some persons cannot be reduced to a vegetative state, and solitude is intolerable; those who can bear it do better, the others must be allowed some insipid reading—not newspapers, which mark time too exactly, and may contain disquieting news. Visitors, especially kinsfolk and sympathetic friends, even letters, should be forbidden if possible. Affairs should be put in the hands of an agent. No case of any severity can be cured in the midst of the fuss and fidgets of home by the family medical man assisted by an ordinary rubber. Yet we find one patient submitted to massage without isolation; and to another massage is applied without that rest which is a condition of its use, for even a healthy person would find it hard to undergo daily massage and, concurrently therewith, to go about his ordinary work. The massage is as much in the day as a weakly woman can bear in the way of exertion, mental or bodily. We have to remember that a neurasthenic patient is usually made worse by efforts to fight against the debility, whereas in hysteria such efforts are beneficial. Unfortunately, the former patients are ready, often but too ready, to make such efforts, while the latter are not. In traumatic cases, especially if a blow has fallen at all smartly upon the head, absolute rest in bed is imperative for ten days at least, and if the blow were a severe one for a proportionately longer time; for we cannot be sure of the absence of some disturbance of the encephalic fluids, of punctiform lesions, or of a wider jar of the brain substance.

As the Weir Mitchell method is especially effectual in cases of loss of flesh or where, if this be inconsiderable, there is yet reason to suppose that the processes of nutrition are languid and perhaps perverted, the feeding has to be calculated on a special scale; having regard, of course, to the needs of each case, and the wide differences of individual needs. To calculate the food in grams is pedantic. Four or five pints of milk are usually added to the daily rations, beginning with five ounces every three hours. Some persons who dislike mere milk can consume it well enough with cocoa or rice, or as "white soup." As neurasthenics are intolerant of alcohol, as they often are of tea and coffee and drugs, it is better forbidden altogether. It is difficult to find rubbers endowed by nature with the refined physical qualities necessary for their art, and with the temperament necessary for the management of the class of patients they have to deal with. It is not for any shopman or housemaid who is tired of service to "take up massage." These difficulties, and the inevitable expense of the method, are a great embarrassment to us in dealing with cases of neurasthenia in persons not well-to-do. Even if a cure could be absolutely guaranteed, it is a great responsibility to press the expense upon persons of small means. An inexperienced or inattentive operator, and there are only too many of them, or one of coarse, dictatorial, or unsympathetic temper, may so annoy the patient as to spoil the whole thing. Of whatever social degree, a successful massor (if I may coin a word we stand in need of) of either sex must have agreeable manners, a peculiar form of hand, a delicate touch, and no inconsiderable technical knowledge; these, together with muscular strength and endurance, and a cheerful, wholesome, hopeful temper. It were to be wished that massage could be performed always by medically qualified persons. Few operators in my experience have been so successful as medical men and women. Abdominal massage and local applications of the method seem especially to need medical training. Unguents should not be used for the hands, but a dry powder. So far as we can see, then, the method must be an expensive one. However, if the cost can anyway be met it is cheaper to be well than ill, for the interminable expenses and losses of illness mount up in time to no less considerable sums.

In a certain class of cases, namely, in neurasthenic and emaciated persons, not hysterical, a cure by the Mitchell method, if skilfully performed, may almost be promised; moreover, the cure in most of these cases will abide unless some new cause of stress overpower the patient again; no other can approach the Mitchell method in the comparative certainty and permanence of its results. But these are by no means all the patients we have to treat. Many of our patients have not fallen into neurasthenia by way of innutrition, but by overwork or care;

or by insomnia or hereditary proclivity. For instance, in one of my cases of neurasthenia, attended with insomnia and incapacity for thought and attention, the patient, being a good horseman, amused himself in the enforced quiescence of his eager mind by riding matches with his friends. Such persons may try the Mitchell treatment if they have the money to spare, but there are other and better means.

Cases of this last kind are more frequent in men; neurasthenia in women is rarely attended with good physical health, although many neurotic women are far from emaciated; many are fairly well nourished, some are fat. Fat neurasthenic women are not always unfit subjects for the Mitchell method, but they must be placed in very careful hands; first the fat, which may be a sign of sluggish metabolism, has to be "rubbed off," after which process rebuilding on a more solid foundation is commenced; the process is a difficult one, to be entrusted only to very skilful persons, and it is not very certain in its results. Irritable heart is no bar to it; indeed rapid action is calmed by it. I should add that the Mitchell method answers best in cool weather. It is of no specific value in hysteria, or for the hysterical element of a traumatic case, except in so far as any removal from home and submission to medical discipline are effectual.

In cases in which the abdominal walls are lax, and the splanchnic venous system over-full, Barnard recommended abdominal massage followed by certain exercises; and the adaptation of belts to support the feebly acting walls. Sir Watson Cheyne recommends the "Evelyn corsets" to which a kidney pad may be fitted. For such apparatus the patient must be personally measured. Sir V. Horsley is opposed to any mechanical supports in "strain of the back," as they cause irregular distributions of stresses and reliefs.

For "post-operative neurasthenia," the treatment should, in part, be pre-operative—and then the operation might not be needed. In many cases, as we have seen, some local lesion is keeping up the general disorder, and immediate operation is imperative; but local "congestions," "catarrhs," etc., are often grossly exaggerated by specialists.

Local massage for the cure of local disorders is often very important, as in those cases of intestinal spasm which I have described on p. 758; specially skilled operators are, of course, required for these methods. Internal massage, in my experience, has never proved very useful, while it tends to fasten the attention of the patient injuriously upon particular organs; "uterine massage" is thus as mischievous as it is morally revolting.

Sir V. Horsley has never seen any definitely good result, even of palliation, from the actual cautery to the spine.

Climate.—Residence for some months in the mountains of a dry sunny country comes perhaps next to the Weir Mitchell method in value, and supplements it admirably. The altitude, however, should not be great; it is well to rise gradually from 1000 feet to 2000, and from 2000 even to 3000; but to stop at this limit. A few cases are benefited in very high places, such as the Upper Engadine, but these are in persons not gravely affected; too often such altitudes reduce sleep and exacerbate respiratory or cardiac symptoms. The mountain cures are for summer; in winter the arduous games outside and the hot-air heating inside are bad even for mild cases. The effect of residence at the seaside is very uncertain. Some patients do well there, others become more nervous, and sleep worse than ever; an irritative effect, however, may generally be avoided by removing a mile or two away from the shore where the virtues of the sea-air are not lost. Sea air is better for winter than mountain air, especially if the strand be sheltered to the north and west. I have found the eastern bays of Scotland to offer sheltered and sunny sites not too near the sea, which are suitable in some cerebral and cerebrospinal cases. Wind is bad for neurasthenics. Sea voyages promise less; weather may be dreary, food uninviting, or company tiresome. And some persons dislike the sea as much as others enjoy it. Sea-baths come late in the cure, and should be cautiously commenced, yet in earlier phases of the malady warm brine baths are often of great service. Malvern, Buxton, and Harrogate are restorative airs and have the advantages of special therapeutic means. Dyspeptic and so-called "bilious" persons, speaking generally, do not do well at the sea. In winter the Riviera near the sea is for most cases too irritating, Grasse or Cimiez are more suitable. The warmer English winter resorts are too muggy. Montreux and Meran are good winter places, high and fresh, yet sunny and sheltered. In all these resorts the patient must avoid dancing, bridge parties, and such like excitements. Motoring is said to promote sleep, but "travelling"—what Eulenburg calls "vagabonding about in globe-trotter fashion," with its irregular calls on endurance, its sight-seeing and other excitements, is to be prohibited. It may or may not do for hysterics, moral weaklings, topers, crossed lovers, hypochondriacs, and even for substantially healthy persons fagged by overwork; but it is death to the neurasthenical.

Spas.—So many are the coincident factors it is hard to analyse the effects of mineral waters on the general state; they seem scarcely indicated except for complications, such as rheumatoid arthritis. The irrigation method in use at Plombières, and at Harrogate, appears to be beneficial in mucous colitis. Luchon (about 2500 ft.) seems to deserve its reputation for neurasthenia; the air is bracing and dry yet not too keen, and the bathing system is said to alleviate insomnia. The temperature of its baths (34° C.) seems to be suitable to these cases. Colder douches are given in convalescence.

The *Sanatorium* is successful, I believe, with the patient and methodical German, but it is not for the English patient; in the Liege-Halle, unless his case be a very severe one, he is a "centre of unrest"; he chafes his neighbours and adopts their complaints. Still, in some of them there are excellent features, especially the *Arbeitstherapie*, as practised in Dr. Forel's Institute at Zurich and Dr. Veraguth's at Sonnenberg; namely, graduated gardening, carpentry, photography, nature study, landscape painting. But the method is designed rather for the feeble-minded, the "psychopath" or the bad sleeper than for neurasthenics. If for some of these the method be suitable, it must be after some considerable measure of restoration has been attained; then, away from little social jars or business cares, the convalescent may be led forward from one easy task to another. In institutions there cannot be much individualising, but at certain stages of recovery it is well if the patient's chief complaint is that no one listens to him.

Hydrotherapeutics.—Wet sheets, packs, sitz-baths, and douches are of great value in neurasthenia, and, if judiciously used, in all kinds of it. Simple means, such as the wet sheets or douches, may be used at home, but neurasthenics do not bear cold water very well; a douche or shower bath should begin tepid and cool off. But, as with the massage cure, the judgment with which the method is used, the particular applications and graduations of it in individual cases are essential conditions. It would not be becoming in me to comment upon hydropathic establishments either at home or abroad; the medical attendant must make his choice on the facts and information within his reach; but in general terms I may say that in Germany these institutions are managed on more scientific principles and methods and with more serious purpose than with us. However, some German authors mock at their "pompose Einrichtungen." Eulenburg recalls to our memory Chapman's icebags, saying that both hot and cold are very useful. He applies them for some length of time. Locally, for the "neurotic heart," he also finds them efficacious.

Electricity may be applied in one way or another with considerable advantage, but again much depends on the tact, confidence, and experience of the operator. Of the application of the continuous current to the head or spine in cerebral and spinal cases I can say little: I have never had time to apply it myself for any length of time, nor is the clinical evidence in its favour as yet very strong; however, the method has ardent supporters, and in mixed cases may bring some relief by "suggestion." Savill advised "descending spinal galvanism" daily, or three times a week. It will, of course, form but a part of a more extended treatment prescribed according to the other requirements of the case. Of more service in my experience is the faradic bath, the use of which is described in the first volume (p. 458). Used for about twenty or thirty minutes, not so far as to produce muscular spasms but a tingling of the skin, it seems to soothe the nerves, to bring sleep, to improve the appetite, and to mitigate muscular debility and weariness. By local application faradism is said to arrest the local sweats of neurasthenics, as of the hands or feet. Of the application of the continuous current to the neck in cardiac cases I can speak also with some encouragement. These processes can be carried out at home without difficulty, and the family attendant after a few sittings may be able to entrust the operation to others. At best, however, the part of electricity in the cure of neurasthenia seems unlikely to be more than subsidiary; it is of more effect in hysteria. Erb, I see, is of the same opinion.

The electrostatic or Franklinic method in simple neurasthenia, in the practice of those who are best able to judge of its value, is not gaining ground. Of high-frequency currents in neurasthenia, we have as yet but little matured experience. It is said to relieve insomnia when without obvious cause, such as pain or other distress.

Hypnotism.—In neurasthenia, as we have seen, the fault is not in sectional abeyances but in penury of store, and cure is not a matter of readjustment but of replenishment. The machinery is working at low pressure. In hysteria there is store enough if only a better equilibrium can be brought about; but in neurasthenia no more is to be expected from personal influence than that relatively insensible reinforcement by which we all help each other. When, therefore, we speak of the ascendancy of the physician as an important ingredient in the cure, we do not seek from him miraculous influences, but only a reviving breeze, a fanning of that glow which is always radiating from one to another, and by which we reinforce each others' energy. My experience agrees then with Ballet's, that hypnotism, or suggestion without hypnotism, effective as it often is in hysteria, is of little use in neurasthenia. It can readjust an unbalanced nervous system, it cannot create its energy.

Suggestion may, however, be incidentally useful in sleeplessness, in phases of irritation, and especially in relief of some morbid mental proclivity or obsession. Again for the often considerable hysterical elements in traumatic cases, as for anaesthetic and paretic hysteria generally, it is more effectual. Brusque methods, which may answer in hysteria, are out of place in neurasthenia.

Music.—We are apt perhaps to forget this influence, so potent in Saul's case, and in that of many another great man, fretful and sleepless, down to Bismarck, whose wife was wont to calm his nervous excitement, and to soothe him to sleep "by playing one gentle air after another."

Drugs are not to be forgotten in the treatment of this malady, although their part is but subsidiary. In relief rather than in cure their place is to be found; and, within this sphere, many drugs are of great service. Constipation may be much ameliorated or disappear under skilful massage. The bromides, from an extreme of excessive adminsitration, have now fallen into discredit, yet we should do ill without them. The secret of success with the bromides is to use them in small doses and for brief periods of time. It is said that while taking them table salt should be avoided. Erb finds a combination of bromides with codeine to be useful. The bromide of strontium is said not to produce acne. Their place is chiefly, of course, in the management of cerebral cases, or of the cerebral symptoms in any case. In spinal cases with leg weariness their use is very restricted. In sleepless and fretful persons the bromides are effectual, and my custom is to prescribe, not large doses at bedtime, but not more than five grains of one of them with a little hydrobromic acid and a grain of quinine, or again with bismuth, thrice daily. Strychnine, unless in very small doses, is rarely appropriate in neurasthenia, and its use should not be long continued. Physostigmine has lately been recommended by Winquist, a Finnish physician, in doses of 0·3 to 0·6 mgrm. once a day, in a powder with magnesia or sugar. It is said to be of no use for mental (melancholic) conditions.

In gastric cases arsenic is sometimes invaluable; it must be used with care, of course, but for a period of some weeks' duration; the doses are gradually increased until five drops of Fowler's solution are taken with each of the three principal meals. A coated tongue is no bar to its use. Many gastralgic cases of long standing are cured by arsenic. The medicine is useful also as a general "tonic," as Dr. M. Clarke also testifies. If the stomach be dilated lavage may be required occasionally or for a short time; but the patient—in ordinary circumstances—should not be allowed to make a habit of it.

In cardio-vascular cases also arsenic is useful; digitalis is rarely helpful, and the nitrites are mischievous. Cactus (as "cactina") calms the heart; so, as I am told, does adonis. In a few cases small doses of strychnine over a long time are efficacious. In cardiac cases flatulence should be carefully prevented.

The hypophosphites or "glycerophosphates" of sodium and lime have seemed to me to be of service in some cases. By certain recent writers the formates are preferred; a dram to a dram and a half of a 50 per cent solution of formic acid is diluted with water to a pint, and sweetened; one half is taken before breakfast, the other half before luncheon; or the draught may be taken in effervescence with bicarbonate of sodium. Of formate of sodium 45 to 60 grains may be taken daily in divided doses. Iron is often beneficial, presumably in the more pallid and ill-nourished patients; it is at any rate in these that we have more experience of it.

The action of the bowels must be gently but regularly maintained. Massage usually suffices for this. A little calomel occasionally is beneficial, but purgation, especially by salts, does harm. Sulphur, liquid extract of cascara, syrup of senna, aloes, and belladonna are the kinds of laxative required. A dose, however small, must be taken daily, if such means be required. To allow the bowels to close and then to force them with larger doses is bad practice. On unsuccessful mornings—and the morning habit should be maintained—a glycerin enema or suppository is a useful auxiliary. In cases of vasomotor relaxation ergot is said to be of service; in vaso-constrictory cases, on the other hand, the nitrites seem to be ineffectual; indeed, even in "pseudangina pectoris" they give little help.

Valerian is an old-fashioned remedy too often forgotten in neurasthenia. In cerebral cases, with loss of memory, vertigo, vague sensations of incapacity or confusion, acute obsession, and the like, it often gives relief; as also in visceral discomforts and perturbations. It has been administered in the form of the ammoniated tincture in liberal doses, but concentrated forms, recently introduced by Merck and others under the names of bornyval, gynoval, valyl, are said to be more efficacious, and are certainly more convenient. Of these during periods of distress 2 grains may be given every half hour. Klemperer says valyl (2-3 capsules of 2 grains each) with a little hot milk relieves "nervous insomnia." All preparations of valerian are given on the full stomach, to prevent unpleasant eructations; few patients object to it if they are warned beforehand that it is as valuable as it is nasty; indeed, I often think that neurotic patients are especially tolerant of valerian, galbanum, and asafetida, or find compensation in the comfort they give. There are, of course, many other drugs of subordinate value which I need not enumerate.

Opium cannot be wholly set aside because of the perils of its abuse; the physician, keeping the drug in his own hands, and prescribing it under cover of such names as Dover's powder or soap pill, may avoid the peril and secure invaluable aid. In recurrent pain, as for instance in digestive intolerance, a pill containing a fractional dose of opium administered every four or five hours is a means we could ill spare. Opium thus carefully used is often helpful against the "dreads" and depressions also. I need not say that the opium is to be suspended as soon as possible, and codeine may occasionally take its place.

Cocaine often gives a sense of well-being which for the moment is cheering, but the effect is transient, and is heavily paid for in the reaction which follows. Hyoscine is occasionally needed, in cases of mental agitation. For the relief of nervous pains phenacetin is perhaps the best remedy of its class. Camphor monobromide is said to be helpful, especially in pelvic irritation and nocturnal pollutions; in these cases it is best administered in the form of suppository. Drugs to relieve impotence, real or imaginary, must be avoided; useless at best, they may be injurious. Caffeine relieves headache, but is rather an irritant to nerves on edge, and must never be used in cases of insomnia. Local aches (myalgias ?) are often much relieved by an adjustable air-pad with a belt or brace fitted to the individual.

Headache is the most obstinate and harassing of all the symptoms of neurasthenia, and is often very wearing. Minute investigation of the eyesight will be made, if only for fatigable accommodation or the strain of excessive glare. And "eye-strain" may be an important factor in cases of which headache is no considerable feature. Occipital headache is often muscular and alleviated by support to the back of the head.

Organotherapy has so far proved useless.

Soporifics in many cases, perhaps in most at some critical period, are inevitable, but are to be used with the greatest precaution, and never entrusted to the patient himself. All other means of procuring sleep must be tried first. Narcosis and sleep are not identical; Verworn's and Waller's researches suggest that in narcosis while catabolism may be unchecked repair is retarded. The physician must assure himself from independent testimony that the sleep is as defective as is alleged, for to the patient two or three dreary hours of nocturnal vigilance seem interminable. According to Jendrássik, and Eulenburg agrees with him, neurasthenics need less sleep than healthy persons ("ihr wirkliches

Schlafbedürfnis oft auffallend gering ist"), and are little the better for long spells of sleep. I think there is some truth in this. The evening diet must be minutely prescribed, and be free, of course, from tea and coffee. Tobacco, in sparing use, may be regulated by individual habit. Occupation of an evening must be amusing, but not exciting to either intellect or emotion. Patients who awake and lie awake at 3 or 4 a.m. will often find that to take a little food, left at the bedside overnight, will be not only a comfort, but a restorer of sleep.

Some patients who lie awake after 3 a.m. recall some slumber by standing up and performing gentle arm and leg exercises for a short time. A bath at 90° F. for ten minutes, or half an hour's "pack," at bedtime may compose to sleep (*vide et* "High Frequency," p. 783). Aschaffenburg says that to secure success the bath may have to be much prolonged, after preliminary trial even to 1-2 hours. This may be done, he says, at 34°-35° C., without debilitating effect; and the patient may read a storybook or newspaper while in the bath. In some cases a cold water bandage on the brow is sufficient. In palpitation cases a mixture of digitalis with a bromide may be effectual. The oldest and most cautious of physicians are driven to prescribe hypnotic drugs, even audaciously, in cases in which, for lack of sleep, the patient is frantic or in despair, and his friends are at bay. Happily we are armed with new and various soporiferous drugs. Veronal must not be pushed beyond 5 or 6 grain doses, as in larger quantities its toxic effects increase in greater ratio than its soporific. Chloral has, I think—at any rate in cases of sound myocardium—been too readily laid aside for more novel claimants; but its use should be restricted to critical moments, for it comes nearest to opium in creating a habit. On critical occasions, as I have said, our practice has to be audacious. I remember a stubborn case of Dr. Colin Campbell's in which, after failing with chloral, nepenthe, and sulphonal, at his wits' end he administered 60 grains of trional in hot draught stout on three consecutive evenings: on the first it gave six hours' sleep, on the second eight, on the third nine hours and a half; and thereupon the patient turned the corner, and the case became manageable by simpler means. Such practice in careful hands is justified, because its purpose is, by a more decisive but temporary interference, to avoid perpetual drugging. Reluctantly, and as a last resource, we have seen that morphine may be unavoidable. "Hypnotism," valuable in hysteria, and appropriate therefore in many traumatic cases, in simple neurasthenia has been in my experience of little effect.

Diet.—Into the large question of dietetics I need not enter except to indicate certain special applications of the subject. In cases of atonic dilatation of the stomach the diet must be regulated as directed in the article on these cases. Again, in cases of disorder of the digestive tract, as in that mentioned (p. 758), the diet will receive the proper attentions. But we must not be meticulous in these respects, for a dirty tongue may mean indolent rather than vitiated digestive functions, and a decided, however cautious, enlargement of the diet is often justified by results. For it must not be forgotten that in many cases—in most, if we except the strong and well-nourished men with good appetites to whom I have already referred—our chief indication is to push feeding as far as the patient can be made to assimilate it, farther than many patients willingly accept it. This rule applies more to women, many of whom have been shirking food for years. All faddy diets should be discouraged. In health women need food more frequently than men do; we all know that the secret of making an excursion go off well is to see that the women have frequent meals, however small. Anorexia nervosa is discussed elsewhere (Vol. III. p. 403). Even in gastric cases then a cautious advance to a more liberal diet is generally to be attempted from the outset of treatment, although many of these patients have spared their stomachs until they will not perform any sufficient work without resentment. Meat should not form a large proportion of the diet; a moderate quantity once a day will suffice, and fish or other nitrogenous foods, such as sanatogen, may be used in part. Carbohydrate and fat foods, and even a well-refined cod-liver oil, or oil and malt, are of most value in meagre habits. For some fat neurotics a diet must be prescribed in which starches and sugars are reduced.

Alcohol is to be allowed very sparingly, and tea and coffee used cautiously and not towards evening.

Exercises are of three kinds: as means of cure, as weaning back to work, as self-management on recovery. We may be surprised to hear on the basis of experiment (53), that change of work is not rest; although it may be some refreshment. During the first stage of cure the massage takes the place of voluntary effort; but in a forward stage the patient must learn to exert himself again, to re-educate his will, and to test his acquired endurance. These efforts must at first be most carefully graduated, because to trespass beyond the powers of the patient is inevitably to throw him back, and the up-hill task has to begin again. To rush back into full life will waste much of what has been won; still some little work must be tried, even if slightly against the grain. Objectless exercises—ergostats, Zander tasks, and even formal gymnastics—are, at any rate to Englishmen, irksome. And the Oertel methods are rather empty in this respect unless enlivened by other interests. Walking on the level is too uniform a movement; by its attitudes and uniformity it tires the joints and ligaments. And, as Ballet says, the exercise should be such as, without stress, to put the patient slightly out of breath, so that his lungs may be filled. Gentle climbing in such an upland as recommended on p. 780 does well, especially for "cerebral" cases. Boat sailing may be recommended in fine weather, so long as the patient is not called upon for any quick or unexpected efforts. The same conditions apply to riding. The arts, such as music, easy lectures, and unemotional books, are useful, even if it be for no more than to pass the time. All medical books, not quack books only, should be eschewed. Whether for cure or re-initiation into life, the ordinary duties of shop or office seem undesirable; confinement and monotony are staling, and standing about and walking on the level, as engaging certain muscles only, cause fatigue. For women cooking should be avoided, even on recovery; but various household occupations of a light kind, not done under stress, are helpful. Jendrássik finds that military camps—not, of course, active service—do good; the regularity of system is tonic, and shyness is worn off by formal comradeship. A military or naval career, however, in his experience, as in mine at Osborne and elsewhere, is for such persons hazardous.

Games must be advised with great discretion, and no game allowed in which the patient has to keep up with companions; the patient must feel at liberty to stop at any moment, for a few minutes of effort beyond his supply of energy may throw him back, and discourage him. Ballet carefully considers the use of games, and, besides the precautions already suggested, dissuades us from advising amusements which make a keen demand upon the attention; he specially mentions fencing as in this respect too exacting.

Whatever the exercises or rest, the treatment should be on the "open air" system; this mode of life, moulded upon that of the sanatorium for the tuberculous, and needing no description here, is of the utmost value in neurasthenia; and to pass the night out of doors, after the novelty of the experience wears off, promotes sleep and well-being. Motoring is said to make people sleepy.

Except during special periods of rest-treatment, every care must be taken to avoid or to avert the relaxing habits of idleness, lounging, and frivolity.

As regards mental application, something depends on the area of the exhaustion; in a "cerebral" case we shall avoid too early a call on the brain. But if the case be one of general neurasthenia it is desirable, for the reasons just given, to order short spells of work—say half an hour to an hour twice a day, and so gradually onwards. If the effort prescribed be too much, and a reaction follow, we may fear lest the patient be discouraged; if it be not unsuccessful we shall gain his confidence: much then depends on the tact of the physician. The results of the study may be nil; but, as in the military discipline mentioned above, to portion out the day instead of letting it dawdle out anyhow, is a bracing change from coddling treatment, and promotes self-respect.

And thus we are led to "psychotherapy"—"*moral education*" or re-education. In the worse stages of the malady the patient is fit for little but repose, and for a month or six weeks may be wrapped in cotton-wool. With replenishment of the capital of the body, however, the time comes for renewed exercise of the will. Hereditary cases especially require this side of our art. Emotional women, who, whatsoever their converse with religious indulgences, may never have been practised in moral discipline, are sometimes very difficult to deal with. Usually they have been cajoled, or bullied and scolded, or, worst of all, allowed their own way. Many of these persons are not neurasthenics but hysterics, but anyhow with them there is above all things the need for the velvet and steel glove ; and the physician must never allow himself to be persuaded to undertake such a case at the home, unless, of course, certain definite guarantees can be given so that he and his nurse may have a free hand without the interference, or even the access, of fond or critical friends. In all these difficult steps from one stage of treatment to another perhaps the best prognostic guides are the degrees and duration of the quickening of the pulse on change of position or on sniffing ammonia (Kraus), and notes of the tendon-jerks taken at various hours. The blood-pressure trials, also suggested for this test, are too imposing and too capricious for highly-strung persons.

Adults are so often fixed in habits, good or bad, as to offer less advantage for moral education than young people. On this part of treatment Ballet writes well. In all we do, whether with adults or children, we must beware of putting notions into the patient's head. In local treatment we must beware of creating an "obsession." We must never let a child hear that he is of nervous temperament and must cocker himself accordingly. Children are to be watched by their seniors reticently, for the very docility of a child has its dangers ; so that we must avoid giving the child, or the childish adult, the "formula of his defects," lest he act up to the character. Children must not think or dream, they must be made to do things with their hands, while we give them confidence, hope, and all deserved promotion, and point out the ways of improvement.

Marriage.—Eulenburg's article, if in some parts verbose and rather "cataloguish," is interesting, and on marriage particularly he is wise and helpful. He cautions us not to forget the bolts and suicides on marriage, or the eve of it ; whether in fear of impotence, or in vaguer dreads. He is quite clear that if the patient admits such apprehensions the engagement should be at least postponed, if not terminated. If postponed, the interval must be a long one and occupied by a thorough course of treatment and change. If, however—in man or woman—marriage be agreeably brought about, amendment often follows ; not merely by coitus nor only by child-bearing, but by new interests and continual serious occupations. I may refer the reader to a little book on this subject by Senator.

Suggestion and the personal ascendency of the physician enter so largely into all methods of treatment that, as I have said, we find great difficulty in making much way with old disillusioned patients. The only chance with them is, in some institution, to suppress their sophistries by a strict and imposing system of life which they cannot elude.

CLIFFORD ALLBUTT.

REFERENCES

1. ABUNDO. "Stati nevropatici consecutivi al terremoto in Sicilia," *Riv. ital. d. nevropat.*, ii. 2.—2. AMBERG. Kräpelin's *Arbeiten*, 1896.—3. ARCHER, W. *America of To-day*, Lond., 1900.—4. ARNDT. *Die Neurasthenie, ihr Wesen*, etc., Wien, 1885

(good historical introduction).—5. Aschaffenburg. "Die psychasthenischen Zustände," in Curschmann's *Lehrbuch d. Nervenkrankheiten*, Berl., 1909.—6. Bailey, P. *Diseases of the Nervous System due to Accident and Injury*, Lond., 1906.—7. Ballet, G. *Neurasthenia* (translated by P. C. Smith), Lond., 1908.—8. Barnard. *Lancet*, 1899, i.—9. Béni-Barde. "Clinique hydrothérapique," Masson et Cie. (especially on Neurasthenia), Paris, 1908.—10. Binswanger, O. *Die Pathologie und Therapie der Neurasthenie*, 1896 (a valuable treatise).—11. Bourcart. *Compt. rend.* X^{me}. *Congrès français de méd.*, Genève, Sept. 1908.—12. Bury, Judson. "Trauma and the Nervous System," *Brit. Med. Journ.*, 1904, i. 997.—13. Clarke, J. M. *Hysteria and Neurasthenia*, Lond., 1905.—14. Cramer, A. *Die Nervosität*, etc., Jena, 1906 (hysteria and neurasthenia well distinguished, as also their treatment).—15. Dana. "The Partial Passing of Neurasthenia," *Boston Med. and Surg. Journ.*, 1904, cl. 339.—16. Dent, C. T. *Trans. Med. Soc.* (Annual Oration), 1908, xxxi. 331.—17. Dercum. F. X. "Hypochondria (differential diagnosis from neurasthenia, etc.)," *Phila. Med. Journ.*, Feb. 8, 1902.—18. *Idem. Textbook on Nervous Diseases*, Phila., 1895.—19. Drummond, D. *Brit. Med. Journ.*, 1906, ii. 11.—20. Eichhorst. *Handbuch*, 6th ed., 1907, etc.—21. Eppinger u. Hess. "Pathologie d. vegetativen Nervensystems," *Ztschr. f. klin. Med.*, Berlin, 1909, lxviii.—22. Erb. *Therap. d. Gegenwart*, Berl. u. Wien, 1907.—23. Eulenburg. "Neurasthenie Behandlung," *ibid.*, Aug. 1908.—24. Fales, L. H. "Tropical Neurasthenia," *Amer. Journ. Med. Sc.*, Phila., 1907, cxxxiii. 582.—25. Goldscheider. Quoted Virchow's *Jahresbericht*, 1906, 210-11.—26. Graham-Brown, J. J. *Treatment of Nervous Diseases*, Edin., 1905.—27. Guthrie, L. *Brit. Med. Journ.*, 1906.—28. Hartenberg. *Psychologie der Neurasthéniques*, Paris, 1908 (said to give a new conception of neurasthenia ; it is written with French lucidity and logic, and is pleasant reading ; but this seems to be the only novelty).—29. Herman, G. E. *Clin. Journ.*, 1908, xxxiii. 177.—30. Hirsch. *Beiträge z. Erkenntniss u. Heilung der Spinalneurosen*, 1843 (good clinical descriptions, bad nosology).—31. Horder, T. J. *St. Barth. Hosp. Journ.*, 1903, x. 67.—31*a*. Hurry, J. B. "Vicious Circles in Nervous Disorders," *Lancet*, 1910, i. 1197.—32. Jendrássik, E. "Über Neurasthenie," *Samml. klin. Vortr.*, Leipzig, 1906, n.F., No. 426-427.—33. Knapp. "Traumatic Neurasthenia and Hysteria," *Brain*, 1897.—34. König. *Arch. f. Augenh.*, Wiesb., 1890, xxii.—35. Kräpelin. *Münchcn. med. Wchnschr.*, 1902, xlix. 1641.—36. Lebeaupin. (Entero-colitis and Muco-membranous Colitis), *Journ. de méd. et de chir.*, Paris, 1906, lxxvii. 853.—37. Leubuscher u. Bibrowitz. *Deutsch. med. Wchnschr.*, 1905 (neurasthenia in the working classes).—38. Löwenfeld. *Die psychischen Zwangerscheinungen* (Obsessions), Wiesbaden, 1904.—39. Löwenfeld, L. *Pathologie und Therapie der Neurasthenie und Hysterie*, 1894 (an able study of hysteria as well as of neurasthenia, with a bibliography of both diseases).—40. Lubenau. "Herzkrankheiten in d. Berlin. Arbeiterbevölkerung," *Ztschr. f. klin. Med.*, Berlin, 1906, lx. 134.—41. Maguire, R. *Proc. Roy. Soc. Med.*, Lond., 1909, iii. (Med. Sect.) 1.—42. Martius. *Neurasth. Entartung einst u. jetzt* (pamphlet, 44 pp.), Leipzig, 1909 (a wholesome protest against the degeneration bogy).—43. Morris, C. A. *Lancet*, Lond., 1901, ii.—44. Mott, F. W. *Ibid.*, 1909, i. 1664.—45. Müller, E. "Behandlung d. Neurasthenie," *Deutsche med. Wchnschr.*, 1909, xxxv.—46. Müller, F. C. *Handbuch der Neurasthenie*, 1893 (this handbook is written in co-operation by nine contributors, is full of information, and especial attention is given to hydrotherapeutics ; it contains a full bibliography.—47. Nägeli. (Report on Cases of Traumatic Neurasthenia), *Deutsche med. Wchnschr.*, Dec. 16, 1909.—48. Page, Herbert W. *Injuries of the Spine and Nervous Shock*, 2nd ed., 1885.—49. *Idem. Railway Injuries*, 1891.—50. Petrén. "Verbreitung d. Neurasthenie unter verschiedenen Bevölkerungsklassen," *Deutsche Ztschr. f. Nervenh.*, Leipzig, 1900, xvii.—51. Rankin, G. *Brit. Med. Journ.*, 1906, i. 492.—52. Rivers. *Influence of Drugs on Fatigue* (Croonian Lectures R.C.P., 1906), London, 1908.—53. Rivers and Kräpelin. Kräpelin's *Psychol. Arbeiten*, 1896, Bd. i.—54. Rivers and M'Dougall. *Brit. Journ. Psychol.*, 1905, i.—55. Ross, H. C. *Proc. Roy. Soc.*, Dec. 9, 1908.—56. Russell, R. "On Tendon-Jerks," *Clin. Journ.*, Lond., 1902, xx. 369.—57. Savill. "Hysteria," *Lancet*, Lond., 1909, i. ; and *Neurasthenia*, 4th ed., Lond., 1908.—58. Schofield and others. *Brit. Med. Journ.*, 1901, ii. 1236.—59. Smith, P. C. "Neurasthenia, Degeneracy, etc.," *Brit. Med. Journ.*, 1903, i. 781 ; 1906, i. 494.—60. Specht. (In Kräpelin's Laboratory) "Clinical Measurement of Fatigue" (well translated from *Arch. f. d. ges. Psychologie*, 1906, ii. Hft. 3), in *Journ. Ment. Sc.*, July 1906 and July 1907.—61. Stiller. *Die asthenische Konstitutionskrankheit*, Stuttgart, 1907.—62. Stilling. *Physiologische, etc., über die Spinalirritation*, 1840.—63. Terrien. *Progrès méd.*, 1906, 3. s., xxii. 33 (rural and urban "psychoneuroses"), summarised in *Lancet*, Feb. 10, 1906.—64. Upson. "On Dental Causes of Neurasthenia," *Cleveland Med. Journ.*, Aug. 1909.—65. Warner. "Lectures on Mental Hygiene," *Clin. Journ.*, Lond., 1909, xxxv. 113. Article in vol. viii. of the first edition of this *System*.

On **Visceroptosis**, Sir W. Watson Cheyne's article in the *Lancet*, 1906, i. 947; Professor Keith, *Lancet*, 1903, i. 631, 709; and articles in this *System*, Vol. III. 860, Vol. IV. Part I. 11.

C. A.

DISORDERS OF SLEEP

By Prof. J. B. Bradbury, M.D., F.R.C.P.

Insomnia—Somnolence—Dreams and Nightmare—Sleep-talking—Sleep-walking

Physiology.—It is unnecessary in this article to enter into a discussion on the physiology of sleep. Certain symptoms—diminished metabolism, shallower respiration, less frequent pulse, diminished secretory activity, and loss of consciousness—are recognised as constant accompaniments, but a satisfactory explanation of the condition is not yet found. There are several hypotheses as to the causation of sleep. It has been attributed to cerebral anaemia; to chemical changes in the brain cells or neurons, such as an exhaustion of their intramolecular oxygen, or an accumulation of fatigue-products; to a contraction of the dendritic processes, and a consequent break in the transmission of nervous impulses; to an expansion of the neuroglial cell processes insulating the nerve-cell processes, and producing the same effect; and to a purely psychological condition, namely, loss of consciousness apart from any physical or chemical change. The last explanation is simply a cloak for our ignorance. The most probable hypothesis is that of an altered metabolism of the cerebral cells dependent upon exhaustion and diminished influx of stimuli.

Anaemia, or relative anaemia, is an important factor, according to many authors the most important. As the vascular contents of the cranial cavity must remain practically constant, any loss of blood in the arterioles and capillaries must be compensated by some increase elsewhere. According to Cappie, this occurs in the pial veins; according to Howell, in the veins at the base of the brain. In any case there is a relative anaemia—a diminished blood-flow through the cerebral cortex, and consequently a diminished metabolism and depressed function; but the bearing of this upon the production of sleep we do not know. Howell believes it is important. From plethysmographic observations on the cutaneous circulation, he was led to suppose that exhaustion of the vasomotor centre is the all-important condition; but this opinion has been strongly criticised by Dr. L. Hill. As far as we can see, neither the circulatory, nor the histological, nor the psychological hypotheses are sufficient to account for sleep; and, with the exception of the circulatory, none of them bears any practical significance. The chemical hypothesis, on the other hand, is of direct importance, as it explains in some measure why, in order to induce sleep, it is necessary to rid the organism of fatigue-products and toxins.

Amount of Sleep.—The amount of sleep required by a healthy person depends upon his age, the quality of the sleep, and his idiosyncrasy. A baby sleeps the greater part of his time; as the child grows up less sleep is needed. Dr. T. D. Acland has lately drawn attention, and rightly so, to the insufficient amount of sleep which boys in some public schools obtain. Ten hours, at least, of undisturbed sleep should be procured for the younger boys—age thirteen to sixteen—otherwise they may become imperfectly developed both physically and mentally (*vide* Vol. I. p. 175). Between sixteen and twenty-five years of age eight hours should be allowed; afterwards the amount must depend upon the work and requirements of the individual. Some people can do with from four to five hours; and others with even less; for the majority of people, however, from six to seven hours are usually enough. Idiots and persons of weak intellect often sleep excessively, and even in more normal individuals a condition of abnormally prolonged sleep is occasionally recognised. The question of sleep in the aged has been dealt with in Vol. I. p. 207. When sleep is profound and continuous, less is required than when it is broken and superficial.

INSOMNIA.—By this term is meant loss of the normal amount of sleep. It includes all grades, from simple restlessness to total loss of sleep. From what has been said it is obvious that the term is comparative; that what is sleeplessness in one person is not so in another. Furthermore, the kind of sleeplessness varies. Some people go to sleep directly after getting into bed, but awake every hour for several hours, sleeping well the rest of the night; others, especially the gouty, awake punctually at three or four, or earlier, and are unable to sleep again, or, at any rate, until it is time to get up; others again find great difficulty in getting off, but, once asleep, do not awake until the morning. Some persons, as we have said, do not sleep at all, and these cases are of serious omen. It has been stated that sleep is more necessary than food; that animals die sooner from sleeplessness than from starvation; yet patients occasionally assert that they have not slept for weeks. In most cases this is probably illusive: they sleep without realising it.

In general, sleeplessness cannot be considered a disease in itself; it is, however, an important symptom of many diseases, and in certain cases, especially of children of abnormal nervous excitability, of neurotic women, and overtired men, it is the predominant one. As the essential feature in the treatment of this condition is to discover, and, if possible, to remove the cause, it will be convenient to consider insomnia with regard to (*a*) its causes, (*b*) its treatment.

Causes.—These are many, hence it is necessary to adopt some principle of classification. This has been done by most authors, but no system has yet received general acceptance. Germain Sée recognised nine divisions: dolorous, digestive, cardiac and dyspnoeic, cerebrospinal and neurotic, psychical, insomnia of cerebral and physical fatigue, genito-urinary, febrile, and toxic. Others admit only four or five. We shall adopt a division into four. But it must not be assumed that all causes can be uniformly arranged in these classes; in some cases the immediate cause will fall under two or more divisions. Such divisions are of course only justified for descriptive purposes.

1. *Irritative Causes.*—This class includes all forms of insomnia caused by pain and like uneasiness. In children, teething, indigestion, especially flatulence, and worms are frequent causes. Eye-strain, as in hypermetropia and astigmatism, the irritation of eczema, faucial adenoids, and other similar conditions, are also etiological factors of insomnia. After surgical operations, even in the absence of pain, insomnia is not infrequent; this may be due in part to the constrained position of the patient and some irritability of the wound, in part to toxaemic and psychical causes. Cold feet, vesical affections, asthma, bronchitis, and other diseases accompanied by troublesome if slight irritation, for example, cough, or pruritus (local and general), also fall under this head. But the most important causal factor of this group is undoubtedly pain. Of all diseases accompanied by pain, sleeplessness is a symptom. The insomnia of all or many of these may involve other etiological factors, but the pain is the predominant one, and that which necessitates treatment.

2. *Toxic Causes.*—Diseases due to the presence of some toxic agent in the blood, or accompanied by it, are often attended by troublesome insomnia. Alcoholism and nicotism, the exanthems (measles, small-pox, scarlatina), enteric and other continued fevers, erysipelas, diphtheria, pneumonia, influenza, hepatic disorders, dyspepsia and other gastric and intestinal disorders, rheumatism, gout, and Bright's disease are some of the conditions which fall under this head.

The insomnia accompanying heart disease and altered vascular conditions (high and low blood-pressure, arteriosclerosis, etc.) may also be included in this division, as their origin in most instances appears to be a blood poison. The sleeplessness induced by certain beverages, such as tea, coffee, and cocoa, requires mention here. These, especially if taken strong and late at night, are in most people powerful preventives of sleep. Strychnine and other nervine tonics, when taken regularly, often cause sleeplessness during the earlier part of the night; but in certain conditions of exhaustion, by improving the circulation and other functions, they prove excellent hypnotics.

Insomnia is also a symptom of certain drug habits, such as opium-eating, morphinism, and cocainism.

3. *Psychical Causes.*—Grief, shock, worry, and mental anxiety are

among the most frequent causes of insomnia. In many persons a disposing factor—a nervous temperament, neurasthenia (*vide* p. 763), hysteria, hypochondriasis—also exists, and among such persons insomnia may easily be established as a habit. In students, overwork, especially if accompanied by irregularity in retiring to rest and the prospect of an examination, are the most frequent causes. Women at the menopause often suffer from depression and insomnia, partly, perhaps, owing to the accumulation of toxic products not eliminated by the catamenia. The various forms of insanity—such as mania, melancholia, general paralysis—are accompanied by insomnia: in some cases it appears as a premonitory symptom; although there can be no doubt, on the other hand, that continued loss of sleep is also a factor in the production of mental aberrations. Cases of severe chorea and of paralysis agitans are also marked by sleeplessness.

4. *Causes arising from Change in the Mode of Life.*—Eating late dinners by those unaccustomed to them, and change of climate, especially to high altitudes, or in some individuals even to the seaside, sometimes give rise to temporary insomnia. Nurses who have been on night duty, after changing to the day frequently suffer from sleeplessness; and the same condition occurs in other people with intermittent nocturnal occupations. Sometimes very simple changes in the mode of life of individuals will lead to insomnia. A patient of mine and his wife both slept very badly for the first three months after beginning to cycle. This may have been due to excessive fatigue, or possibly to increased waste-products in the blood, or to both combined.

Pathology.—Upon this subject we know very little; and so long as the physiology of sleep is ill understood, the pathology must remain unknown. As we have seen, the condition of the circulation and the composition of the blood are important factors in the production of sleep, and when the brain cells are improperly supplied with blood (as in anaemia and heart disease), or with impure blood (as in the various toxaemic diseases), we can readily understand that the nutrition of the nerve-cells is altered, and that some deviation from normal sleep must occur.

Treatment.—This naturally falls under two heads—general or non-medicinal, and medicinal; although these cannot well be separated in practice. In all cases the first thing to look for is the cause, so that, if possible, this may be removed. If it be an indigestion, this should be treated. Acid dyspepsia and that attending gout are frequently accompanied by insomnia. In these cases, careful attention to the diet, the avoidance of tea and fruit (cooked and uncooked), and of all substances containing vegetable acids, except in so far as these may be used in treatment, are essential. Fruit, owing to the acid it contains, is, I believe, an unsuspected cause of indigestion, gout, and sleeplessness in many persons: so also are acid wines. For gouty persons much butcher's meat, especially beef, should be prohibited, and white fish, poultry, and game ordered in its place. Fried fat, such as that on the outside of fried fish, should also be avoided. An alkaline bitter before meals, an occasional mercurial at bedtime followed by a saline aperient the next morning, and, for the gouty, colchicum or sodium salicylate should be prescribed. Sometimes a Gregory's powder at bedtime, or a mild carminative, is of value. An overloaded colon is a common cause of sleeplessness in elderly people; for this an evacuation of the bowels at bedtime is the best means of relief. Dilatation of the stomach sometimes produces serious and obstinate insomnia, and treatment directed to the prevention of fermentation in the stomach and intestinal canal is necessary (Broadbent). One of my patients found that a little peppermint water taken at bedtime gave him a better night than any hypnotic. In the insomnia of the aged and the overtired, and in that due to cardiac and vascular disease, strychnine is beneficial: it tones up the circulatory system and removes indigestion and flatulence. In cardiac cases and in neurasthenics with low arterial pressure, digitalis and the other cardiac tonics may be used. In anaemia, iron, nux vomica, and sometimes digitalis are required; and similarly other diseases which are causal factors of insomnia must be treated in a more or less specific way. The appropriate remedies for each disease are described elsewhere.

In all cases of insomnia certain general precautions must be taken. The bedroom ought to be in a quiet part of the house, well ventilated, and of moderate temperature. Light should be carefully excluded, and the apartment scantily furnished. The bed should vary according to the habits of the individual; for young and middle-aged adults a firm mattress is the best, but for the old a softer bedding may be necessary. The covering should be light and warm; but in the use of pillows no general recommendation can be made. Some people sleep better with the head raised, others prefer to keep it on a level with the body. In heart disease it will be found necessary to raise it; but as a rule this precaution may be left to the choice of the individual. For broad-shouldered people Sir W. Whitla recommends the wedge-shaped pillow used by the Germans. Invalids find much refreshment in the use of two beds, one for day and the other for night.

For ordinary cases of sleeplessness simple means very often suffice. Some people read themselves to sleep, some count, others, like Southey, think of some monotonous discourse. One of my patients used to hang his feet out of bed for some time and then put them in again. Walking about naked, or a cold or tepid bath is often useful. Massage, especially of the abdomen, thighs, and legs, is sometimes advantageous. This method is believed to produce temporary anaemia of the brain, by causing a determination of blood to the manipulated parts; and it may be further aided by a hot compress to the abdomen. In the case of cold feet, standing in cold water and vigorous rubbing with a rough towel (Broadbent), or a hot bottle, or a hot footbath with mustard in it, is beneficial; or again a hot sitz-bath may be used. Attention should also be given to the work of the stomach. As a rule a light supper is the best, and for many, and especially those who awake in the middle of the night, a little hot milk, or a teacup of strong, hot beef-tea or meat juice containing a small amount of alcohol is helpful. The evacuations should also be attended to, and the bladder especially should be relieved.

Sleeplessness from overwork, especially from literary work, requires mental rest and change of air and scene. Motoring is especially good for it. Temporary exposure to the cool air of the bedroom, or the wet pack, or a bath is often of use; but if the insomnia continue, it is necessary to give a mild hypnotic, such as 5 to 10 grains of veronal or 20 grains of sulphonal or trional, or 30 or 40 grains of bromide of potassium, to break the habit of sleeplessness. Capsules containing ♏xxx. of turpentine given at bedtime are sometimes beneficial in the insomnia of overwork and worry. The drug acts as a stimulant and derivative, and is stated to succeed best in plethoric cases. No beverages containing caffeine should be taken after breakfast.

In nervous and hysterical women, and especially in women at the menopause, the bromides are very useful. I have long been in the habit of giving a mixture of bromide—either of potassium, sodium, or ammonium—tincture of sumbul, and tincture of hop, in camphor water, at the climacteric; and it has helped to remove the insomnia as well as the mental depression and flushing heats; bromural is also useful.

The sleeplessness of the insane requires careful management. In the early stages of acute mania the bromides, chloral, hyoscine hydrobromide, and other sedatives are useful; but a hot bath at a temperature of 104° F., and cold water simultaneously poured upon the head are most efficacious in inducing sleep. In melancholia, in which arterial pressure is usually high, paraldehyde in doses of ♏xl. to ♏xc., or even more, is a valuable hypnotic; so is morphine; but a 1-grain dose of erythrol tetranitrate, by reducing blood-pressure, will frequently act better than anything else.

In mild cases of delirium tremens sleep usually comes on after a time, whatever treatment be adopted; in the more severe cases chloral and bromides, alone or in combination, are beneficial. Paraldehyde is recommended by some physicians. Opiates may be given, but in most cases hyoscine is probably a more efficient remedy. Vigorous cold affusion sometimes sends the patient into a sound sleep, even when full hypodermic doses of morphine or hyoscine seem to have no effect (Broadbent). Among the medical officers of the United States Army 20 grains of powdered capsicum in the form of a bolus is the favourite hypnotic in this complaint. Cerebral depressants should be given as little as possible, and the treatment should be confined chiefly to feeding and tonic measures.

In pneumonia sleep comes usually at the crisis; but where this has not occurred I have occasionally seen a hypnotic, such as chloralamide or paraldehyde, turn the scales in favour of the patient.

In pleurisy, and most other serous inflammations, 5 to 10 grains of Dover's powder usually conduce to sleep; mainly by relieving the pain. A hypodermic injection of morphine may be given with the same object in view, but the application of a few leeches is safer and usually quite as efficacious.

In bronchitis, chloral and chloralamide are safe hypnotics; as a rule opiates are to be avoided, with the possible exception of heroin.

The sleeplessness of asthma is relieved by remedies which cut short an attack, such as chloral hydrate, the fumes of Himrod's and other anti-asthmatic powders, the hypodermic injection of morphine, or, in some cases, a dose of 5 to 10 grains of citrate of caffeine. Bromides are also useful and so is paraldehyde, which both relieves the asthma and causes sleep. A change of locality, if only to another part of the same town, often succeeds.

The insomnia of heart disease is benefited by digitalis, strophanthus, strychnine, and other cardiac tonics; but in some cases it is necessary to resort to morphine, either by the mouth, or still better, hypodermically, as first suggested by Sir Clifford Allbutt. Paraldehyde and chloralamide are most useful; they are less depressing to the circulation than chloral hydrate. Ice to the head is recommended where the vital forces are not too low, or the temperature subnormal (A. Morison). It often produces sleep rapidly, with a more regular cardiac action. Heat may possibly answer in cases with a subnormal temperature.

In chronic Bright's disease insomnia is occasionally very troublesome. Eliminants, such as aperients, should be tried, and if they do not succeed chloral hydrate may be given; it is a safer drug in kidney than in heart disease, the accompanying reduction of blood-pressure being usually beneficial. Morphine and hyoscine hydrobromide, subcutaneously injected, have been recommended in obstinate cases; but their employment requires great caution. Erythrol tetranitrate, by reducing blood-pressure, often acts like a charm even when sedatives have failed; and in one of my patients thorough rubbing of the skin by means of a flesh-brush induced sleep, and very materially relieved the restlessness of this complaint. In the sleeplessness dependent upon granular kidney Dr. Nestor Tirard recommends hyoscine, sulphonal, and paraldehyde.

When pain is the causal factor of insomnia morphine is the best general remedy, and it should be pushed until relief is obtained. In cases of neuralgia, locomotor ataxia, and so forth, some of the synthetic analgesics—phenazone or phenacetin—are of value. These drugs act also, I believe, as hypnotics in cases in which there is no pain.

Calcium salts are valuable in the insomnia due to pruritus.

This article would be incomplete without a more detailed reference to some of the more important hypnotics. These are comparatively few, although the number of the drugs of this class which have been advocated from time to time is large.

The *bromides* of potassium, sodium, and ammonium are mild, safe, and trustworthy hypnotics. They tend to depress the functions of the spinal cord, but their effect upon the heart, lungs, and other organs is small. Potassium bromide is the most depressing of the three, ammonium bromide the least; but in therapeutic doses this action is not very obvious, and the potassium salt is the most convenient to use. By many it is regarded as the most trustworthy.

Various other bromides also — for example, lithium bromide and hydrobromic acid—have been given as hypnotics; but none of these presents any material advantages over the salts more commonly used.

The bromides are useful in cases of insomnia due to worry or overwork. When relief cannot be obtained by non-medicinal means they are valuable to break the habit of sleeplessness, and thus to restore the brain to a more normal condition. In the presence of pain, however, they are generally useless. They are also the most useful drugs for repeated administration, as they appear to produce less serious ill-effects than other remedies of this class. The dose may be given just before retiring, or, as recommended by Macfarlane, in two or three doses during the latter half of the day.

Chloral hydrate is a more powerful, but also a more harmful drug than the bromides. It produces general nervous depression, affecting both the brain and spinal cord; and it is also markedly depressant to the heart and vascular system. On this account it should be avoided in all cases of heart disease with symptoms of heart failure, and generally in diseases with a low blood-pressure, such as enteric fever. In cases of high arterial pressure, however, it may usually be given with impunity. Also in the more simple forms of insomnia, when bromides prove insufficient, to combine them with chloral hydrate is judicious and often successful. It should, however, be used with care, both on account of its circulatory effect and its tendency to form a habit. A suitable dose is 15 to 20 grains, or, combined with bromide, 10 to 15 grains; but sometimes more is required. Large doses should, however, be avoided.

Butyl-chloral frequently relieves insomnia due to trigeminal neuralgia. As substitutes for chloral hydrate, certain derivatives, purporting to possess its useful properties without its ill-effects, have been recommended. Of these one only, chloralamide, is of practical importance.

Chloralamide, or chloral-formamide, is undoubtedly less toxic than chloral hydrate, and does not produce the same depressant effect upon the circulation and respiration; but it is also a less powerful hypnotic. It may be used in doses of 25 to 40 grains in all cases in which chloral is indicated, and in some forms of heart disease and bronchial affections.

Paraldehyde is a safer drug than any of the preceding. It produces sleep without any distinct respiratory or circulatory effect; but it is slightly irritant to the gastric mucous membrane, and possesses an unpleasant smell, which can be detected in the breath after its administration. On this account, indeed, it is not so likely to cause a habit, although a few cases of this are on record. It may be generally used as a hypnotic, and is especially serviceable in asthma, heart disease, and melancholia. It is best administered with tincture of orange, in a dose of xl. to xc. ♏, or upwards. Dr. Clouston has given it in 4 to 6 dram doses.

Sulphonal and *trional* are valuable hypnotics; the former in doses of 15 to 30 grains usually produces sleep in two to three hours, followed by no ill effects. Drowsiness is sometimes experienced during the following day; and the sleep of the succeeding night is often as sound, or sounder, than that of the night of administration. It possesses no action on the circulatory and respiratory systems. As it is slightly cumulative, repeated dosage may lead to haematoporphyrinuria and other untoward effects, and on this account it should not be continued longer than from four to five days. An interval of an equal or longer period should be allowed to elapse before resorting to it again, if this be necessary. Owing to the insolubility of the drug in cold water, it is best given in hot fluids, such as milk or soup; in this way a more rapid effect is obtained. Sulphonal is useful in all mild forms of insomnia, and one dose often produces two nights' sleep. It is adapted for continued administration, provided that intervals are allowed to elapse every few days, and that care be taken to avoid constipation.

Trional possesses similar properties to sulphonal, and exerts a similar effect. It is somewhat more powerful, and, being more soluble, it does not lead to a second night's rest, like sulphonal. Its uses are the same as those of the latter drug. It is valuable in the insomnia of children. Some authors say that it relieves pain as well as induces sleep.

Alcohol, in those unaccustomed to its use, is a valuable hypnotic in mild cases; especially in the form of beer, stout, or whisky, at bedtime.

Veronal (diethyl-malonyl-urea) is one of the quickest, safest, and most reliable hypnotics. In doses of 5 grains for women, and 5 to 10 grains for men, given in some hot liquid, at bedtime, it almost invariably induces in half an hour quiet refreshing sleep without any unpleasant after-effects, such as drowsiness the next day or constipation. Its action in some cases is not limited to the night in which it is taken, but is continued for two or three or even four nights. It is a safe and efficient hypnotic in all forms of sleeplessness in which pain is not a prominent symptom. When the latter is present a combination of aspirin with veronal has been recommended. I have never found it necessary to increase the dose beyond 10 grains. Fatal results have followed the administration of larger doses such as 30 grains and upwards.

Veronal-sodium is much more soluble than veronal, and has been recommended for rectal and subcutaneous administration.

Bromural (α-monobrom-isovalerianyl-urea) is a powerful nerve sedative and mild hypnotic, which in 10-grain doses has been found useful in the insomnia resulting from overwork or slight excitement. It is said to cause no by- or after-effects, and forms the preliminary for healthy dreamless sleep.

Opium and *morphine* are the most useful hypnotics when there is great pain, but they should be used in few other cases; for instance, in recent and acute insomnia from worry, overwork, or shock. Their influence on the heart and vascular system is small, so that in heart disease, especially if attended with pain, they may generally be employed; but their influence upon the respiration is well marked, and in

KING'S COLLEGE HOSPITAL
MEDICAL SCHOOL.

all respiratory diseases accompanied by impeded respiration from excessive secretion they should be regarded with disfavour. As opium inhibits most secretions, constipation and other undesirable effects usually accompany its use. Rapid habituation also ensues, and in some patients marked excitement results from its first administration. In painful maladies morphine hypodermically should be pushed until the pain is abolished; but on account of the well-known toxicity of the drug, care should be taken in its administration. In heart disease, $\frac{1}{6}$ to $\frac{1}{4}$ of a grain of a salt of morphine may be given subcutaneously, especially if there is stenocardial pain; or the drug may be taken by the mouth.

Opium is more slowly absorbed than morphine, and its action is correspondingly slower and more prolonged. It also possesses a greater local effect upon the alimentary canal, producing more marked constipation. It may be used to prolong the effect of morphine, or in cases, such as gastric cancer, where a local effect may be of value in aiding its general action.

Owing to the rapid establishment of tolerance, its ill-effects, and its tendency to induce a habit, morphine is not a good general hypnotic; and many attempts have been made to discover a substitute possessing its hypnotic and analgesic actions without its inconveniences, but so far without success. Certain derivatives of morphine have been suggested; but these are mainly sedative, and are not sufficiently powerful to be used in those cases in which morphine is indicated. If sleeplessness is due to a troublesome cough $\frac{1}{10}$ grain of heroin hydrochloride will sometimes stop it and ensure a good night.

Hyoscine, in the form of one of its salts, is one of the most powerful hypnotics we possess, and is said never to induce a habit. It is of special advantage in restless cases, such as the insane, where it is impossible to administer drugs by the mouth and undesirable to give opium. It is, indeed, the best substitute for morphine we possess; rarely producing excitement, and an atropine-like effect occurring only after considerable doses. Owing to its great toxicity care must be taken in its administration; $\frac{1}{200}$ to $\frac{1}{150}$ grain of the hydrobromide, given hypodermically, is usually sufficient under ordinary conditions to produce sleep; but in the insane larger doses—$\frac{1}{100}$ grain, or even $\frac{1}{50}$ grain—are often required. These quantities should not, however, be given as initial doses. Valvular disease, so common in acute mania, contra-indicates its use. A combination of chloral with $\frac{1}{120}$ grain of hyoscine, or with morphine, has given excellent results in obstinate insomnia with excitement.

Hyoscyamine has been used as a hypnotic in cases in which hyoscine is of benefit, but it is not so valuable. It produces more numerous ill-effects, and its sedative action is less powerful. Prof. Cushny says hyoscyamine is practically devoid of sleep-bringing properties. The other members of the atropine group are even less useful. Hurd recommends a teaspoonful of the tincture of hyoscyamus at bedtime as a useful hypnotic in cystitis.

Cannabis indica is used by many as a hypnotic. It is, however, apt to produce excitement, and sometimes this is not followed by sleep. It does not produce constipation as opium does, and it is less toxic; in fact no fatal case of poisoning by this drug is known. Its greatest drawback is its inconstant composition owing to its deterioration with age from undergoing oxidation, and so far no official action has been taken to obviate this. Several preparations are on the market. The best are the extractum cannabis indicae of the British Pharmacopoeia, Merck's cannabinon and extractum cannabis indicae, and cannabinol. A convenient dose to begin with is half a grain of the extract, but with many samples this amount has to be exceeded. There are no special indications for its use. It is given in migraine and neuralgia, and often alleviates the pain of gastralgia in old people; but it is not a powerful analgesic. In other forms of pain it is less valuable. It may be regarded as a general hypnotic except in cases in which its excitant action is contra-indicated; and if a good preparation can be obtained, it appears to be a valuable one. "Bromidia" is stated to contain in each dram chloral hydrate 15 grains, potassium bromide 15 grains, extracts of cannabis indica and hyoscyamus $\frac{1}{8}$ grain each.

Two other agents may be used to induce sleep if the measures previously considered fail; namely, electricity and hypnotism.

Electricity.—Electricity is regarded as a valuable agent by those accustomed to use it, but it is generally recognised that its employment is empirical. In some cases it produces excitement rather than sleep, and it is said "to be largely a matter of experiment as to who will and who will not sleep better after its use." In the majority of cases, however, sleepiness is induced, and, according to Eskridge, the cases most commonly benefited are those in which the insomnia is due "to mental overwork, worry, alcoholism, and supposed hyperaemic conditions of the brain generally." Riggs also states that it is most useful in the sleeplessness accompanying nervous and mental disorders.

Various modes of electrification—general faradisation, galvanisation of the head, cervical sympathetic, etc., and static electrification—are in use. Dr. Lewis Jones recommends general faradisation or the faradic bath, but the majority of electricians are in favour of galvanism. Weak currents (1-3 milliamperes) directed longitudinally and continued until the patient feels drowsy, sometimes 15 to 20 minutes, or even 30 minutes, and repeated on alternate days, appear to produce the best results.

Dr. W. F. Somerville speaks highly of the value of high-frequency currents in the treatment of insomnia. Sometimes, he says, the result is immediate, at other times a more prolonged course of treatment is required. He has found it necessary to administer a large amount of milliamperage, reaching 700 to 800 ma. This maximum is not attained at once; but, commencing with a small dose, the amount is gradually increased till the maximum is reached. High-frequency applications probably reduce blood-pressure and are therefore most beneficial to patients of this class.

In some cases static insulation is preferred, and positive insulation is said to be better than negative (Rockwell). The method as practised by Dr. H. McClure is as follows: the patient, insulated on a glass stool, is brought into connexion with the conductor of a Carré or Holtz machine. When thoroughly electrified a fine metal point is held opposite several spots on the scalp and forehead, but not near enough to produce a spark. The sensation is as if a light wind or breeze was playing over the part, and sleep is said often to come on while the treatment is being employed (Whitla). Bonnéfoy has pointed out that statical applications are specially indicated in subjects with a low arterial pressure.

The influence of electrification, in whatever way it is used, is said to be both temporary and permanent. The temporary effect occurs during the first few nights, and is never so great as that obtained from drugs. But the permanent influence is the end to be obtained. As compared with drugs electricity possesses the advantages of being free from ill-effects and the danger of forming a habit, but in some cases it fails to act, and may even aggravate the symptoms; consequently its effects must be carefully watched (Riggs).

Hypnotism.—Cases of insomnia, rebellious to all other forms of treatment, may be treated by hypnotic influence, but only in the hands of a properly qualified and specially experienced medical man. Under such conditions I believe it to be a valuable therapeutic agent.

SOMNOLENCE.—Somnolence, or excessive sleepiness, as previously mentioned, occurs in persons of feeble intellect. Some old people also sleep a great deal. In cases of organic brain mischief, especially cerebral syphilis, and in some epileptic and hysterical persons, somnolence is met with. It has also been attributed to the reflex irritation caused by round worms, as the removal of these has caused the somnolence to cease. Some sufferers from anaemia, leukaemia, and myxoedema sleep too much, the last more especially in the daytime.

DREAMS AND NIGHTMARE occur when certain cortical centres are active, while others are in abeyance. They frequently arise from indigestion; but occasionally they are the forerunners of apoplexy or insanity. "The recurrence of a peculiar and unusual dream night after night, particularly if it is of a terrifying kind, should always arouse suspicion" (Macfarlane). A patient of mine, an old lady of eighty-one, had a most distressing dream a day or two before an attack of right hemiplegia.

For *The Night-Terrors* of children, see art. p. 804; for matutinal or sleep ptosis, in which the eyelids remain closed for some time after waking, see Vol. VII. p. 329; and for recurrent paralysis on waking, see p. 608.

SLEEP-TALKING or somniloquy, and SLEEP-WALKING or somnambulism, are states in which the whole brain is not asleep, but certain centres continue in activity. Some persons, while apparently asleep, may even carry on a conversation, as if they were awake.

Somnambulism, which has been described as an acted dream, runs in families, and occurs about equally often in the two sexes. The subjects of it can perform extraordinary feats, such as unlocking doors, walking in dangerous places, as on the edge of a precipice in the dark, scaling a partition of considerable height (8 to 10 feet) between two sleeping-apartments; and they usually do all this without injurying themselves.

It is necessary in some of the worst cases to keep the patient in a room locked from the outside, to fasten all windows, and occasionally to chain one leg to the bed, so as to wake him if he begins to walk. The general health should, of course, be carefully attended to.

J. B. Bradbury.

REFERENCES

1. Acland, T. D. *On the Hours of Sleep at Public Schools*, 1905. J. and A. Churchill, Lond.—2. Allbutt, Clifford. "The Hypodermic Use of Morphine in Heart Disease," *Practitioner*, Lond., 1869, iii. 342.—3. Broadbent, W. H. "Treatment of Sleeplessness," *Practitioner*, 1906, lxxvii. 1.—4. Bulkley, L. D. "Sleep in its Relations to Diseases of the Skin," *Selected Essays*, New Syd. Soc. 1897.—5. Buzzard. *Diseases of the Nervous System*, pp. 282-290, 1882.—6. Cappie, J. *Intracranial Circulation*, 1890.—7. *Idem.* *Brain*, 1898, 58.—8. Carpenter, W. B. *Principles of Mental Physiology*, 4th ed. p. 568; 1874.—9. *Idem.* *Principles of Human Physiology*, 8th ed. p. 753; 1876.—10. Clarke, C. K. *Brit. Med. Journ.*, 1897, ii. 853.—11. Cushny, A. R. "Treatment of Sleeplessness and Pain," *ibid.*, 1905, ii. 1005.—12. Durham, A. E. *Guy's Hosp. Rep.*, 1860, vi. 149.—13. *Idem.* *Encyclopaedia Britannica*, art. "Sleep."—14. Eskridge, J. T. In Wilson's *American Text-book of Applied Therapeutics*, 1896, p. 974.—15. Folsom, Charles F. "Disorders of Sleep: Insomnia," *Trans. Assoc. Amer. Phys.*, Phila., 1890, v. 131.—16. Ferguson, R. *Brit. Med. Journ.*, 1897, ii. 860.—17. Fothergill, M. *Practitioner*, 1876, xvi. 103.—18. Gould and Pyle. *Anomalies and Curiosities of Medicine*, chap. xvii., Lond., 1897.—19. Granville, J. M. *Lancet*, 1880, ii. 333.—20. Hammond, W. D. *Sleep and its Derangements*, 1869.—21. Hill, L. *The Cerebral Circulation*. 1896; *Lancet*, 1898, i. 282.—22. Holland, Sir H. *Chapters on Mental Physiology*, 1858.—23. Howell, W. H. *Journ. Exper. Med.*, 1897, ii. 313.—24. Hurd, E. P. *Sleep, Insomnia, and Hypnotics*, 1891.—25. Jones, L. *Medical Electricity*, 1895, p. 295.—26. Leech, D. J. *Brit. Med. Journ.*, 1889, ii. 968.—27. Macfarlane, A. W. *Insomnia and its Therapeutics*, 1890.—28. Mackenzie, J. C. *Journ. Ment. Sc.*, 1891, xxxvii. 18.—29. Macnish, R. *The Philosophy of Sleep*, 1830.—30. Manacéïne, M. de. *Sleep: its Physiology, Pathology, Hygiene, and Psychology*, 1897, where an excellent Bibliography can be found.—31. Marvand, A. *Le Sommeil et l'insomnie, étude physiologique, clinique et thérapeutique*, 1875.—32. Mitchell, J. K. In Wilson's *American Text-book of Applied Therapeutics*, 1896, p. 1139.—33. Mitchell, Weir. "Some Disorders of Sleep," *Trans. Assoc. Amer. Phys.*, Phila.. 1890, v. 108; and *Clinical Lessons on Nervous Diseases*, 1897, p. 58.—34. Moore, C. H. *On going to Sleep*, 1868.—35. Morison, A. *On Cardiac Failure and its Treatment*, 1897, p. 91.—36. *Idem.* *Practitioner*, 1889, xlii. 277.—37. Riggs, C. E. In Bigelow's *International System of Electro-Therapeutics*, 1894.—38. Rockwell, A. D. *The Medical and Surgical Uses of Electricity*, 1896, p. 383.—39. Sée, G. *Méd. mod.*, Par., 1889-90, i. 181, 201.—40. Skerritt, E. M., and Stewart, J. "Case of protracted Sleep extending over Fifty Days," *Brit. Med. Journ.*, 1898, ii. 957.—41. Smith, E. *Ibid.*, 1869, ii. 235.—42. Somerville, W. F. "High-frequency Currents for Insomnia," *ibid.*, 1909, i. 1063.—43. Symonds, J. A. "Sleep and Dreams," *Miscellanies*, 1871, p. 145.—44. Tuke, D. H. *Dictionary of Psychological Medicine*, 1892.—45. Weber, Sir H. "On Means for the Prolongation of Life," *Brit. Med. Journ.*, 1903,

ii. 1445 (deals with Sleep in Old Age).—46. Wetherill, H. M. "The Modern Hypnotics," *Amer. Journ. of Insanity*, 1889, xlv. 28.—47. Whitla, W. *Dictionary of Treatment*, 1902.—48. Wilcox, R. W. *Brit. Med. Journ.*, 1897, ii. 855.

J. B. B.

ON NIGHT-TERRORS, SYMPTOMATIC AND IDIOPATHIC, WITH ASSOCIATED DISORDERS IN CHILDREN

By Leonard G. Guthrie, M.D., F.R.C.P.

Even when observed, night-terrors are commonly regarded as trivial matters, and of humorous rather than pathological import. Yet the study of the illusions, delusions, and hallucinations which come "through the ivory gate," may be of pathological no less than of psychological interest.

Symptoms of Night-Terrors.—The attacks are usually described in children aged between three and eight years. Older children suffer from them, but we are comparatively seldom called to treat the disorder in these patients.

Typical night-terrors occur, almost always, within half an hour to two or three hours of going to bed. Except in cases of febrile disturbance, recurrent attacks in a single night are rare; but they may be repeated for many nights on end, or at weekly, monthly, or longer intervals. Piercing screams are heard, and the child is found sitting up in bed, or crouching in a corner of the room, with hands outstretched as if to shield himself. Sometimes he is trying to get out of the window, or to open the door. He may escape from the room, and run screaming down the staircase with fear-winged feet. His face expresses wildest alarm, his eyes are widely opened, with pupils dilated, and he gazes intently at the ceiling, or towards some quarter of the room in which he locates the apparition which frightens him. He often gives a clue to its nature by shrieking, "Black faces!" "Black dogs!" "Horrid man!" or some such exclamation.

These hallucinations appear to be common in children up to the age of five or six; but in older children there is often a difficulty in discovering the content of the hallucination, owing, doubtless, to its being more complex in nature, though no less terrifying than simpler visions. The child may cling instinctively for protection to any one within reach, but he does not at first look at or recognise persons; his attention is fully taken up by the imaginary object of his fear. The period of terror lasts from a few moments to half an hour. The child usually then recognises his surroundings, but he sweats and trembles, turns pale, and seems shocked and exhausted. He will beg not to be left alone in the dark again, or that his hand may be held. He soon dozes, but sobs and starts for a time before sleep becomes sound. Sometimes at the end of an attack he passes a large quantity of pale urine, but more frequently he has wetted the bed or the floor during the stage of excitement.

The foregoing is a general account of the symptoms, but subtle distinctions between the attacks and between the patients have been drawn.

Night-terrors have been divided by Silbermann into idiopathic and symptomatic. The characteristic of the idiopathic variety he holds to be a transitory hallucination of sight, due to an abnormally increased irritability of the brain; whilst the characteristic of the symptomatic form is a sensation of distress arising from digestive disturbance acting through the agency of the vagus nerve, and giving rise to dyspnoea. This distinction is not altogether satisfactory to my mind, because it is too narrow. Although visual hallucinations are commonest in young children, other hallucinations, such as those of hearing and of common sensation, besides those of sight, may occur in night-terrors. Jerome Cardan, the famous philosopher, mathematician, and physician of the sixteenth century, said that from his seventh to his twelfth year he was haunted by nightly visions of a red-winged cock, which frightened him lest it should speak, which it always did; uttering in human voice, "Verba plerumque comminatoria." Dr. W. S. Colman mentions the case of a child, aged two years, who woke screaming that some one was hurting his finger. He pointed out where the person seemed to be, and insisted that his finger was bleeding.

Night-terrors cannot be regarded as idiopathic simply on account of the visual nature of the hallucination; indeed, were it so, nearly all must be so designated, whereas there can be no doubt that the majority are symptomatic in the sense that they are due to reflex irritation of the brain by local conditions or disorders. Moreover Silbermann's symptomatic variety should be widened in order to include night-terrors caused by local affections other than digestive, and not characterised by dyspnoea alone. Nevertheless, dyspnoea from various causes is a potent source of night-terrors.

Dr. Coutts distinguishes between "night-terrors" and "nightmares": the latter he regards as due to reflex disturbances; the former, he thinks, cannot be thus explained. Like Silbermann, he regards it as essential to night-terrors that the child should see visions, whilst in nightmare he only dreams dreams. Other points of distinction which he makes are:—(1) That night-terrors seldom occur in children over eight years of age, whilst nightmare may happen at any age. (2) That night-terrors occur during the best of health, whilst nightmare attends chronic ill-health. (3) That in the course of a night there is usually but a single attack of night-terrors, although there may be several of nightmare. (4) That in night-terrors there is a family history of neuroses, such as epilepsy, chorea, hysteria. In nightmare there is no such history. (5) That infantile convulsions usually precede night-terrors, but not nightmare. (6) As regards the differentiation of symptoms, he mentions that in night-terrors the child seems wide-awake, but is not so. He still sees the vision that has terrified him, and is unconscious of his surroundings. In nightmare he is actually wide-awake, but partially demented by recollection of a troubled dream. He is fully conscious of the presence of others in the room. (7) In night-terrors there is no recollection of the event, whereas in nightmare there is a perfect recollection, and a dread of recurrence. (8) In night-terrors the kind of vision never varies ; in nightmare the objects of apprehension have generally been met in waking hours, and are not invariably the same.

These distinctions are so clearly and well defined that it seems a pity to cast doubt on their practical value. Yet I must confess that they seem to me to be too hard and fast. Hallucinations of vision, as part of night-terrors, are not confined to children under eight years of age ; they may occur at any age. They are frequently symptoms of febrile delirium. Little children are frightened out of their wits at such hallucinations, because they do not know what they are. Older children see them, and are terrified by them, but usually will not admit it. Adults see them, and send an embroidered account of the vision to the Psychical Research Society. Visual hallucinations do not constitute a special type of night-terrors. The statement that night-terrors (visual hallucinations) occur in the best of health, but nightmares (troubled dreams) attend chronic ill-health is perhaps too sweeping. Both are indications of ill-health, chronic, acute, or temporary ; and affecting brain or body, or both. The recollection or oblivion of the event next day can hardly be relied on as a basis of distinction between night-terrors and nightmare. A dream which is most vivid in all its details at the moment of awakening often fades like breath from the surface of a mirror, and is effaced, as it were, by one's morning ablutions. On the other hand, the essence of an alarming dream in early childhood may never be forgotten. But many children will not give any information on the subject because the horror of it is too complex for their powers of verbal description, or they will deliberately conceal their experiences lest they should be laughed at or accused of over-eating. The statement that night-terrors usually occur once only, whilst several attacks of nightmare may be repeated during the night, is not in accord with my experience. There may be several repetitions of an alarming visual or other hallucination in a single night, especially in cases of pyrexia. The unvarying nature of the vision does not constitute a distinction between night-terrors and nightmare. The same dream hallucinations which terrified us as children may haunt us as adults till we die. A dream which has once powerfully affected us is apt to recur as a habit under slight provocation. Its recurrence cannot be held to distinguish night-terror from nightmare. In fact, there seems to be little reason for regarding frequent repetition of the same dream, or peculiarity in its nature, as indications of night-terrors rather than of nightmare. The difference is merely one of degree. A nightmare in one child is a night-terror in another. One child may be merely frightened, another may be driven temporarily insane by his dreams. A stolid, commonplace child may over-eat himself and wake screaming in the night. Next day he will be none the worse, and will only vaguely remember that he has had a bad dream. It is far otherwise with the emotional, imaginative, and neurotic child. To him, the prospect of his "witch-ridden pillow," as Charles Lamb called it, is one of unutterable terror and dismay. If he keep his mental torment on this account to himself, as he often does, there can be no doubt that mind and body will suffer, it may be for a lifetime. Night-terrors may be regarded as nightmare exaggerated by the emotional disposition of the sufferer, and this accounts for the difference in symptoms to which Dr. Coutts has called attention. As he points out, in cases of night-terror there is usually a personal or family history of neuroses such as epilepsy, infantile convulsions, chorea, hysteria. In nightmare there is no such history as a rule.

Although night-terrors and nightmare differ only in degree, it is necessary to have some plan of differentiation; for troubled dreams may be of trivial or of the gravest import to the patient. For this reason, Silbermann's division of night-terrors into idiopathic and symptomatic is perhaps the simplest and best in order to enable us to ascertain in every case the condition with which we have to deal. I believe that all dreams are symptomatic in the sense that they are excited by some dimly-felt bodily sensation or peripheral stimulus; but they are also idiopathic in the sense that the interpretation of the sensations which give rise to them is entirely the dreamer's own. Hence, the character of the dreamer is of more consequence than the nature of the dream. It is perhaps difficult at first to reconcile a reflex hypothesis of dreams with the absence in many cases, of any relation between the tenor of the dream and the presumed stimulus which excites it. For instance, why should a child dream night after night of a horrible red-winged cock because he has a pain in his stomach? The apparent difficulties may be reduced by a psychical explanation. The child has seen a red-winged cock by day and has not liked the looks of it. Its image is firmly impressed on certain neurons of his brain. Pain in the stomach awakes the child to partial consciousness in which the image of the cock is perceived by higher centres, but these being unrestrained by judgment, magnify and distort the vision, and so the cock attains formidable and even gigantic proportions, and utters "threatening words."

In dreams we are all stark-mad, because our sense of judgment, of proportion, of humour, and the ridiculous deserts us. Insanity with all its delusions is but a waking dream. Most delusions have a physical basis which the mind misinterprets because the centres of judgment sleep.

The dream hallucinations of very young children are mostly due to wakened memory of objects which they have seen, and which have terrified them—we may not know when or why—by day, for at the time they may not have shewn any signs of perturbation.

The vision is seldom, in young children, of an inanimate body, but it may be so. Thus a little boy, aged between five and six, used to wake screaming, "It's the house with the blind eyes." No one knew what he meant till, some years later, he confessed that a house, the windows of which had been blocked up in order to evade the window tax, used to frighten him by day and appear in his dreams by night. Oliver Wendell Holmes speaks of a certain glover's sign in the shape of a huge hand which had a similar effect on him as a child. Hector's nodding crest probably figured largely in the dreams of the child Astyanax. In my own early childhood the visitations took the form of Fifth of November guys. I had lost my nurse, and found myself in a street peopled by monsters clad in black and fiery-red fur. Their faces were hideous, and they had the power of looking into nursery windows, and spying out a small boy, even when he got home and hid under the bed or table.

The Content of Dreams as a Clue to Cause and Treatment.—Below the age of five or six the dream-content is usually simple, and there is seldom any difficulty in divining its nature from the child's exclamations. But later it becomes more complex, and therefore more difficult to describe. It is not until childhood is past that one learns what the child's sufferings in this respect have been. It is important therefore to ascertain if the nature or content of the dream throws any light on its cause. In many instances the prominent feature is an elaborate visual hallucination. It is of some gigantic object dimly seen in constant intricate movement; a colossal wave, a seething maelstrom, a whirling mazy web, and so forth. The phenomena are purely subjective, they do not represent any objects which could have been actually seen in waking hours, they are therefore distinguished from the early simple hallucinations which occur in young children. They frequently occur in those who suffer from migraine, and I would suggest that the hallucination is induced by the visual aura which so commonly precedes an attack of megrim. The so-called teichopsia, the appearance as of ebullition in objects, and other curious optical illusions, are familiar precursors of migraine. In the dream these illusions are converted into hallucinations.

Secondly, errors of refraction and astigmatism are now well recognised as commonly the cause of migraine, or as entering into its causation; and they are met with in persons subject to the complex visual night-terror. Thus, the apparently long step between night-terrors and errors of refraction may be bridged over by recognition of the type of night-terror to which I have drawn attention. An excellent example of this form of night-terror is given by Charles Dickens (*Uncommercial Traveller*, Essay on "Chatham Dockyard," p. 283), who wrote :—

> "Sauntering among the rope making, I am spun into a state of blissful ignorance, wherein my rope of life seems to be so untwisted by the process that I can see back to very early days indeed, when my bad dreams—they were frightful, though my more mature understanding has never made out why—were of an interminable sort of rope making, with long minute filaments for strands, which, when they were spun home together close to my eyes, occasioned screaming."

Dickens from the time when he describes himself as a sickly, and not particularly well-cared-for little boy, was subject to attacks of what I take to be enterospasm. He says that his nurse, a ghoul-like young person who told him tales of "Captain Murderer," and "Chips the Carpenter," and of a huge spectral dog whose size he feebly attempted to explain away—not that he deemed it in the least improbable, but because he felt it "to be really too large to bear"—"impaired his digestive powers for life." Dickens also suffered from hemianopsia and migraine in after-life.

Another patient thus described his early hallucinations:—

> His personality consisted of a head only, or, at all events, his body was immobile. He was in the midst of a vast, white, undulating plain, on which some dark indistinct object kept moving in concentric narrowing circles around him. The terror culminated when the thing passed behind him during its gyrations, and he could not see it, but felt that it was there. In another of his dreams there was a wall of thick darkness in front of him, on which a huge spider, as large as a Newfoundland dog, span an endless web, which somehow threatened to entangle him. In both cases he was always conscious of being in a dream, but the hallucinations and the fear of them continued. He, again, is of highly nervous temperament, and is subject to migraine.

It is true that typical migraine with its various well-known auras is seldom complained of before the age of puberty. But the auras may nevertheless occur in young children, and frighten them to distraction whether by day or by night. Adults often experience the aura of migraine without the headache, and when this happens for the first time they are often greatly alarmed by the amblyopia, hemianopsia, giddiness, and other sensations which they take to indicate serious cerebral disorder.

Partial Asphyxia as a Cause of Night-Terrors.—Night-terrors consisting in a feeling of impending suffocation, of being buried alive, or overwhelmed by an avalanche or towering tidal wave, are most commonly attributable to partial asphyxia due to naso-pharyngeal obstruction. Close bedrooms, heavy bed-clothes, the bad habit of sleeping with the head covered, and the common custom of tucking children up in bed so that they cannot move their limbs freely, may account for these and other forms of nightmare, especially the paraplegic variety. The dream in which one is pursued, but cannot run away, is probably due to actual restriction of movements. But another explanation may be offered, namely, that deficiency of oxygen is the exciting cause. For, Marcet concluded that "the conception of volition does not require oxygen, but the manifestation or response of volition cannot be carried out without it."

Aural Vertigo as a Cause of Night-Terrors.—Sir W. Gowers alludes to the feeling, common to most of us, of falling from a height when going to sleep, or just after going to sleep. "I believe," he says, "that it is really slight labyrinthine vertigo due to spasmodic contraction of a tympanic muscle, which suddenly changes the pressure within the labyrinth." A similar explanation may apply to a dream which most of us have experienced, that, though wingless, we can fly. The dream is mostly pleasant. We are extremely proud of the grace and agility with which we exercise this unusual accomplishment. But the fate of Icarus soon overtakes us, for after soaring to some precipitous mountain peak or pinnacle, our powers desert us, and we are left clinging there in agonised fear of falling; and fall we do, down, down, through endless space, until we wake, shaken, shocked, and breathless. To those more fortunate in their dreams, the power to fly when pursued and on the point of being overtaken, may come as a relief.

Night-Sweats as a Cause of Night-Terrors.—Another and more grotesque dream haunted me, as a child, for many years; I was taken captive by Red Indians, pirates, devils, or masked burglars, it mattered not by whom, the treatment was the same. I was slowly tickled under the arms whilst quite unable to stir hand or foot, or even to cry out. The process always implied the utmost malignity on the part of my captors, and caused me most exquisite torment. I dreaded it above all others, and knew beforehand in my dream when it was imminent. It was useless to say to myself, as I often did, "It is only a dream," the reality of the sensation persisted. I would wake with the horror full upon me, and pinch myself to avoid falling asleep again, but the instant I dozed my tortures began afresh. As a child I sweated much at night, and I believe the dream was excited by the sensation caused by drops of perspiration trickling down my sides, for in very hot weather, or when the bedclothes are heavy, I occasionally get it again, with all its anguish unabated. Perhaps its *mise en scène* may be due to a sleepy recollection of a nursery legend that one Simon de Montfort acquired the repulsive habit of tickling his Albigensian prisoners to death.

Night-Terrors from Febrile Disturbance.—These are characterised by visual hallucinations which remain vivid although the child seems wide-awake. He may be horribly frightened at them, and beg for their removal; sometimes he is fully conscious of his surroundings, sometimes not. Very young children do not recognise them as hallucinations, but older ones do, and, if encouraged, may speak of them afterwards. Young children seem to have forgotten all about them next day. A large number of instances of the kind are due to influenza, but they may be occasioned by any febrile disorder, and at once suggest a nightly rise of temperature. Special attention is drawn to this fact in order to shew that the hallucinations of vision which have been regarded as of extremely ill omen are not of necessity so. Nevertheless they indicate ill-health.

Considering the well-known effects of such drugs as belladonna in inducing visual hallucinations in children, it is possible that some cases of night-terrors may be due to absorption of intestinal toxins having a similar action to that of the belladonna group.

Bodily Pain as a Cause of Night-Terrors.—Here the tenor of the dream cannot always be ascertained. Night-screams may result from toothache, ear-ache, stomach-ache, hip disease, calculus, and other painful disorders.

The scream of pain can usually be easily distinguished from that of terror. The pain sometimes wakens the child, and lasts long enough for him to indicate its site. But this is not always so; children suffering from hip disease, who wake screaming at night, seldom complain of being in pain. Probably the pain in such cases is not severe. Very slight uneasiness is sufficient to set up the most weirdly terrible dream. I have often dreamed that all my teeth were falling out, and my face rotting to pieces, and have awaked to find slight uneasiness in the jaws, probably due to grating the teeth. Suspicions of hip disease or calculus always lead to inquiry as to night-screams, but night-screams do not usually arouse suspicion of hip disease or calculus; yet the possibility of this cause and effect should be borne in mind. Dreams of the Inquisition and of tortures at the stake may be produced by dimly-felt bodily pain. Dreams of being exposed on an iceberg in the midst of a vast frozen sea may arise from feeling cold.

In a few cases balanitis, phimosis, and vulvo-vaginitis have seemed to cause night-terrors, usually accompanied by wetting of the bed. Nocturnal enuresis, especially when the urine is highly acid, may give rise to complicated dreams.

Intestinal Worms. — Parents commonly attribute night-terrors and symptoms of all kinds to the presence of lumbrici or oxyurides. Often the parasites produce no symptoms whatever, but cases of obscure abdominal pain, irritability, and night-screams have been known to cease after the expulsion of round or thread worms. Catarrhal appendicitis in children, due to the presence of oxyurides (Still), might give rise to reflex cerebral irritation.

Difficult or delayed dentition in young children is often associated with febrile disturbance accompanied by nocturnal screaming.

Gastro-intestinal affections necessarily arise in the minds of all as causes of night-terrors. Yet, perhaps, too great prominence is usually given to them in this connexion. It will not be denied that an indigestible meal will often cause bad dreams, but inveterate and long-standing dyspepsia, although often coincident with night-terrors, is not necessarily associated with them. Moreover, when so associated, it will be frequently found that night-terrors do not subside under treatment for the dyspepsia. Hence the dyspepsia may be due to the general perversion of the brain which gives rise to the night-terrors. The dyspepsia may, indeed, be a neurosis. Thus night-terrors in cases of this class must be of the idiopathic variety, to which attention must again be drawn.

Idiopathic Variety.—So far I have chiefly dealt with the reflex or symptomatic variety of night-terrors, and have given examples of those which seem to be due to ocular, aural, nasal, faucial, gastro-intestinal troubles, and also of those dependent on close bedrooms, heavy bedclothes, and position during sleep. Cases have been incidentally mentioned of others in which the affection seems purely cerebral or idiopathic. In these there may be harassing repetitions of sights seen, or of events which have happened during the day.

No local or general conditions of ill-health may be apparent. They may be actually absent. Yet it must be remembered that the sufferers from the idiopathic and from the symptomatic variety of night-terrors are equally liable, at one time or another, to local or general morbid conditions, so there is no permanent distinction between the two. The real difference is that the effects are more severe and frequent, and are less relieved by local treatment in idiopathic than in symptomatic cases. In the idiopathic variety the patients are neurotic from the cradle, and they come of a stock of neurotic ancestors.

Unusual timidity is often an early sign. Little infants will start, tremble, and scream at a noise at which ordinary children would be unconcerned. The approach of a stranger, the sight of everything which is new to them, excite, not the normal curiosity of a child beginning to take notice, but wild screams of unreasonable alarm. Mothers of little children in arms will say that they dread to take them out because they become so frightened at everything they see. They are peculiarly liable to infantile convulsions. This unnatural timidity persists as the infant grows into a child. It often takes the form of shyness and morbid self-consciousness; he will not associate with other children, or take part in their games, and he looks bewildered and anxious at the noise they make. He prefers the society of adults, and soon acquires a precocity and old-fashionedness which make him unpopular in the nursery and with his school-fellows. From sheer timidity he may become untruthful, and his character is thereby lost. He often gets shamefully teased and bullied, but bears it all with stolid patience until his wrongs have accumulated, then some trivial annoyance becomes the last straw, and he bursts into ungovernable and almost maniacal passion. He may become reserved, morose, jealous, and spiteful; he does not respond to kindness because he is always brooding over his fancied or real slights and grievances. Yet he is often a prey to agonies of remorse for all his misdoings, real or supposed. His dreams are of the judgment day and hell. His digestion and general health suffer, and he is apt to become melancholic, or, it may be, suicidal, hysterical, choreic, epileptic, or even maniacal. In some children timidity takes the form of most highly imaginative superstition. Their thoughts run on ghosts, witches, and death. All subjects which are terrible and uncanny fascinate them. They will pore all day over Fox's *Book of Martyrs*, or the dreadful pictures in some editions of the *Pilgrim's Progress*, and dream of them all night.

I cannot give a better illustration of the night-terrors thus excited than that contained in Charles Lamb's essay on "Witches and other Night Fears." He says:—

> There was a picture, too, of the witch's raising up Samuel (in the history of the Bible by Stackhouse) which I wish I had never seen. . . . I was dreadfully alive to nervous terrors. The night-time and solitude and the dark were my hell. . . . I never laid my head on my pillow I suppose from the fourth to the seventh year of my life—so far as my memory serves in things so long ago—

without an assurance, which realised its own prophecy, of seeing some frightful spectre. Be old Stackhouse then acquitted in part if I say, to this picture of the witch raising up Samuel (O ! that old man covered with a mantle !) I owe, not my midnight terrors, the hell of my infancy, but the shape and manner of their visitation. . . . All day long, whilst the book was permitted me, I dreamed waking over his delineation, and at night (if I may use so bold an expression) awoke into sleep, and found the vision true. I durst not even in the daylight enter the chamber where I slept without my face turned to the window, aversely from the bed, where my witch-ridden pillow was.

In my own early days I shared Lamb's horror of this particular picture, and it contained a grisly skeleton horse, which, to my mind, rendered it peculiarly aggravating. I can remember being awakened by a younger brother screaming shrilly that "Satan's feet were dangling outside the window." Our nurse attributed the vision to what she called "gormandising" ; but I knew a picture of Christian in the Valley of Death, standing prayerful, with a full-sized devil grinning over his left shoulder. The devil's feet were long and black, with disgustingly uncut nails, and they projected from behind Christian's back. Like my brother, I had seen them in my dreams, but, being older than he, I held my peace. It is interesting to note that Lamb ascribes the form, only, of his dreams to the picture which haunted him. Children such as he—and there are many—are not altogether dependent on what they see or hear for the scenes in their dreamland theatre. A lady, in her desire that her children should learn nothing but what is true, banished fairy tales from her nursery. But her children evolved from their own imagination fictions which were so appalling that she was glad to divert them with *Jack the Giant-Killer.* Doubtless, nervous and highly imaginative children are more liable to aggravated and oft-repeated night-terrors than those who are placid and commonplace. Little Ned, who, "when sent to bed, went without a noise," was probably as stolid as he was healthy, whilst "the young Augustus Edward, who reluctantly went bedward," had excellent though unexplained reasons for his disinclination. John Bunyan, himself, appears to have been a martyr to the night-terrors which his immortal works have inspired in many generations of neurotic children. When about nine or ten years old, he says : "I had but few equals both for cursing, swearing, lying, and blaspheming the holy Name of God. . . . The which, did so offend the Lord, that even in my childhood, he did tease and affright me with fearful Dreams, and did terrify me with dreadful Visions. For often after I had spent this and the other day in sin I have in my Bed been greatly afflicted, while asleep, with the apprehensions of Devils and wicked Spirits who, still, as I then thought laboured to draw me away with them." Both night and day he "trembled at the thought of the fearful Torments of Hell fire," but with a child's quaint humour, he says : "I would often wish that if it must needs be that I indeed went thither, I might be rather a Tormentor than be tormented myself." Perhaps his health, if not his morals, improved, for he adds : "A while after, these terrible dreams did leave me, which

I also soon forgot"—(*Grace Abounding to the Chief of Sinners*, by John Bunyan, pp. 9-10; Andrew Melrose, 1897).

The characteristics, then, of neurotic children who are specially prone to night-terrors are timidity, shyness, and self-consciousness. These may lead, if the children are mismanaged and misunderstood, to highly imaginative superstition, or, worse still, to melancholy and discontent which sometimes culminate in acts of apparently flagrant immorality.

Sooner or later the general health becomes impaired. Chronic dyspepsia, "habit-spasms," hysteria, hystero-epilepsy, epilepsy, and even mania are met with in such cases—and from this class are derived the neurasthenics and hypochondriacs who haunt our consulting-rooms and hospitals in after-life.

Educational Night-Terrors.—In neurotic children there is often a curious and ominous condition in which the trivial events of the day become converted into distressing dreams by night—just as in adults who are mentally over-worked. Early-school tasks, and especially initiation into the mysteries of simple arithmetic, are responsible for many instances of disturbed sleep in children. A common story is that the child is "doing sums in his head all night long." The children who suffer from this form of night-terror are usually precocious and learn without difficulty, but the symptom is an obvious indication of cerebral activity which may soon end in exhaustion. And this is even more likely to affect dull conscientious children who dream of lessons during the process of "being brought on" by teachers of Dr. Blimber's type. Such cases illustrate the danger of routine school-work in a certain class of neurotic children, and the advantage of finding out other occupations which cause them the least mental strain, and are likely to make them useful members of society.

Instances in which school-work or school-life produced disastrous effects on children neurotic by nature or inheritance might be multiplied. These are the cases in which the School-Board authorities, who sagaciously ordain that if children are well enough to play or run errands, they are well enough to attend school, should be defied.

Narcolepsy and Night-Terrors.—I have met with this association in one case. A boy, aged twelve, of highly neurotic family history, after an attack of influenza suffered from headaches and somnolency. He would fall asleep suddenly in the midst of a meal, or whilst dressing or undressing, or at any other inconvenient time. Once he slept from 3 P.M. until 11 A.M. the next day. After an ordinary night's rest he would sleep steadily through the forenoon or afternoon, if allowed to do so. At first sleep was natural, but later it was disturbed by screaming, muttering, violent struggling, somnambulism, slowness and irregularity of the pulse. Once he split his nightdress from top to bottom, and overset the table by the bedside during the night, but had no recollection of what had occurred next day. The attacks of sleepiness, at first infrequent, became of common occurrence. In the intervals he seemed well at first, but gradually his moral nature changed, and he went through what his mother called "a phase of quite grave deceitfulness." He became timid and afraid of the dark. He had no actual epileptic fit, but was subject to sudden attacks of convulsive grasping movements of the right hand and twitchings of the right side of the face. All these alarming symptoms disappeared after removing him from school, and sending him to vegetate in the country for eight months. An elder brother of his, after gaining a scholarship at a public school, suffered from fits of daze or giddiness, with other symptoms of grave cerebral exhaustion, whenever he worked for more than a few minutes at a time. He always improved after a few weeks' holiday, but return to school-work produced so many relapses that the parents wisely gave up all attempts at ordinary education. He was sent to do out-door work on a farm for two and a half years. This effected a complete cure.

Day-Terrors.—In rare cases precisely the same symptoms which attend night-terrors occur by day, and when this is the case the suggestion that the attacks are those of masked epilepsy has to be considered. At the time of an attack the child is apparently wide-awake, and Prof. Still has suggested the terms "sleeping" and "waking" terrors to convey the distinction. It may be doubted, however, whether the child is really wide-awake, or only in a dreamy mental state such as that which occurs in adults who see ghosts of departed friends and are firmly convinced that they are not dreaming.

In day-terrors the hallucinations may be visual as well as auditory. Consciousness of surroundings does not seem to be lost, but the child is beside himself with fear of what he thinks he sees or hears, and rushes to his mother for protection. In some cases, as has been suggested with regard to certain forms of night-terrors, the hallucination may be due to an aura of migraine, although the aura may be far more remote from the headache than is customary in ordinary migraine.

Sometimes both "sleeping" and "waking" terrors occur in the same subjects. A girl of six, for instance, would stop in the midst of play and shriek, "There's something black," with every appearance of the liveliest terror—shuddering and shrinking back as though something were following her or being thrown at her. Then she would scream piteously, "I can't help it!" over and over again. These attacks lasted ten minutes or so and were followed by exhaustion, sleep, and headache. Sometimes at the commencement of an attack she would shriek, "I am giddy!" or, "A fly is buzzing in my ear!" but as a rule the hallucination was visual, and took the form of something black. She had been subject to them both by night and day for upwards of three years. They formerly occurred about every third day, and she would have as many as three in one day. On one occasion, three years ago, an attack culminated in general convulsions with unconsciousness lasting several hours. This child had an excessively timid and hunted expression, as though full of dread apprehensions, which she doubtless was. She had slight naso-pharyngeal obstruction from adenoids, but no other sign of disease. Under bromide of ammonium, the attacks rapidly disappeared, but she remained hysterical and emotional. A brother suffers from "day-somnambulism." Another girl, aet. five years, had for six months almost weekly attacks, both by day and night, in which she would suddenly begin to shriek, "I cannot see!" or, sometimes at night, "A pig is after me!" or, "Things are dancing on the walls!" On going to her assistance her mother would feel her at first rigid, then quivering and quaking from head to foot. The child was never unconscious, but always much terrified. An attack would last about ten minutes, after which she would seem exhausted, and sleep, waking subsequently with a bad headache. During a month's treatment by bromide of ammonium she had no more day attacks, and only one occurred at night. Enlarged tonsils and adenoids were removed by operation, and the attacks then ceased. In both girls large tonsils and adenoids might have been the exciting cause of "sleeping" and "waking" terrors. But in both the improvement took place under the use of bromides, and in the first the nasal obstruction was but slight and an operation was not advised owing to the extreme nervousness of the child.

In two out of three of Prof. Still's cases the hallucinations were auditory. Each one would stop in the midst of play, look terrified and run to his mother, screaming sometimes, "Some one is after me!" or sometimes unable to explain why he was frightened.

Aural vertigo and tinnitus may account for day-terrors in some cases, although middle-ear disease in children is seldom associated with such subjective symptoms. A girl aged seven, for instance, after operation for mastoiditis, has been subject to attacks in which she staggers, becomes wildly excited and alarmed, clings to her mother, and screams that the houses or trees are falling on her.

Children may become quite maniacal in these attacks, as Dr. Goodhart says, and, as in the first case mentioned, may be driven into convulsions by such intense paroxysms of fear, but the convulsions are rather of the nature of hystero-epilepsy than of true epilepsy.

In younger children, aged about three years, the place of such attacks seems to be taken by fits of paroxysmal screaming without adequate cause. The children shed no tears, but seem greatly agitated, and scream incessantly for half an hour or longer, until exhausted. Paroxysmal screaming should not be mistaken for a fit of temper. It is sometimes associated with constipation arising from atony of the bowel in a neurotic child.

Mucous Disease as Cause of Night- and Day-Terrors.—Prof. Still lays stress on the rheumatic history of his cases of day-terrors, and also on the condition present which Dr. Eustace Smith has described as "mucous disease." In this the patient is languid, irritable, pale and sallow, with puffy eyelids, the bowels are constipated rather than relaxed, and the motions contain an excess of mucus. Often abdominal pain or discomfort is complained of, together with cyclic albuminuria and all its curious vasomotor disturbances.

It may be doubted whether either rheumatism or the "uric acid diathesis" or "mucous disease" is the *fons et origo* of night- or day-terrors. Any or all may be the result rather than the cause of the neurotic symptoms which prevail. Perhaps it would be more correct to say that the phenomena are part of a vicious circle.

Relation of Night- and Day-Terrors to Epilepsy.—Epileptics may suffer from night-terrors, but the subjects of even exaggerated forms of night-terrors are rarely epileptics. The analogy between night-terrors and epilepsy is no closer than that which some suppose to exist between epilepsy and migraine. Migraine and night-terrors are extremely common affections, and are apt to occur together in nervous, active-minded subjects. They cannot be intimately related to epilepsy, for were they so, epilepsy would be far more common than it is. At the same time, fright and emotional shock, both sudden and prolonged, may excite epilepsy, and so its occasional sequence to night-terrors may be explained.

The significance of day-terrors, whether, as suggested, they may be caused by vertigo or by a migrainous aura in a highly neurotic child or not, seems to be more grave than that of night-terrors alone. Yet even in such severe cases of day-terrors as have been mentioned, true epilepsy has not made its appearance. Day-terrors are more closely allied to hystero-epilepsy and hysteria, dreamy mental states, and narcolepsy than to epilepsy itself. The coincidence of day-terrors and day-somnambulism in the sister and brother to whom reference has been made on p. 815, illustrates this relationship.

Treatment of Night-Terrors.—Idiopathic night-terrors being the result of dimly felt external or internal impressions upon a morbidly excitable brain, should be treated by correction or removal of exciting causes, and by endeavouring to quiet the emotional temperament of the child.

The sleeping room should be well ventilated; soft feather-beds and heavy bedclothes should not be used. Naso-pharyngeal obstruction from enlarged tonsils and adenoid vegetations are perhaps the most frequent exciting causes. Eye-strain from errors of refraction and astigmatism are also occasional causes. Many subjects of night-terrors are of the rheumatic or so-called uric acid diathesis. In some, cyclical albuminuria, in others, mucous colic is associated with night-terrors, whilst pyrexia from any cause may be attended by terrifying visual and other hallucinations.

All these conditions need appropriate treatment, but it should be remembered that neither one nor all of them constitute the whole cause.

Removal of adenoids, scybala, intestinal mucus, threadworms, correction of refraction errors, anti-rheumatic remedies, and tonics will not always provide a cure, except in purely symptomatic cases. In idiopathic ones it is necessary also to correct morbid tendencies to brood over terrors seen and unseen, to protect active and sensitive, as well as dull brains from educational over-pressure, and to shield nervous children as far as possible from thoughts, words, sights, and deeds which are calculated to increase emotional proclivities.

As to the management of cases like his own, Charles Lamb says: "Parents do not know what they do when they leave tender babes alone to go to sleep in the dark . . . the keeping them up till midnight, through candle-light and the unwholesome hours, as they are called, would, I am satisfied, in a medical point of view, prove the better caution." If we cannot altogether approve of Charles Lamb's proposed method of treatment, we can agree with him as to the cruelty and senselessness of ignoring the mental torture which darkness inflicts on an imaginative child. The assurance that unseen powers are watching over it is but small comfort compared with that afforded by a simple night-light. I have heard of a child who, when told that she need not fear the dark because "God would be with her," said, with innocent profanity, "I wish you'd take God away, and leave the candle."

With regard to over-pressure, we must be on the watch for the irritating effects of home life as well as of school life on neurotic children, and must endeavour to counteract them. It should be remembered that the term over-pressure is relative to the mental and physical capacity of the subject. A light task to one is heavy to another. A light task is rendered heavy by physical deficiency no less than by mental incapacity. For instance, I have already alluded to errors of refraction as producers of certain forms of night-terrors and migraine. They may also lead to mental over-strain. In many schools the memory is trained by the visual method. This is excellent for those whose vision is normal, but in others, who cannot see clearly, it implies over-use of other faculties. A figure in Euclid chalked on a blackboard is no help to a boy who, at a distance of a few feet, cannot distinguish the letter A from B. He has to learn the proposition by heart beforehand, and usually breaks down in class, and is punished for idleness or obstinacy. He is usually too timid to offer the legitimate excuse; often he is quite unaware that his class-mates see better than he does. Eye-strain often means mental strain in children, and mental strain is often first indicated by night-terrors.

Treatment by Drugs.—Bromides are always essential. A dose of 5-10 gr. bromide of potassium, given an hour or so before bedtime, is often sufficient to secure a night's rest. Chloral hydrate, 2½-5 gr., may be added. Fright usually upsets digestion, gastric and intestinal, and indigestion of either kind excites night-terrors. When it can be ascertained that chronic fear and dread complete the vicious circle, bromides, besides wholesome consolation, suitable diet, and drugs which aid digestion and elimination, are needed. Bromides may be combined with bismuth, gentian, rhubarb, castor-oil emulsion, in gastro-intestinal troubles; with ferri et ammonii cit. in anaemia; with strophanthus, digitalis, citrate of caffeine, when cardiac stimulants are required; or with nux vomica and hypophosphites in emulsion of cod-liver oil in debility. In rheumatic cases, salicylate of quinine and aspirin are valuable drugs. Quinine as a rule is not well borne by highly neurotic children; when given, it should be with hydrobromic acid.

In cases of mucous diarrhoea, citrate of potassium often acts well, and is also useful when night-terrors are associated with enuresis and the presence of concentrated highly acid urine.

Somnambulism is often associated with night-terrors, but may occur independently as a habit, in children upwards of five or six years of age. Sleep-walking in any case is indicative of an overwrought brain. It occurs in epileptics, and in their families, but there is no necessary relationship between somnambulism and epilepsy. It is proverbial that sleep-walkers rarely come to any harm unless suddenly awakened. They are more apt to alarm others than themselves.

Sleep-walking may best be prevented by wearing a belt at night fastened at the back, and having a ring attached through which a bandage should be passed and tied beneath the bed. The bandage should not be tight enough to restrict movements in bed, nor loose enough to allow excursions outside.

Summary of Argument.—(1) Night-terrors are always to be regarded as evidence of ill-health.

(2) They may for convenience be divided into those of symptomatic and idiopathic origin. But *per se* the symptoms of the two cannot be distinguished. The difference between night-terrors and nightmare is only one of degree.

(3) Hallucinations of vision are usually due to febrile disturbance.

(4) The content of the dream may, in some cases, throw light on its cause.

(5) The character of the dreamer is of more importance than that of the dream.

(6) In simple cases, simple treatment based on the causation suffices. In idiopathic cases, not only local causes of irritation, but the environment and nature of the patient have also to be considered.

LEONARD G. GUTHRIE.

REFERENCES

Textbooks: 1. ASHBY and WRIGHT. *Diseases of Children*, 5th edit., London, 1905.—2. DONKIN, H. B. *Diseases of Childhood*, London, 1893.—3. GOODHART and STILL. *Diseases of Children*, 8th edit., London, 1905.—3*a*. GUTHRIE, G. L. *Functional Nervous Disorders in Children*, 1907.—4. HOLT, E. *Diseases of Infancy and Childhood*, 4th edit., London, 1907.—5. MONEY, A. *Treatment of Diseases in Childhood*, 2nd edit., London, 1890.—6. SMITH, EUSTACE. *Practical Treatise on Diseases of Children*, 3rd edit., 1909.—7. WEST, C. *Lectures on Diseases of Infancy and Childhood*, 7th edit., London, 1884. **Special Articles**: 8. BEYRAND, A. *Les terreurs nocturnes de l'enfant*, Thèse, Paris, 1900.—9. BOUCHUT. *Traité pratique des maladies des nouveau-nés et des enfants.*—10. BROWNE, Sir J. C. "Dreamy Mental States," *Lancet*, London, 1895, ii. 3.—11. COLMAN, W. S. "Night Terrors," *Trans. West-London Med.-Chir. Soc.*, 1895, vi. 145.—12. COUTTS, J. A. "Night Terrors," *Amer. Journ. Med. Sc.*, Phila., 1896, cxi. 156.—13. GOWERS, Sir W. *Diseases of the Nervous System*, London, 1892, ii. 792.—14. HENOCH. *Lectures on Children's Diseases*, London, Transl., New Syd. Soc., 1889, i. 241.—15. HESSE. *Über das nächtliches Aufschrecken der Kinder im Schlaf*, Altenburg, 1845.—16. HUBER, F. "Night Terrors," *Pediatrics*, N.Y., 1900, ix. 279.—17. LITTLE, E. G.

"Causation of Night Terrors," *Pediatrics*, N.Y., 1899, viii. 357.—18. Lyman. "Insomnia and other Disorders of Sleep," Pepper's *System of Medicine*, London, 1886, v. 370.—19. Marcet. "History of the Respiration of Man," *Lancet*, London, 1895, ii. 77.—20. Moizard. "Des terreurs nocturnes chez les enfants," *Rev. mens. d. mal. de l'enf.*, Paris, 1884, ii. 305.—21. Osler, W. "Unusual Types of Night Terrors," *Montreal Med. Journ.*, 1895-96, xxiv. 778.—22. Putman. "Night Terrors," Keating's *Encyclop. of Dis. of Child.*, London, 1890, iv. 1008.—23. Rey, J. G. "Beiträge zur Pathogenese und Aetiologie des Pavor Nocturnus," *Jahrb. f. Kinderh.*, Leipzig, 1897, n.F., xlv. 247.—24. Russell, J. "Nocturnal Fright in Children," *Birmingh. Med. Rev.*, 1882, xii. 26; 1884, xv. 1.—25. Silbermann. "Zur Lehre von Pavor Nocturnus," *Jahrb. f. Kinderh.*, Leipzig, 1883, n.F. xx. 266.—26. Steiner. "Das nächtliche Aufschrecken oder Aufkreischen (Night-Terrors) der Kinder," *Jahrb. f. Kinderh.*, Leipzig, 1875, n.F., viii. 153.—27. Still, G. F. "Observations on *Oxyuris vermicularis* in Children," *Brit. Med. Journ.*, 1899, i. 898.—28. Tompkins, E. L. "Night-Terrors," *Amer. Journ. Obstet.*, N.Y. 1899, xl. 485.—29. Wood, H. C. "Nervous Diseases," Pepper's *System of Medicine*, London, 1893, i. 664.

L. G. G.

MENTAL DISEASES

Introduction.

Vice, Crime, and Insanity.

Idiocy and Imbecility.

Mania.

Acute Delirium.

Toxic Insanities.

Alcoholic Insanity.

Insanity and Surgical Operations.

Epochal Insanities

Insanity and Epilepsy.

Recurrent Insanity.

Melancholia and Hypochondriasis.

Mental Stupor and Catalepsy.

States of Mental Weakness.

Systematised Delusional Insanity.

Hypnotism: and the Treatment of Insanity and Allied Conditions by Suggestion.

Criminal Lunacy in England.

English Law and the Practice of Lunacy.

MENTAL DISEASES

INTRODUCTION

By G. H. Savage, M.D., F.R.C.P.

The following division of this work is devoted to the consideration of unsoundness of mind and of insanity, which, as I shall point out, are not the same thing: for there is much unsoundness of mind which does not seriously affect the relationship of the individual to his circumstances, and which cannot therefore be considered as insanity; and though insanity is always believed to be connected with some disorder or disease of the nervous system, it will be seen in the study of moral insanity that in those suffering from this and some allied forms of disorder, little change in the ordinary intellectual processes can be demonstrated.

A complete and satisfactory definition of insanity is wanting. I submit those of Bucknill and of Maudsley as well as my own: "Insanity is a condition of mind in which a false action of conception or judgment, a defective power of will, or an uncontrollable violence of the emotion and instincts have separately or conjointly been produced by disease" (Bucknill). "By insanity of mind is meant such derangement of the leading functions of thought, feeling, and will, together or separately as disable the person from thinking the thoughts, feeling the feelings, and doing the duties of the social body in, for, and by which he lives" (Maudsley). Insanity is such a disorder or disease of the nervous system as prevents the individual from reacting normally as a member of the society to which by birth and education he belongs. Conduct or behaviour in relationship to society is the gauge. It is not all insanity which requires outside control.

Unsoundness of mind may arise from some slighter defect of the nervous system which interferes in a minor degree with the normal reaction of the patient to his circumstances, but which need not effect his social life in any important way or degree.

Great difficulty is found in distinguishing the normal and the abnormal reaction of the individual to his circumstances, and hence also in arranging and classifying such reactions. Classification, as we shall see later, is, and for the present must be, provisional, and meanwhile must rest mainly upon considerations of custom and convenience.

The legal test of insanity is found in conduct; mere disorder of mental processes has little weight as a legal test, as will be seen when we study the relations of insanity and crime.

The insane, out of harmony with their social surroundings, are not necessarily in opposition to them; the criminal, on the other hand, is in direct voluntary antagonism to the laws of social life. Another important distinction between insanity and crime is that the criminal is supposed to be conscious of the nature and quality of his acts; to know that they are injurious or noxious, and worthy of punishment; and to be able to control them. An insane person may cause the same injury to society as a criminal; but, seeing that he is not able to control his act, or is impelled by some false idea for which he is not responsible, or is ignorant of the nature and quality of his act and its consequences to himself and others, he is not punished. In the one case the person is supposed to be able to recognise right and wrong, and to act accordingly; in the other case he is not able so to distinguish and to decide.

Though criminality and insanity may approach each other, yet it must not be supposed that all insanity is allied to crime; and I think serious harm still comes of the prevalence of the legal view of insanity. Some mentally degenerate persons are insane, and some are criminal. Unhealthy circumstances, including bad education, may lead to one aberration or the other; yet many of the insane are as far from being criminals as are the most healthy-minded.

Insanity and sanity being relative terms, the standard to be applied cannot be a general one, it must be individual. The sanity of the conduct of a person must be gauged by conduct. Insanity has in most cases to be reckoned as a deviation from the ordinary line of conduct; the person is not "himself," he is no longer able to understand and respond to stimuli in the way he did. Sanity, then, is a question of time and degree; the patient may conduct himself reasonably to-day and insanely to-morrow. Insanity, so far as it depends on disease of the brain, is related to changes in the fore-brain—that is, of the highest cortical centres; it may be due to organic alteration of structure in these centres, or it may follow temporary alteration of nutrition in the same areas; these alterations in nutrition may arise from bodily disease, the highest nerve-centres being the signal points of the disease; or the malnutrition may come from poisons generated in the body, as in uraemia or septicaemia, or from poisons derived from without, such as syphilis, alcohol, lead, or malaria.

Insanity may depend on defective power of accommodation of parts of the nervous apparatus to their surroundings; in these cases there is not exactly disease of organ or tissue, there is simply unfitness for the work. Such unfitness may be due to original defect, or to insufficient or improper education; in such defects of accommodation may lie moral, sensory, or physical incapacity.

Insanity, then, may be associated with disease of the brain, or with disease of the body indirectly affecting nutrition or growth of the brain; the insanity in this case being the mental expression of the bodily disease: or it may be due only to maladjustment of the individual to his surroundings.

It is generally assumed that though any one may suffer from the coarser forms of brain disease, such as tumour, certain persons only, the so-called "neurotics," can become insane. This is not true without qualification. It is probable that in the neurotic the nervous system is more likely to be disturbed by general diseases than it is in others; such persons are certainly more liable to delirium with febrile diseases, and I believe are more likely to be affected by general infective causes, such as syphilis, than others. On the other hand, persons of highly neurotic instability do not always tend to insanity, and in the neurotic who become insane special forms of insanity arise (*vide* art. "Neurasthenia," p. 747). Many highly neurotic persons become permanently invalided by hypochondriasis or hysteria, yet do not go mad. Neurosis may be acquired by the individual if exposed to its special conditions.

What is meant, then, by neurosis, and what is its relation to insanity? I consider a neurotic person to be one whose nervous instability causes him to react unduly to stimuli, such reactions being called mental or nervous symptoms. Neurosis arises under certain conditions of general and nervous degeneracy; as a tendency to nervous weakness it may be transmitted, and it may vary greatly both in form and in degree.

Hereditary neurosis generally originates in some physical weakness in the parents, such as insanity, epilepsy, intemperance, old age, wasting diseases (phthisis and cancer), damage to the higher nervous system by injury or by rapid and premature exhaustion of nervous energy, marriages contracted between aged parents, or between parents widely differing in age. Mere consanguinity, when both parents are healthy, is not enough of itself to generate neurosis.

Neurotic children may exhibit their nervous weakness in various ways: there may be want of brain mass, or there may be some inability on the part of the brain or some of its parts to develop; there may be sensory loss of one kind or another which hinders or prevents healthy nerve-stimulation from without; and such neurotic children may present degrees of idiocy or imbecility.

A minor degree of neurosis may be seen in a restless reaction to all stimuli from without which prevents storage of perceptions, and thus modifies or arrests mental growth from defective memory of experience; neurotic weakness may also be seen in "impulsive" reaction to outside stimuli, which may even cause convulsions. The restlessness of the neurotic is well marked throughout the years of growth and development; and is seen in loss of control, want of power of application, lack of moral sense, somnambulism and night-terrors, and a tendency to automatism. Though restlessness, loss of control, and undue tendency to react to stimuli are the most evident symptoms, those most dangerous socially are the moral defects and perversities; some neurotics have no sense of truth or honesty, and no altruism; they are cruel, destructive, and sensual. In some such patients special aptitudes appear to be developed at the expense of the other faculties: of such aptitudes are the memory for isolated facts; the power of musical reproduction, and of rapid mental calculation; thus musical prodigies and calculating boys may be weak in mind, or without moral sense.

The neurotic youth, who may have exhibited some of the abnormalities described, may later exhibit noteworthy and characteristic peculiarities of his own. He is alternately exalted and depressed, full of plans which never come to anything; he is self-asserting and conceited; then will-less, self-indulgent, and indolent. With the period of exaltation sexual desire becomes dominant, and may lead to immoral and degraded habits.

The neurotic and nervously degenerate who become insane exhibit a special tendency to recurrent insanity, and to the various forms of systematised delusional insanity. The natural tendency of highly neurotic families is to extinction; and though a slight amount of neurosis in a family may be associated with brilliancy, a little more leads to destruction. Neurosis may be eliminated from a family or modified by healthy marriage; if modified it may reappear as chorea, asthma, diabetes, gout, and, probably, in an undue susceptibility to fevers and toxins.

Genius and eccentricity are said to be allied to insanity. Genius, of some kinds, is not infrequently met with in neurotic families, and occasionally a man is at the same time a genius and subject to insanity. Poets, artists, and those concerned chiefly with works of the imagination are most liable to symptoms of insanity, the blaze of brilliancy being often followed by a gloom of nervous exhaustion. Here again the genius of insanity is not the genius of hard work and steady application, but of uncontrolled reaction to stimuli. Little genius, indeed, if any, is met with in asylums, at any rate among the chronic lunatics. The insane have no remarkable powers, either bodily or mental; they may disregard general opinion, or may be so self-absorbed as to appear to be gifted with undue powers; but essentially all insanity is weakness. Thus some persons, in bursts of unreasonable excitement, expend their nervous energy in a short period, leaving themselves for a time nerve-bankrupt. Genius is in some cases associated with eccentricity or moral defects of various kinds, though this is by no means the rule.

The eccentric person is regardless of the smaller conventions of society: he is not injurious to society, and he is not ignorant of the nature of his acts; he is more isolated than the normal, but less so than the insane. Just as genius does not connote eccentricity, so eccentricity connotes neither genius nor insanity. The eccentric person is often of neurotic stock, but, in the majority of cases, he does not pass into insanity. In some cases, however, the conduct of the eccentric person is such that for his own sake he needs especial care, and to this extent he may be regarded as insane.

The relation between criminality and insanity is specially treated hereafter (p. 842). I shall at once proceed, therefore, to the classification and causation of insanity.

Classification.—Though the basis of any scientific classification of mental disorder should be the pathological changes in the nervous system which give rise to insanity, we are not yet advanced enough in neuropathology to be able thus to classify insanity.

There may be classifications by general similarity of symptoms, such as mania, melancholia, or dementia; classifications by general causation, such as alcoholic, puerperal, adolescent, or senile; and classifications on mixed principles, such as that adopted by the Royal College of Physicians, in which the form of the insanity is qualified by the ascribed cause—thus, Mania—puerperal, alcoholic, etc. Though this last method has some advantages, we think the classification suggested by Drs. Clouston and Mercier for the Educational Commitee of the Medico-Psychological Association is better. It is as follows:—

1. States of mental weakness—(*a*) primary, idiocy and imbecility; (*b*) secondary, dementia. 2. Stupor. 3. Depression. 4. Exaltation and excitement. 5. Systematised delusions with hallucinations. 6. Impulsive and moral insanity. 7. General paralysis.

Classification proposed at the Congress of Psychiatrics taken from Bianchi's book:—

1. Congenital Psychoses.—Arrests and deviations of psychical development; phrenasthenia; moral insanity; psychopathia sexualis.

2. Simple Acute Psychoses.—States of mania; states of melancholia; amentia; sensory psychoses.

3. Chronic Psychoses, Primary and Consecutive.—Paranoia; periodic psychoses; states of dementia, (*a*) senile, (*b*) secondary.

4. Paralytic Psychoses.—Classical paralytic dementia; luetic dementia; alcoholic dementia; encephalomalacic dementia.

5. Psychoses from Neuroses.—Epileptic; hysterical; neurasthenic; choreic.

6. Toxic.—Pellagrous; alcoholic; morphinic; cocainic, etc.

7. Infective Psychoses.—Post-influenzal; enteric, etc.; syphilitic; acute delirium.

Causation of Insanity.—The causes which give rise to insanity vary greatly with the individual. Probably nearly all insanity depends on some alteration in the most developed parts of the fore-brain.

Causes cannot be clearly divided into physical and moral. The usual division of causes is as follows:—Predisposing and exciting; physical and moral (that is, psychical); simple and complex.

Similar insane courses of conduct may depend on disease of different kinds affecting the same parts of the nervous system. The brain, like other organs, has its own ways of expressing disease; so that various causes may produce identical forms of insanity. Mental disorder may arise from temporary malnutrition of the fore-brain; it may follow decay of the same parts of the brain, or it may depend on incapacity to accommodate to surroundings, though no structural disease of nervous system is discoverable. Temporary alteration in the finer structures of the brain may arise from direct injury or local change in the brain itself; or it may follow toxic influences derived from without,—as in fevers, alcoholism, or from within,—as in anaemia, uraemia, and the like.

Insanity may follow decay of the brain, due to structural dissolution or premature decay, or coarse changes resulting from tumours, apoplexies, arterial changes, and the like.

Remoter causes of insanity are either general or special. Among the general causes we have to consider whether particular races are more liable to mental disorder than others. It is clear that, as insanity is a deviation from the normal mental standard, this deviation will vary according to the state of civilisation of the time and place. With developing complexity of society there will be increase of mental disorder, not necessarily from increased mental work but from increase in the variety of relationships. Insanity is not more common among the southern and more excitable races than among the more phlegmatic. The habits of a nation have more to do with the tendency to disorder than the race. Those living more simple temperate lives suffer less than those living in cities who take much animal food and alcohol. Though country life is more free from some causes of insanity than city life, yet in the country there are other and special causes of mental stress. Solitude and lack of external stimulus tend to mental disease. Some forms of cerebral disease, such as general paralysis of the insane, are almost unknown among those who live away from cities, who take little animal food, and who are less liable to syphilis.

The religion, as part of the civilisation of a nation, plays some part in colouring the insanity, and to a smaller extent has an influence in producing it: the Protestant religion, for instance, has its special mode of colouring melancholic feelings.

Whether there is any marked and serious *increase in insanity* in England is a question continually asked; and I think it must be admitted that there is an increase, but not to an alarming extent.

At present (January 1909) there are in England about 32 recognised persons of unsound mind per 10,000 of the population (31·15 males, 33·80 females). The nominal increase greatly depends on the accumulation of chronic cases; on the collection in asylums of many harmless dements who formerly would have been retained in workhouses; and on the reception into county asylums of many harmless persons who, but for the increase in number and comfort of asylums, would be kept at home. A considerable number of patients belonging to the upper and the middle classes are still kept at home, and are thus not recognised by the Commissioners in Lunacy. The increase reported by the Commissioners in their report for 1908 during the previous year was 2096; this increase may be compared with the increase of 2009 in 1906, of 2150 in 1905, and 2630 in 1904. Particulars as to distribution and general statistics will be found in the official reports.

Sex.—There is a larger number of insane women in England than of insane men; in January 1908 the percentage was 46·3 males and 53·7 females; the general proportion in the population being 48·8 males and 51·2 females. The men being more subject to incurable forms of insanity do not provide so many recurring cases as the women do. For instance the male general paralytics die, whereas the puerperal patients generally recover and may relapse.

There are special conditions, both physical and moral, which dispose to insanity; in women, menstruation with its irregularities, childbirth, and change of life are potent influences. The stress of poverty and ill-treatment fall most heavily on them; and women who become widows are specially exposed to misfortune. In looking through the returns we find that over half the insane are married, which is about the same proportion as that met with in the general population.

The relationship of *age* to insanity is well marked; any form of mental disorder arising in childhood tends to arrest mental growth, and therefore to produce idiocy or imbecility or feeble-mindedness.

Though rare, acute insanity may arise in early childhood. I have seen cases of acute mania in children under seven; and it is not uncommon to meet with precocious development of sexuality leading to mental disorder in very young children. Melancholy is not very rare in children of from ten to fifteen, and may end in suicide. Stupor is also met with at similar ages, though this is rare. Hallucinations of the senses occur in very early youth, and may be the starting-point of organised delusions; such cases almost always belong to very insane stock.

TABLE FROM APPENDIX A. TO SIXTY-SECOND

Shewing for England and Wales :—(*a*) The number of patients in county and borough asylums,
licensed houses, who were living in those institutions on 31st December 1906. (*b*) The number of
number of deaths during 1906 to the number of patients living, on 31st December 1906, in the
tion to the whole population during the year 1906 (kindly furnished by the Registrar-General).

	Ages.							
	Under 5.		5—9.		10—14.		15—19.	
	M.	F.	M.	F.	M.	F.	M.	F.
(*a*) Number of patients living on 31st December 1906 in—								
County and borough asylums	10	6	184	96	330	208	952	765
Registered hospitals (excluding idiot establishments)	..	..	..	..	..	1	10	6
Metropolitan licensed houses (excluding idiot establishments)	..	..	..	..	..	1	5	9
Provincial licensed houses (excluding idiot establishments)	..	..	..	..	..	1	5	10
Naval and military hospitals	..	..	..	..	..	..	1	..
Criminal asylums	..	..	..	..	..	..	3	1
Total	10	6	184	96	330	211	976	791
Idiot establishments	3	2	107	69	314	153	318	177
GRAND TOTAL	13	8	291	165	644	364	1294	968
(*b*) Number of patients who died during the year 1906 in—								
County and borough asylums	..	2	10	11	21	12	84	56
Registered hospitals (excluding idiot establishments)	..	..	..	..	..	..	..	..
Metropolitan licensed houses (excluding idiot establishments)	..	..	..	..	..	..	..	..
Provincial licensed houses (excluding idiot establishments)	..	..	..	..	..	..	..	..
Naval and military hospitals	..	..	..	..	..	..	..	..
Criminal asylums	..	..	..	..	..	..	..	..
Total	..	2	10	11	21	12	84	56
Idiot establishments	..	..	1	3	9	2	8	5
GRAND TOTAL	..	2	11	14	30	14	92	61
(*c*) Ratio (per 1000) of deaths during 1906 to number of patients living on 31st December 1906 in—								
County and borough asylums	..	..[1]	54·3	114·6	63·6	57·7	88·2	73·2
Registered hospitals (excluding idiot establishments)	..	..	..	..	..	..	..	..
Metropolitan licensed houses (excluding idiot establishments)	..	..	..	..	..	..	..	..
Provincial licensed houses (excluding idiot establishments)	..	..	..	..	..	..	..	..
Naval and military hospitals	..	..	..	..	..	..	..	..
Criminal asylums	..	..	..	..	..	..	..	..
Total	..	..[1]	54·3	114·6	63·6	56·9	86·1	70·8
Idiot establishments	..	..	9·3	43·5	28·7	13·1	25·2	28·2
GRAND TOTAL	..	..[1]	37·8	84·8	46·6	38·5	71·1	63·0
(*d*) Ratio (per 1000) of deaths in the whole population to the whole population (estimated for the middle of the year) during the year 1906	49·3	41·2	3·4	3·6	2·0	2·2	3·0	2·7

[1] The ratios in these divisions are not given, as, owing to the small

RT OF THE COMMISSIONERS IN LUNACY.

ered. hospitals, idiot establishments, naval and military hospitals, state asylums, and
ts who died in those institutions during the year 1906. (*c*) The ratio (per 1000) of the
institutions. (*d*) The ratio (per 1000) of the number of deaths amongst the whole popula-
ged according to ages.

Ages.															Total.	
24.	25—34.		35—44.		45—54.		55—64.		65—74.		75—84.		85 and upwards.			
F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
1984	7907	7678	10,049	10,849	9241	11,479	6681	8960	3424	5175	909	1663	89	196	42,101	49,059
39	166	162	225	271	244	360	210	301	124	212	41	82	2	11	1,049	1,445
14	56	89	100	167	122	223	141	228	87	168	27	79	5	10	562	988
31	99	119	151	223	155	271	152	226	77	176	32	72	5	15	698	1,144
..	26	..	40	..	23	..	31	..	24	..	11	..	1	..	164	..
3	85	37	134	49	147	60	122	40	76	12	24	3	3	1	611	206
2071	8339	8085	10.699	11,559	9932	12,393	7337	9755	3812	5743	1044	1899	105	233	45,185	52,842
92	178	81	106	48	56	38	32	15	10	3	..	1	..	..	1,289	679
2163	8517	8166	10,805	11,607	9988	12,431	7369	9770	3822	5746	1044	1900	105	233	46,474	53,521
123	565	489	1,007	754	879	765	827	733	792	795	378	534	68	90	4,795	4,364
1	4	2	14	8	14	11	13	10	12	15	8	10	2	4	67	61
..	4	5	9	4	15	11	13	20	8	23	12	14	1	4	63	81
1	2	6	6	12	13	7	5	15	13	15	2	15	1	2	42	73
..	4	..	11	..	2	..	3	..	1	..	3	..	..	..	24	..
..	1	..	2	2	2	1	4	..	3	1	3	..	3	1	18	5
125	580	502	1,049	780	925	795	865	778	829	849	406	573	75	101	5,009	4,584
1	1	2	2	3	4	5	1	2	..	1	..	..	..	..	27	24
126	581	504	1,051	783	929	800	866	780	829	850	406	573	75	101	5,036	4,608
62·0	71·5	63·7	100·2	69·5	95·1	66·6	123·8	81·8	231·3	153·6	415·8	321·1	764·0	459·2	113·9	89·0
..[1]	24·1	12·3	62·2	29·5	57·4	30·6	61·9	33·2	96·8	70·8	195·1	122·0	..[1]	..[1]	63·9	42·2
..	71·4	56·2	90·0	24·0	123·0	49·3	92·2	87·7	92·0	136·9	..[1]	177·2	..[1]	..[1]	112·1	82·0
..[1]	20·2	50·4	39·7	53·8	83·9	25·8	32·9	66·4	168·8	85·2	..[1]	208·3	..[1]	..[1]	60·2	63·8
..	..[1]	..	275·0	..	..[1]	..	..[1]	..	..[1]	..	..[1]	..	..	..	146·3	..
..	11·8	..	14·9	40·8	13·6	16·7	32·8	..	39·5	..[1]	..[1]	..	..[1]	..[1]	29·5	24·3
60·4	69·6	62·1	98·0	67·5	93·1	64·1	117·9	79·8	217·5	147·8	388·9	301·7	714·3	433·5	110·9	86·7
10·9	5·6	24·7	18·9	62·5	71·4	..[1]	..[1]	..[1]	..	..[1]	..	..	..	..	20·9	35·3
58·3	68·2	61·7	97·3	67·5	93·0	64·4	117·5	79·8	216·9	147·9	388·9	301·6	714·3	433·5	108·4	86·1
3·3	5·6	4·7	9·5	7·8	16·8	13·0	33·2	25·3	68·7	56·1	136·9	123·2	319·5	287·4	16·4	14·4

of patients in them, they are likely to mislead.

Adolescent general paralysis of the insane in my experience is always associated with parental syphilis. There is ground for thinking that heredity plays an important part in the early onset of insanity. Persons coming of very neurotic stock tend to insanity, especially at the critical periods of life; that is at puberty, adolescence, reproduction, the climacteric, and in senility: such persons also tend to systematised delusions and recurring insanity.

It will be seen from the Commissioners' reports that of fresh cases 82·9 per cent were first attacks.

The largest number of cases of insanity occurs between the ages of twenty-five and fifty, that is during the period of greatest mental activity. Both puerperal insanity and general paralysis occur chiefly between these ages. The true delusional cases are, as a rule, first brought under notice about the age of thirty. With the menopause there is in the neurotic a special tendency to break down, and this is well marked in those who have had other symptoms of mental disorder, particularly if such disorder had been connected with the reproductive function.

In some families the nervous system has a special tendency to wear out; the members of such families as a rule becoming either demented or melancholic in old age. In extreme old age, however, as in childhood, mania or stupor may occur.

The *social position* may act in some ways as causative of insanity. Occupations which interfere with healthy sleep, or which keep the nervous system always on the strain, tend to produce insanity; thus Stock Exchange and similar work is dangerous, but it is hard to know how much depends on the excitement and how much on free living. In England I have recently seen many patients in whom the worry of unprofitable farming seemed to be the cause; a constant anxiety as to the future, more particularly when nothing can be done to prevent mishap, may lead to sleeplessness, indigestion, and nervous break-down.

The position and the profession of the individual will colour the nature of the insanity: a doctor tends to hypochondriasis, a parson to remorse; a man is more egotistical, a woman more altruistic in her morbid bent. The life of a governess in England tends to produce insanity; but here again we have to recognise that governesses are often gifted with active and unstable nervous systems; moreover, their lives in too many cases are narrowed out of proportion to their powers and to their desires. Beggars are not very liable to insanity, but prostitutes provide a large number of lunatics.

Solitary confinement has been credited with the production of a good deal of insanity among prisoners, but this has not been proved. The criminal classes are nearly allied to the insane, and it is not astonishing that a good many prisoners should become mad. Prisoners differ among themselves in their insanities, but I believe that in them there is a great tendency to organised delusions, such as those of persecution.

The *season of the year* may have something to do with the incidence of the disorder. In England the largest proportion of admissions to asylums is in the early summer, but this does not necessarily imply that these cases began then. There is no known relationship between the phases of the moon and insanity.

Heredity.—Insanity in a parent may reappear as insanity in a child; the insanity in a mother tends to reappear most commonly in the daughters; that in the father tends nearly equally to both sexes, but is not so potent as that of the mother.

Insanity in a parent may reappear in the children, as idiocy or imbecility; or physical or mental defects which have been described as degenerative—such as epilepsy, chorea, asthma, or irregular nervous discharge of one kind or another. It is in the upper classes that heredity is most commonly traced as a cause of insanity. Mere consanguinity is not a cause of idiocy or insanity unless there be disease or degeneracy in the family. Neurotic and insane heredity may cause eccentricity, moral obliquity, tendency to delusions, imperative ideas, and recurring attacks of various forms of insanity. I believe that in fully one-third of all the cases of ordinary insanity a history of insanity is to be detected in the family. In the case of general paralysis of the insane, however, this is much less marked.

Insane persons may have perfectly healthy children; and in families in which most of the children are idiotic or imbecile, it is not uncommon to find the parents apparently normal. The danger to the children, in my experience, is directly in relation to the recency of the insane attack; thus a father, having begotten healthy children, has an attack of insanity, and while still insane, or shortly after the passage of the acute symptoms, begets a child which becomes insane; but later he may beget healthy children. Again, the child of a mother who has an attack of puerperal insanity may be affected, though the mother did not become actually insane till after the birth of the child. The child of a degenerating parent, such as one in the early stage of general paralysis of the insane, or one suffering from senile mental changes, is likely to be idiotic or unstable.

The relation of drunkenness in the parent to insanity in the child is not quite clear; but no doubt a drunken parent not infrequently begets weak-minded, idiotic, alcoholic, or criminal offspring. It has been said that a child begotten during drunkenness runs a special risk, but I can hardly accept the evidence on this point. In some rare cases the form of insanity seems to be transmitted directly; this is seen in some cases of melancholia, and in some of systematised delusional insanity; in these latter instances similar minds living under similar conditions originate similar delusions.

The neuroses are in most cases interchangeable, so that one form of neurosis may appear in the parent, another in the child; an insane parent may have an epileptic child, or an epileptic parent an insane child. There may be certain other alternations; thus a neurotic parent may have diabetic offspring, or conversely. Megrim, asthma, and gout may alternate in a similar way. In strongly inherited insanity there is a tendency to sudden outbreaks of disorder, and a tendency even to a certain toleration of attacks; so that a highly neurotic person may recover from dozens of attacks of acute mania from which the non-neurotic person would not have mentally recovered. Remissions and recurrences are common. Neurotic heredity tends to delirium, somnambulism, moral perversity, and general nervous instability.

Education, if properly conducted, never leads to insanity. Spoiling may cause some insanity, but as a rule the spoiling itself consists in organic defect. In premature mental effort the danger is in interfering with natural growth. The education of the present day may do harm in immature youths who come up to a city, where they live a narrow life, cram themselves during their few spare hours with the rudiments of a modern language and with shorthand; they become sleepless, dyspeptic, and break down with depression or delusions; but fag and cram are not education. Too bookish an education, again, may be harmful when the general hygienic conditions are bad. Generally speaking, "overwork" is work on too narrow lines. I have seen overwork of the following kind do harm:—A parent sets her clever daughter to learn everything at once; to study science, languages, music, art, dancing, riding, and the rest; and, to balance all, encourages bicycling and hockey in order that, by the diversion of nervous force, a balance may be conserved; but excessive physical training has its dangers also, and I think I have seen over-muscular training in unsuitable subjects act as the starting-point of neurosis.

Broad and general education does good; but it must be remembered that the neurotic child needs special and individual education; as a rule such children have to be kept back and not forced, the great purpose of their education being less to acquisition and more to inculcation of habits of order and steadiness.

Such, then, are the general and special remoter conditions of insanity, which prepare the way for the proximate causes which may act variously, some directly on the brain itself, others on the general nutrition; some act instantly—this is rare; others by prolonged strain; others, again, by frequency of recurrence.

No essential difference can be made between moral and physical causes; for instance, either a mental shock or a blow on the head may produce a similar disorder of nutrition. We all know how a fright or a blow may arrest the heart; so it is with the rest of the body: a fright, it is said, may affect the mother's milk so that it injures the child; and similarly the brain nutrition may be injured by a shock, such as reception of bad news, or by the prolonged effect of a constant dread of impending evil.

The more immediate causes of insanity as registered by the Commissioners in Lunacy are tabulated as follows:—

CAUSES OF INSANITY

MORAL

Domestic trouble (including loss of relatives and friends).
Adverse circumstances (including business anxieties and pecuniary difficulties).
Mental anxiety and "worry" (not included under the above two heads), and overwork.
Religious excitement.
Love affairs (including seduction).
Fright and nervous shock.

PHYSICAL

Intemperance in drink.
,, sexual.
Venereal disease.
Self-abuse (sexual).
Over-exertion.
Sunstroke.
Accident or injury.
Pregnancy.
Parturition and the puerperal state.
Lactation.
Uterine and ovarian disorders.
Puberty.
Change of life.
Fevers.
Privation and starvation.
Old age.
Other bodily diseases or disorders.
Previous attacks.
Hereditary influence ascertained.
Congenital defect ascertained.
Other ascertained causes.
Unknown.

But few remarks on these causes will be necessary. Domestic anxiety falls most frequently and most heavily on women, business anxiety on men;—though many women suffer as much or more than men from the stress of business, they are less exposed to it. The *domestic anxiety* is frequently met with in women under such conditions as these:—A wife nurses a paralysed husband night and day for some years; then, as a result of nervous exhaustion, she can neither sleep nor eat, becomes full of notions that she might have been more attentive, or that some slight duty was neglected, and that this led to his death: or a daughter nurses her mother till she dies; then, left alone to think over the illness, she thinks she had wished for her mother's death, and so perhaps hastened her end. The sudden reception of ill news may cause stupor, melancholia, or acute mania.

The *worry of business* affairs is generally associated with defective digestion, want of sound sleep, and imperfect respiration; these lead to defective nutrition of the brain and a liability to further upset. Repair of brain cannot go on normally under these conditions, and either progressive degeneration or temporary acute disorder may occur.

Religious excitement is like the match to tinder; given nervous instability, religious excitement may easily start disorder; but in England very little insanity nowadays springs from this cause. Religion often colours delusions and gives rise to insane sayings or doings; but the disorder is rooted in other causes.

Excitement or *shock*, either of a painful or, more rarely, of a pleasant nature, may suddenly upset the nervous balance, and may start general disorder which may lead to insanity. Fright may lead to stupor or to acute delirious mania; joyous emotions in excess occasionally tend to mania or to stupor.

Among the more obvious causes of insanity *intemperance in alcohol* is given as the most common. It is found that insanity among the working-classes increases rather with abundant work and free living than with poverty and privation. Those who have had previous attacks of insanity are more easily upset by drink than others who have not had previous attacks. Some neurotic persons may be driven mad by a sudden bout of drinking; others are upset after long series of debauches. Any cause of delirium may be a cause of insanity.

Love affairs have very serious effects on some persons. I have pointed out that a very large number both of men and women are upset for a time by marriage engagements. It is certain that premature sexual desire and excessive pondering on sexual matters tend to many nervous evils. In many cases, however, the so-called love affairs, which are alleged as causes, are delusions. Men, but more particularly women, who have not exercised the reproductive function before middle life, tend to special forms of mental disorder of a delusional kind. Seduction leads in not a few cases to temporary mental disorder, but not so frequently as might have been expected; for the kindly plea of insanity in many infanticides, like the verdict of temporary insanity in suicide, is a pious fraud.

Excess in *sexual indulgence* is so difficult to measure that I always feel inclined to regard excess as a symptom rather than as a cause. In some recently married persons such excess produces profound hypochondriasis or stupor; in others a morbid and insatiable eroticism. Powerful lust is an early symptom of ordinary mania and of general paralysis.

Venereal disease produces insanity in some instances by the mental worry of it; in others syphilis causes marked mental disease of various forms, such as syphilophobic melancholia; or there may be a morbid self-consciousness due to some syphilitic lesion which has left its scar visible: it may lead to tabes dorsalis, with various insane interpretations of the symptoms of this disease; or it may give rise to degenerative changes in the arteries, causing organic dementia or progressive (general) paralysis. Congenital syphilis may give rise to epilepsy, idiocy, or adolescent general paralysis. Syphilis is, in my opinion, the chief cause of general paralysis of the insane.

Self-abuse is one of the most commonly cited causes of adolescent insanity; it certainly is an accompaniment, and occasionally the cause of such disorder. It occurs very frequently in both sexes, and the more sensitive the person the greater the danger of the practice; not so much from the physical harm of it as from the notion of its being debasing and sinful. The indulgence itself is of less moment, except in the very young or very weak; the thoughts about it and dreads of its consequences are more harmful. In some cases, in which the habit began in infancy and was continued to mature years, there is a defect of true sexual desire, or indeed an inversion or perversion of this feeling. Most of such sexual perverts belong to neurotic families. Precocious sexuality with masturbation interferes with normal mental growth; and such youthful indulgence produces loss of memory, moral perversion, hypochondriasis, systematised delusional insanity, stupor, or hysterical instability; but, to speak generally, too much importance may be attributed to the habit as a cause of insanity; and I prefer to look upon it, in many cases at any rate, as a symptom rather than as a cause. At the climacteric in women the habit is a very marked symptom, and leads to great moral depression. In puerperal insanity, too, this symptom is common and may retard recovery. With senility it is not uncommon, and tends to further moral and physical decay.

Over-exertion is not often met with as a simple cause of insanity; but in some immature persons I have seen training for athletic sports and racing, whether on foot or on bicyles, lead to acute mental disorders.

Sunstroke is not, as a rule, looked upon as an efficient cause of insanity in England; but I have no doubt that during the summer months this alone, or combined with over-exertion, excitement, and alcohol, may start mental disorder. In the tropics various forms of insanity follow insolation. Various degrees of mental weakness may follow an over-exposure to the sun; this may range from simple loss of recent memory to stupor or dementia. The symptoms resemble those met with in early general paralysis of the insane, and are not always easily distinguishable from those of this disease. The prognosis, however, is often very favourable.

Accident or injury may be local or general. The shock of a railway accident or a shipwreck may cause stupor or set up acute mania; injury, or blood-loss in neurotic subjects, may cause a recurrence of insanity. The shock of a surgical operation may be a cause of insanity (*vide* art. p. 921). Local injury to the head in drunkards may start a delirium which may persist; or an unconsciousness which may give rise to some permanent or temporary loss of memory, so that the patient has no knowledge or recollection of his whereabouts. Injury to the head may give rise to mania, to partial or temporary dementia; or it may be the starting-point of general paralysis of the insane in predisposed subjects.

Pregnancy.—Parturition, the puerperal state, and other Epochal Insanities are discussed in subsequent articles (*vide* p. 926).

Uterine and ovarian disorders, though so frequently met with in general practice, yet have in my experience a very small effect in the production of insanity. Amenorrhoea is a frequent symptom in melancholia, and in allied states of physical and nervous depression; this may give rise to such a delusion as that the patient is pregnant; that she is an immaculate but pregnant virgin; or is pregnant by the devil, and so forth. In many cases there is prolonged amenorrhoea in cases of mental disorder depending on nervous and not on general conditions. With amenorrhoea in the insane there is constantly disorder of feeling; the patient complaining of being unnatural, dead, or without human feeling. In many cases these symptoms pass with the return of the menses. With uterine and ovarian diseases there may be hallucinations of smell. Insanity is met with in some cases of ovarian disease, but there are no special symptoms of this condition. It is not common for the uterine or ovarian hypochondriac to become actually insane.

Fevers may set up delirium, which may pass into acute delirious mania or simple acute mania; more commonly, however, we meet with mental weakness following fevers. This is often well marked after enteric fever; in this case partial dementia, with loss of recent memory, is the most common. This may pass off completely or may leave permanent mental defect behind. The higher the temperature, and the more marked the delirium, the greater the danger of consequent mental disorder. In some cases a form of chronic delirium persists, which may be called chronic mania. Any febrile disorder giving rise to delirium may cause maniacal excitement, or mental weakness. A febrile disorder may relieve or mask mental symptoms already existing. Cholera may leave behind it profound depression, melancholia, or stupor. Influenza is due to a specific nerve poison which may set up any form of mental disorder; as a rule it gives rise to sleeplessness, and to various forms of nerve and mind pain, such as neuralgia or melancholia. It is a frequent cause of suicide. It tends to great loss of power of attention, and affects the memory. It frequently causes relapses and starts recurrence of attacks in chronic or recurrent insanity. In the already degenerating it accelerates degeneration, and in such persons it leads to dementia; in early general paralysis it accelerates the acute symptoms.

Privation and starvation are rarely seen singly as causes of insanity: we can only say that such causes acting on young persons may interfere with physical and mental development; in middle age they may cause nutritional defects leading to depression, and favour more rapid degeneration. In times of great social anxiety and distress there is no parallel increase in the amount of insanity.

Old age is associated with special forms of mental disorder. Old age is relative, and depends more directly upon the state of the arteries than upon lapse of years. In some families age is marked by nervous disorders, so that in succeeding generations similar symptoms of decay are met with in individuals about the same age; such symptoms are generally melancholia or mental weakness. With age, symptoms similar to those occurring in youth may reappear; such as loss of self-control, hysteria, and sexual faults. Though age, with signs of distinct mental loss, indicates an unfavourable prognosis, yet, if no such loss be manifest, old age is not necessarily a bar to recovery from acute mental disorders.

Other bodily diseases or disorders may give rise to insanity. The brain, like a master with many servants, may be badly served by one of them. It is not possible in this place to refer to all the *bodily diseases which may tend to cause insanity*, and I must content myself with referring to groups of disorders and to their effects.

Coarse brain disease may give rise to mental disorder, though, as a rule, the chief mental symptoms of such diseases as tumour, cerebral apoplexy, and the like are those of progressive mental weakness. In some cases of secondary malignant growths in the brain the symptoms resemble those of general paralysis. Syphilitic and other thickenings of the membranes may cause degrees of dementia, or may simulate general paralysis.

Insular sclerosis may lead to progressive mental and physical weakness hardly to be distinguished from general paralysis. An intracranial growth often gives rise to some degree of mental weakness, but occasionally more acute mental symptoms may be present.

With tabes dorsalis, which frequently occurs in neurotic subjects, there may be various delusions, generally of the "persecutional" kind; the patient attributing his pains and aches to the machinations of his foes.

With epilepsy there may be various forms and degrees of insanity (see p. 941); the tendency to dementia varying rather as the frequency than as the severity of the fits. With paralysis agitans there may be a marked tendency to mental weakness, while at the same time there are irritability and ideas of persecution and annoyance. With chorea there is almost always some mental weakness, which may pass into a very profound degree. Chorea in early years may leave permanent mental defect. Acute delirious mania may originate in chorea. With Huntington's chorea there is progressive dementia.

Disease of the sensory organs may lead to insanity. Deafness is not infrequently associated with suspicion, and with the notion that persons are referring to, talking against, or maligning the sufferer. Imperfect sight likewise may lead to illusion and delusion; hyperaesthesia may give rise to false ideas of space, and thus a patient may believe himself to be of prodigious size.

Diseases of the respiratory tract may give rise to insanity, or be associated with it. Whooping-cough, a nervous malady, may give rise to or alternate with some mental affection, and may be the starting-point of permanent feebleness of mind. Asthma may alternate with mental disorder, generally of a melancholic kind.

Phthisis is so distinctly associated with a particular form of insanity that this has been described as phthisical insanity (p. 939). In this form there is marked suspicion with fancies that the food is poisoned; in some cases as the lung disease increases the mental symptoms are mitigated. It is probable that a phthisical and neurotic union gives rise to very degenerate offspring. Meningitis may start mental defect and lead to imbecility. Any acute lung disorder, specially septic pneumonia, may give rise to delirium, and this may engender acute mental disorder.

Diseases of the circulatory system are frequently met with among the insane, but that there is any very direct and distinct causal relation between them seems doubtful. In some cases of neurotic instability a rapid weak pulse seems to be related to the restless instability of the nerves. A man's mental power and ability must be considered as the resultant of the brain and the circulation, and hence defective heart's action must be considered as an efficient cause of mental disorder. This disorder, however, will be found to vary greatly with the age and circumstances of the patient; in one instance, perhaps, the result is morbid self-consciousness, in another restless ill-considered action. I think that with heart disease which has already begun to affect the general circulation in neurotic subjects, there is often an exaggerated "self-feeling," which may give rise to hypochondriasis, or to false ideas of self-importance. In some cases anxiety and a feeling of impending ruin are associated with cardiac failure. Feeble circulation with congested aspect of hands is common in stuporose and adolescent states. Anaemia and chlorosis are often associated with defective will-power, loss of higher control, and morbid self-consciousness leading to melancholia.

Disorders of the reproductive organs have been already noted: in women such disorders tend to defect of feeling and to ideas of unworthiness; in men similar disorders lead to jealousy and to the notion of impotence, the former being dangerous from a homicidal and the latter from a suicidal tendency.

Kidney disease bears no recognised relation to insanity; in fact it used to be thought that kidney disease is not apt to occur in the insane. This is not true; but, although in Bright's disease there is a tendency to mental depression, there is no established connexion between any form of insanity and kidney disease. Recently a great deal of attention has been drawn to the connexion between melancholia and movable kidneys, and many cases have been described of recovery after surgical treatment of such cases. I have no experience to support this.

Diabetes often occurs in neurotic families; it may alternate with attacks of insanity, or it may be present as a result of progressive degenerative brain change. Temporary glycosuria may occur in puerperal insanity, and in some other forms of acute bodily disease.

Gout, like diabetes and asthma, may be replaced by an attack of insanity, generally of the melancholic form. Gout may be preceded or followed by mental disorder of a temporary kind. I do not recognise any special gouty insanity, but that gouty people have a tendency to mental excitability and to depression is certain, and this tendency may develop into mania or melancholia.

Cancer has been supposed to occur very frequently in neurotic families. I cannot give any statistics to prove or disprove this statement, but I very often meet with the association. Cancer may give rise to secondary brain disease, and thus to mental symptoms; and concealment of it, it may be for years, added to worry, pains, exhaustion, and sleeplessness, have often paved the way for an attack of melancholia in women.

Rheumatic fever may be associated with insanity; thus, during an attack of rheumatic fever the rheumatic symptoms may be replaced by insanity, which may again pass off to be replaced by the rheumatic fever. Rheumatic fever in the hyperpyrexial form may give rise to acute delirious mania or lead to mental weakness.

Chronic rheumatoid arthritis is not uncommon in neurotic families, but I know of no real connexion between the diseases.

Any febrile state may upset the mental balance and may lead to mania; and any febrile state may leave mental weakness.

Toxaemia, whether due to external poisons or to changes in the body itself, may give rise to symptoms due to more or less grave loss of higher control; and the symptoms will depend on the amount of change produced by the poison in the nerve-centres. (*See* Toxic Insanities, p. 904.)

Influenza, malaria, typhoid, cholera, syphilis, and the like may produce insanity of various forms and degrees. Similarly uraemia and cholaemia may cause nervous disorders. In some general diseases, such as myxoedema, disorder of the mind is well recognised. In myxoedema there is slowing of perception and reaction, defect of memory, and loss of judgment, which may result in simple mental weakness; or the malady may give rise to false ideas of personality, or of self-importance.

With Graves' disease there may be exaggerated self-consciousness, with fancies that persons are noticing the facial peculiarity, or with great fretfulness or mental depression. On the other hand, not very infrequently, acute attacks of mania occur during the course of the disease, and may lead to a fatal issue. Some or all of the symptoms of this disease may recur with recurring attacks of insanity, and with general paralysis of the insane exophthalmic symptoms may be present.

Skin disease of one form or another may be associated with insanity, leading through exaggerated self-consciousness to delusions of being watched or noticed, or that the sufferer is noxious to others.

Disorders of general nutrition and of digestion, by pain, worry, and sleeplessness, may give rise to mental disorder. But I am often surprised that so many of the causes of general malnutrition are not also more active as causes of insanity. The sufferer from gastric ulcer and the hysterical patient with anorexia nervosa as rarely become insane as the confirmed hypochondriac.

Malignant disease of the stomach may give rise to delusions of persecution, or to the belief that the bowels are closed. As a rule the insanities which may be called reflex—that is, depending on organs other than the brain—are either mental weakness, the result of malnutrition, or delusional, depending on false interpretations of sensory impressions.

Most bodily diseases indeed have certain mental features which may become so well marked as to interfere with the ordinary mental life, and to take their place as characteristic symptoms of the particular disease.

G. H. SAVAGE.

REFERENCES

1. BALLET, G. *Traité de pathologie mentale*, Paris, 1903.—2. BIANCHI, L. *Textbook of Psychiatry*, translated by J. H. Macdonald, London, 1906.—3. BLANDFORD, G. F. *Insanity and its Treatment*, 4th edit., London, 1892.—4. BUCKNILL, J. C. and D. H. TUKE. *Manual of Psychological Medicine*, 4th edit., London, 1879.—5. CHURCH, A. and F. PETERSEN. *Nervous and Mental Diseases*, 5th edit., Philadelphia, 1906.—6. CLOUSTON, T. S. *Clinical Lectures on Mental Diseases*, 6th edit., London, 1904.—7. CRAIG, M. *Psychological Medicine*, London, 1905.—8. DANA, C. L. *Textbook of Nervous Diseases and Psychiatry*, 7th edit., London, 1909.—9. KRAEPELIN, E. *Psychiatrie*, 6te Aufl., Leipzig, 1899.—10. *Idem. Lectures on*

Clinical Psychiatry, translated from the German revised edition, by T. Johnstone, London, 1904.—11. Lewis, W. Bevan. *Textbook of Mental Diseases*, 2nd edit., London, 1899.—12. Maudsley, H. *Pathology of Mind*, 2nd edit., London, 1895.—13. Mercier, C. *Textbook of Insanity*, London, 1902.—14. Robertson, W. Ford. *Textbook of Pathology in Relation to Mental Diseases*, London, 1900.—15. Savage, G. H. and E. Goodall. *Insanity and Allied Neuroses*, 4th edit., London, 1907.—16. Stoddart, W. H. B. *Mind and its Disorders*, London, 1908.—17. Ziehen, T. *Psychiatrie*, 3te Aufl., Leipzig, 1908.

G. H. S.

VICE, CRIME, AND INSANITY

By Charles A. Mercier, M.D., F.R.C.P.

It is sometimes alleged, and more often suggested, that a person who exhibits vicious, and especially criminal, conduct, is *ipso facto* insane; and it is more often alleged or suggested that vicious and criminal persons, if they are not insane, yet constitute special varieties of the human race—varieties which can be recognised and distinguished, not only by the peculiar conduct they display, but by distinctive physical characters, and by the fidelity with which they reappear in generation after generation, or "breed true." In no discussion on the subject with which I am acquainted is there any explanation or definition of the meaning or nature of vice, of crime, or of insanity; and, in the absence of any such definition, it seems that discussion on the subject is beating the air. Few people realise how easy, nay, how habitual, it is to attach different, and even inconsistent, meanings to the same word in different connexions in the same discussion. Without assuming any authority to fix the meanings of these terms for others, I shall therefore indicate the meanings that I intend to attach to them in the following pages.

Vice and crime have in common the character that they are reprobated and regarded as wrong by mankind. It is true that conduct which is regarded as vicious or criminal in one place, and by one community, may not be so regarded in another place and by another community; but this does not invalidate the statement that vice and crime are everywhere and by all men reprobated. It merely means that the standard of vice and crime varies among different communities, so that what is reprobated by one is tolerated by another, and looked on as praiseworthy by a third. The differences of standard are profound. In civilised communities matricide is abhorred as among the most dreadful of crimes. In some savage communities it is looked upon, in certain circumstances, as a duty. But however different the standards may be, in every community there is a standard of conduct, departure from which is reprobated by the community.

In determining the nature of vice and crime, we are therefore to discover, first, what modes of conduct are reprobated; and to narrow the scope of the inquiry, we will confine it to the modes that are reprobated by Western communities at the present day. First of all, we find those modes of conduct most vehemently reprobated that are directed against the integrity of the community itself; and this is a natural consequence of that struggle for survival among communities which is, in its turn, a phase of the struggle for life among individuals, and especially among stocks, or genetic successions of individuals. Of all the devices that have been brought into existence by the struggle for life among different stocks of animal organisms, none, it is safe to say, has been of any efficacy comparable with that of living in organised communities. The enormous size and strength of the *Megatherium* and the *Diplodocus*, the defensive armour of the gigantic armadillo, the powers of flight of the *Plesiosaurus*, the minuteness of certain animalcules, the remoteness of the great auk, have failed to preserve them from extermination. Of living forms, the whale and the elephant in spite of their size, many species of antelopes in spite of their speed, the lion in spite of its strength and ferocity, are dying out. But those insects that live in organised social communities flourish in undiminished numbers; and man, in whom social organisation has reached an unprecedented height, prevails over all other animals in spite of his nakedness, his want of weapons, his deficiency in strength and speed, his want of modes of concealment. It is often said, and truly said, that man owes his survival and his prevalence to the superiority of his mind; but this superiority of mind could never have been attained had the habits of man been solitary, like those of many of the lower animals. It is the habit of living in organised communities that permits of cultivation of crafts and science; and it is by crafts and science that highly-organised communities prevail over those that are less organised, that man prevails over all other forms of life, and is now extending his realm even over protozoa and bacteria, which have so long been his masters. This combination of man into highly-organised communities could never have taken place if it had not been prompted and supported by powerful instincts—instincts which are often of necessity antagonistic to those which subserve the preservation of the individual alone. If the individual components of a community were not ready and willing to sacrifice their own welfare and even, on occasion, their own lives for the benefit of the community, the community would be dissipated; and not only would it cease to exist as a community, but its component individuals, deprived of the immense advantages of common life, would soon perish. Hence it has come about that each individual in a community is not only instinctively impelled to modify his conduct in accordance with the needs of the community, as distinguished from his individual needs; but also he has developed in him, by gradual intensification through innumerable generations, an instinctive detestation of every act, and of every actor, by which the integrity of the community is imperilled. It is clear that, generally, those communities will have prevailed and become dominant whose members have acted together most harmoniously, and with the greatest singleness of purpose

It is an old observation that a city divided against itself cannot stand; and, manifestly, the city which is most completely of one mind will stand the firmest. Now, those communities that act together with the greatest efficacy will be *ceteris paribus* those in which the governing power is most authoritative and despotic; whilst those which will be the most firmly compacted will be those that think and act with the greatest uniformity. Hence the survival of the fittest will ensure the prevalence, in every successful community, of a hatred of those persons and of those acts that tend to diminish the power and authority of the ruler, and of those that tend to diminish the binding force of the motives that preserve uniformity in thought and action. The fiercest reprobation of the community will be directed against attacks on the established order of things.

The reason of the reprobation which is visited upon treasons—attacks upon the ruling power of the community—is manifest, and needs no explanation; and from what has just been said it will appear why the community has always reserved its fiercest reprobation and its direst punishments for those who attack its religion and its customs; for religion and custom are the two great binding forces which keep together primitive communities, and impose, upon violent, masterful, and self-seeking natures, a coercion which compels them to subordinate their self-regarding activities to the welfare of the community as a whole.

A good housekeeper, who does not like to set her butter on the table in a smeary state, keeps it in summer on ice. By cooling the mass—by diminishing the independent movements of the several component particles—she binds those particles more firmly together. When a smith desires to cut an iron bar, he first heats it to redness. By increasing the intrinsic movements of the component particles, he loosens their cohesion to one another. So it is with all aggregates, inorganic and organic. By increasing the independent movements of the components, their cohesion in an aggregate is diminished; by lessening their independent movements, their cohesion in the aggregate is increased. If some of the animals in a herd start off at a gallop, while others remain still, or walk, the herd is dispersed. If some of the birds in a flock fly away, while others remain feeding, the flock is disintegrated. If a herd or a flock is to remain a herd or a flock, and is not to be dissipated into incoherent individuals, each of its members must do as the rest do. All must move in the same direction, at the same rate, at the same time. They must all start together, and all settle together. Each must make his conduct conform to the general standard of the whole. On no other terms can the community continue to exist. What is true of the simple movements of the components of these simple communities is true of the more complex conduct of the components of more highly organised communities. All must submit to the same authority; all must follow the same customs; all must agree in the same religion. In the highest development of communities, when the concept of duty is formed and becomes binding on the members, it takes the place of these more primitive bonds, which may then be relaxed without allowing the community to fall to pieces; but until the instinct of duty attains a high development, these binding forces cannot be dispensed with.

But this is not all the surrender of individual freedom of action that is demanded by the social state. Not only must each individual conform to the common standard, but each individual must so limit his own activities that they do not interfere with those of his neighbours. If we watch the flight of a flock of fieldfares or pigeons, we are struck with an appearance of discipline. The birds fly together, or there would be no flock. They start, they rise, they wheel, they turn, they alight simultaneously and close together. Yet, close as they fly, they always leave room on all sides, so that no bird interferes with the flight of its neighbour. If one were to direct its course across the line of flight, or to approach its neighbours too nearly, there would be a collision, perhaps several collisions. Some would be injured and fall to the ground, the order of flight would be deranged, and injury would be done to the commonweal. Each, therefore, must so limit its movements as not to interfere with the rest. The solitary bee makes its cell in globular or cylindrical form. The social bee, crowded on all sides by its neighbours, is debarred from complete freedom of action. The cylindrical cell is modified by the proximity of its fellows into the form of a prism. Each, as soon as it reaches the boundary of its neighbour's activity, refrains from encroaching thereupon, and since neither can project its cell-wall into the realm of the other, they combine to form a flat partition between them. It is apparent, therefore, that every influence which tends to produce uniformity of action in the members of a community helps to bind the community together; and every influence which induces the members of a community to limit the freedom of their individual action, and to submit to the limitation of their own activities, so as not to encroach upon the corresponding sphere of action of their fellows, is a preservative influence, and helps to preserve the integrity of the community.

The enormous influence of *Custom* as a factor in the preservation of communities now becomes apparent. Custom ensures uniformity of conduct. As far as all act in conformity with custom, all will act alike. Custom discountenances and checks individual initiative. It prevents each member of the community from forging in front or lagging behind, or from diverging from the direction, and so crossing the paths of his comrades and producing collisions. It keeps every member of the community duly spaced from his fellows. Break up custom, and, unless some equally powerful substitute is provided, the community will fall to pieces. Some communities, as, for instance, the castes of the East, are held together by custom alone. If the custom were broken, the caste would be utterly destroyed; and yet, so strong is custom, that the castes remain in the most rigid sameness, and in complete integrity, generation after generation. It is the dim and blind perception of the immense power and importance to the community of the binding force of custom that has bred in all successful communities such a tenacious adhesion to custom, and such an abhorrence of innovation. Innovators, in whatever department of life, have always been denounced, abhorred, persecuted, and slain. It matters little whether the innovation is in matters important, or in matters unimportant; whether it is the abolition of rotten boroughs, or the substitution of knickerbockers for skirts; the instinct of the community is outraged by the proposal for change, and the innovator becomes the object of scorn, detestation, and reprobation.

From a biological point of view, that is to say, as a factor in the struggle of communities for life, the influence of *Religion* is in some respects similar to that of custom, but the influence is wider and deeper. Religion is one of the sanctions—perhaps the main sanction—of custom, and the connexions between religion and custom are very intimate. Religion frowns upon innovation with a severity at least equal to that of custom, and has never been slow to lend her aid in discountenancing and exterminating innovators; but the peculiar aid that religion has given to the maintenance of society has been in the inculcation and enforcement of self-sacrifice. We have seen that self-sacrifice, self-restraint, the surrender of some part of individual freedom of action, is a necessary condition of the social state. A society in which each individual followed his own inclinations, without regard to the welfare of his fellows, would cease to exist—would be disintegrated into its individual components. Whatever else is inculcated by religion—and among different religions there is almost infinite variety of tenet, dogma, exhortation, prohibition, observance, and practice,—all religions agree in certain respects. Every religion offers a theory of the cosmos; every religion inculcates a belief in the supernatural; and, what concerns us here is that every religion inculcates a rule of conduct which, in the first place, is enforced by supernatural sanction, and in the second, demands some self-sacrifice on the part of its votaries. All religions exhort to certain practices and forbid others, and the institution of *tapu* shews how effectually even a primitive religion can enforce its prohibitions. The influence of religion, therefore, by its power of inculcating and enforcing self-restraint, is a very powerful factor in the conservation of societies. It is one of the strongest of the controlling powers that maintain, in social aggregation, individuals whose turbulent, restive, self-seeking activities are continually urging them to independent action, which, if unrestrained, would break up the community and resolve it into its separate components. The blind recognition of the importance of religion as a preservative influence has led to the formation of a very powerful religious instinct. Every community has safeguarded its religion with the most sedulous care, has visited with terrible punishments those who call its religion in question, and regards with the deepest reprobation every act which appears subversive of religion.

The reprobation of the community falls, therefore, with great intensity upon the three classes of acts above enumerated—attacks upon the community itself, especially upon its ruling power; attacks upon custom; and attacks upon religion; but none of these is regarded as a vice, and, though the first is punishable as a crime, it stands upon a footing very different from ordinary crimes, and is recognised to be quite different in nature. For the true nature of vice and crime we must look in a different direction, and we shall find that, though they are reprobated, they are reprobated for reasons different from that for which these three forms of treason are reprobated.

Wrong-doing.—The reprobated acts which have been dealt with bear upon the community as a whole, and tend to its disintegration. The instinctive discernment that such acts are destructive of the community, and therefore of the stirp, arouses the antagonism of every individual member of the community, and the community as a whole reacts against the destructive agent. But the stirp is menaced, not only by acts directed against the community as a whole, but by acts directed against individuals. Any act that diminishes the power, ability, and competence of any individual to survive in the struggle for life—any act that diminishes his life-worthiness—arouses the antagonism of that individual, and prompts him to a defence which is usually retaliatory in character. Such aggressions against individuals are, in advanced communities, made the concern of the community as a whole, which regards them with reprobation, and takes upon itself the function of punishing them. It is often supposed that this interference of the community in the affairs of its individual members is due to the perception that aggressions by one of its members upon another is disintegrative of the community itself, and that if they became the rule, the community could no longer hold together; and this no doubt is ultimately a reason for the corporate retaliation on the offender. But originally the retaliation seems to be partly a consequence of sympathy with the injured person, chiefly a feeling on the part of the bystanders that "hodie tibi, cras mihi." In primitive societies, at any rate those of Aryan race, the unit is not the individual, but the family. When an individual is killed or damaged, the family suffers injury, and pursues its remedy against the offender and his family. Hence results a state of internecine warfare, manifestly perilous to the community to which both families belong; for both are withdrawn, by their mutual strife, from attacking or defending on behalf of the larger group to which they both belong. To abate this danger, in very early times a tariff was fixed, by which the aggressor might compensate the family of the injured man, and, if compensation according to scale were afforded, the feud must cease. The whole power of the community would then be set in motion to see that the treaty was observed. Any infraction of it rendered the infractor an outlaw. He was driven forth from the community, and ceased to belong to it. Any member of the community might thereafter aggress against him without incurring any penalty save that which he could himself inflict. In this we seem to find the earliest intervention of the state in private quarrels. If no compensation was afforded, the feud went on, and the slayer was left to be slain by the family that he had injured. As the community grew in numbers, and became more compact in organisation, the ruling power—the tribal chief or king—took a larger and larger part in the settlement of private quarrels, and at length assumed the function of keeping the peace, that is to say, first of punishing, then of discovering, and at last of preventing crime. The peace of the community became "the king's peace;" and every infraction of it became an act of aggression, not only against the individual attacked, but against the king, the representative of the community as a whole; and thus was brought into the same category as those other transgressions, already considered, against which there was already so strong an instinctive repulsion. Thus, every member of the community came to reprobate acts of private wrong-doing; and this reprobation was partly from the perception that such acts were against the king's peace, and noxious to the community at large; partly from the growth of sympathy, which led onlookers to feel the injury done to the injured person; and partly from the inference, already alluded to of "hodie tibi, cras mihi," and resentment against the feeling of insecurity thus engendered. Thus it was that the community at large came at length to look with reprobation upon those acts of private aggression upon its individual members that were, in an early stage of society, regarded with indifference.

How, then, are these wrongs characterised that are regarded with reprobation by the community, and how are they distinguished from vices? Wrong-doing is inflicting harm upon others. No doubt it is possible to harm other people without doing wrong, but wrong cannot be done without the infliction of harm on others. A man who lives in complete solitude—an Alexander Selkirk—cannot commit a crime, cannot be guilty of wrong-doing, for he has no companion to whom the wrong can be done. We should, indeed, regard it as wrong for him to beat his dog unmercifully, but "exceptio probat regulam," we should look upon such an act as wrong for the very reason that his dog is his companion, and the sympathies of civilised man are so extended as to include dogs as, in a sort, members of their community. We should not look on it as wrong for Selkirk to injure an alligator and leave it to die; and the reason we should not look on such an act as wrong is that the alligator, unlike the dog, is not regarded as included within the social pale. It is an outsider—an enemy, potential if not actual; and therefore it is that the injury which it is wrong to inflict on the dog, it is not wrong to inflict on the alligator. The first element or ingredient in wrong-doing is therefore the infliction of harm on a being that we regard with some sympathy—as a member of our community, or of that greater community which our continually extending sympathies at length include, a community which now to us includes all the human race except those with whom we are actually at war, and even these, when they are wounded and harmless; which includes also a considerable proportion of the lower animals.

But not all infliction of harm is wrong-doing. The judge may sentence a prisoner to death, and the hangman may perform his office, without doing wrong. The parent may chastise his child; the surgeon may mutilate his patient, the magistrate may fine the defendant before him; and in each case no wrong is done. The element which deprives these harmful acts of their wrongness is evident. It is that the harm was not inflicted for the gratification of the actor, but for some other purpose. Wrong is done when, and only when, gratification is sought by intentionally inflicting harm on others. The formula is a very wide one. It includes injury by direct violence, by neglect of precaution, by failure to furnish necessaries or to adopt obvious safeguards, and by other means. It includes injury to property, either by forcible robbery, by theft, by fraud, and by damage, whether wilful or arising from neglect or indifference. It includes injury to the feelings, either by acts or words that give pain, or by the omission of offices that are due or expected. It extends from murder to discourtesy. In any case, wrong is done when one person seeks gratification for himself by means of injury to another. The root of wrong-doing is defective sympathy. It is in the inability to feel the sympathetic pain which the distress of others should produce. The antidotes and preventives to wrong-doing are first of all the presence of this sympathetic quality, which makes the infliction of pain in itself painful; second, the repulsion to wrong-doing that is instilled and cultivated by training, especially in youth; third, the awe of the religious sanction; fourth, the dread of reprobation; fifth, the dread of retribution from the injured party; and sixth, the dread of punishment by the community at large.

Besides treasons, which may be comprehensively understood to include attempts to damage the community itself, and especially its governing power, to lessen the reverence to religion, and to violate custom; besides wrong-doing, as above defined, there is a third class of acts that are regarded with reprobation. These are acts which are not aggressions against either the community at large or against individual members of it, but yet are acts of inordinate self-indulgence. The spirit of self-sacrifice is so essential and integral a part of the mental equipment of every member of a society, social life is so intimately dependent upon the existence, prevalence, and intensity of this spirit, that any glaring instance of the want of it arouses an instinctive repugnance; and hence it is that unbridled self-indulgence is always looked upon by the community with reprobation, even though the self-indulgence is not gained by the infliction of harm upon others, and is therefore not a wrong; and is not gained by a direct attack upon the stability of the community, and is therefore not a treason. Self-indulgence, whose evil influence is thus restricted, constitutes Vice.

The differentia between wrong-doing and vice is, therefore, that the former is gratification sought by injuring others, whilst the latter is gratification not so gained, but yet felt to be excessive. I formerly thought, and so expressed myself in the previous edition of this work, that a necessary mark of vice was immediate self-indulgence at the cost of a greater benefit in the future; and I still hold that this is a usual, if not an invariable, quality of vice; but I do not now think that the recognition of this detrimental effect of vice is a necessary part of its concept, or enters of necessity into the causation of the instinctive abhorrence or reprobation that we feel towards it. If we see a man bolting his food with wolfish voracity, his whole attention absorbed in his task, his face flushed and the veins in his forehead swollen with passion, his elbows squared, and uncouth noises issuing from his stuffed mouth, we are disgusted; we regard him with reprobation; we consider him a conspicuous instance of the vice of gluttony; but in so condemning him, we do not take into account the gout that he may be storing up for himself in the future. We call him vicious because of his present lack of self-restraint, not because this lack of self-restraint may entail upon him future disaster. Self-restraint is so important for the commonweal, that any display of it excites our commendation, and when it merges into self-sacrifice, our admiration; and on the contrary, any conspicuous lack of self-restraint arouses reprobation in the onlooker, because it is an anti-social quality; because it exhibits the absence of that quality on which all society is based. It is true that vicious self-indulgence often does entail future damage to the vicious person, but such future damage does not necessarily follow. If a healthy man, and especially a young man, whose occupation is not nocturnal, habitually lies in bed till 10 or 11 a.m., we regard him with contempt and reprobation; and yet, if his living is secured to him, the habit need not expose him to any ulterior ill-consequences.

Vices include all forms of self-indulgence which the common voice declares to be excessive; and depending, as they do, on the view that is taken of self-indulgence by the community, they will vary from country to country and from age to age, according to the opinion which is prevalent. In the time of the Commonwealth, to witness a play was vicious. In the time of Charles II., gambling, drunkenness, and profligacy were not vicious. In Scotland it is a vice to play cards on Sunday, in England it is not. The antidotes and preventives of vice are those influences that make for self-restraint and against self-indulgence, and the chief of these are, first, innate disposition; second, training and inculcation in youth; and third, the influence of religion.

Whilst the distinction in nature between wrong-doing and vice is that which has been laid down, the practical difference is that the one evokes a retaliation of the community upon the wrong-doer, whereas the other does not. Wrong-doing is punished by law, whereas the vicious man, as such, is not awarded any punishment, but is left to suffer the natural ill-consequences that his vice so frequently entails upon him. It may happen, however, that, by indulgence in vice, a man, without actually seeking his gratification by the injury of others, yet indulges in gratification with reckless disregard of the injury that he may thereby indirectly inflict upon others, and, in such a case, the community will not content itself with a mere passive attitude of reprobation, but will actively interfere to punish. Thus, a man may get as drunk as he pleases, without laying himself open to punishment for his vice; but if he, by his drunkenness, becomes a nuisance or a danger to others, he becomes punishable. So, too, is he punishable if, by his drunkenness, he makes himself incapable of performing his obligations to the community, for example, of supporting his wife and family. The man who is sexually vicious is not punished by law, unless it is proved that by his vice he has injured others, and the matter is always complicated by the fact that the person who is usually the most injured has been a consenting party, and is thus deprived of complaint. Where there has been no consent, the vice becomes at once a wrong. The degree of reprobation with which the community regards the woman who is sexually vicious, is so great as to constitute in itself a severe punishment, although this punishment is outside the law.

To summarise this preliminary examination of the acts that are reprobated, we find that there are three classes:—Treasons, which are attacks upon the bases of society, government, religion, and custom, and are punished with extreme severity; wrongs, which are acts by which gratification is sought by harming others, and are punished with less severity; and vices, which are acts of excessive self-indulgence, are not punished at all, but are regarded with reprobation. The reprobation with which all are regarded is the protest of the social instincts against acts that are felt to be anti-social, and subversive of society. Acts of the first class are manifestly so subversive, and the primary social instinct naturally revolts against that which directly threatens society at large. Wrong-doing injures individuals only, but it not only threatens indirectly the stability of the community, but outrages the feeling of sympathy which is one of the foundations of the social state. Vice furnishes a conspicuous example of the absence of self-restraint, another of the qualities fundamentally necessary to the existence of society, and its display naturally arouses reprobation.

Vice and Insanity.—Insanity is a dissolution; it is a retrogression; it is a traversing of the path of development in the reverse direction. It is a peeling off of those superimposed layers of development which have been laboriously deposited by the process of evolution. Agreeably to the nature and law of dissolution, those qualities which are the latest addition to the fabric of development are the first to be lost in the process of general decay, and those which were earliest acquired are the longest preserved. Of all the qualities which go to make up the highest type of humanity, that of self-restraint, of self-control, the power of foregoing immediate gratification, as it is one of the highest, so it is one of the latest to attain a high degree of development. It is more developed in man than in the lower animals. It is more developed in civilised than in savage man. It is more developed in the highly than in the lowly civilised. Indeed, the degree in which this quality is developed is often taken as a measure of the height of civilisation attained by a community, of the height of moral development attained by the individual. So far has this quality been adopted as a standard of morality, that, as in so many other cases, that which was originally cultivated, and is still of value only as a means to a further end, is cultivated as an end in itself. Self-restraint, which is of no value except as far as it enables us to attain a greater future good by the sacrifice or renunciation of a present lesser good, has been apotheosised, so that, in many places and by many people, it has been, and is, regarded as in itself desirable and admirable, without regard to any benefit obtainable in compensation for that which is foregone; and this we see in the practice of rigid asceticism, and the unbounded honour which is paid to ascetics. The ascetic is the antithesis of the vicious. The one displays in excess the quality in which the other is deficient; and by as much as the one is reprobated for deficiency in a quality which is essential to social life, by so much is the other admired for excess in that quality. Being a quality of late acquisition in the history both of the race and of the individual, self-restraint is lost early in that reversal of the process of evolution that is witnessed in insanity; and all insane persons are in some degree vicious, in the sense that they are all lacking in self-restraint. Every form of vice is seen among the insane. Every insane person exhibits some form of vice. The most flagrant and shocking extremes of vice are not seen except in the insane; and even the most moral and refined among the insane exhibit that lack of self-restraint which deprives them of those minor benevolences that we recognise as courtesy, obligingness, and good breeding. They lack politeness. They are neglectful of those little offices which oil the wheels of social intercourse. They do not yield any deference to age or sex. They do not hand a chair to a lady; they do not hand things at table. They do not take their fair share of the little labours of games. Others may collect the balls for them at tennis; may spot the red at billiards; may shuffle the cards and mark the points at whist; but when their turn comes, they do not reciprocate these little offices.

Whilst all insane persons are vicious, in the sense that they are unduly lacking in self-restraint, and whilst, therefore, much vice is due to insanity, and is a manifestation of insanity; yet, undoubtedly many sane persons are vicious, and it is often a matter of the greatest importance to determine, in any given case, whether the vicious conduct that a person exhibits is simple vice or is insane vice. This is a matter that cannot always be determined. It is impossible, without a previous and fairly full knowledge of his character, to say whether the vice that an insane person exhibits is a part and a manifestation of his insanity, or whether it is the product of his original nature; but there is a degree of vice which is never exhibited except in insanity, and there is a degree of vice so enormous that of itself it constitutes insanity. The man who refuses to work to-day, with the knowledge that his refusal will put him on short commons next week, is viciously idle. The man who wets his bed rather than take the trouble to get out and make water, is insanely idle. The man who gets drunk on Saturday night, at the known cost of hot coppers on Sunday morning, is a vicious drunkard; the medical man who, with the clear knowledge that he is on the brink of delirium tremens, is drinking himself to death, and ruining his wife and family, still continues to drink, is an insane drunkard. The woman who goes on the streets is viciously profligate. The woman who assaults indecently every man she sees, whatever the occasion and however many the witnesses, is insanely profligate. The man who, in the course of years, muddles away his fortune in horse-racing, gambling, loose women and extravagance, is viciously prodigal; the man who spends in a few weeks ten times the amount of his capital on similar objects is insanely prodigal. Between these extremes there is every possible gradation; and it is evident that it is possible to construct a graduated series of instances, beginning with moral, virtuous, and praiseworthy conduct at one end, and passing through every degree of simple vice, until it culminates in vice so outrageous that it is manifestly insane. In a continuous series no sharp division is possible, and hence it is not possible to draw any determinate boundary and to say here simple vice ends and insane vice begins—all above this line are examples of simple vice, all below it are examples of vice that is insane. All that we can say is that enormity in vice is itself insanity; and the degree of enormity is to be measured by the standard already set up—*the gravity of the difference* between the pleasure that is gained and the benefit that is forfeited by immediate indulgence; that is to say, we have to compare the amount of pleasure enjoyed with the magnitude, the certainty, and the proximity of the evil that is incurred. It is manifest that there will be instances which give rise to legitimate doubt. It is clearly an act of insanity to accept £100 to-day for the reversion to £300 which will certainly be paid to-morrow; and it is clearly prudent to accept to-day £100 for a reversion to £300, which will certainly not accrue for thirty years, and may never fall in at all. But whether to accept £100 down, or a certain £200 in twenty years time, is a matter of indifference as far as the actual value is concerned, and depends upon the ability to employ the money profitably now, and on many other considerations.

Deficiency of self-restraint, or inability to forego an immediate pleasure, is the mark of vice; and vice is that degree of lack of self-restraint which brings upon him who exhibits it the reprobation of those who witness it. In any given instance a vicious act may be due to one of three causes: it may be that the ultimate ill-result of the present indulgence is not foreseen by the actor; it may be that the actor is generally deficient in self-control; it may be that the actor is temporarily deficient in self-control. In the first case, in which the ultimate ill-result of present indulgence cannot be foreseen by the actor, the vice of the act is minimised, but it is not necessarily removed. Self-restraint is a quality so valuable, so essential to the constitution and integrity of a community, that any conspicuous exhibition of self-indulgence is abhorrent to the witness of it, quite apart from any consideration of resulting ill-effects. A greedy feeder is disgusting and repulsive, quite irrespective of any forecast we may make of the resulting indigestion of the surfeit. A drunken man is disgusting, quite apart from our prediction that he will suffer next day from his indulgence. But if we know that the drunkard is a savage who has never before tasted spirit, and is utterly ignorant of its ill-effect, the knowledge mitigates very materially the reprobation with which we view his conduct; and if the glutton is an imbecile who cannot foresee the nausea and vomiting that he is incurring by his gluttony, we regard him as less blameworthy, even though our disgust is not diminished. Here we see one of the modes in which vice may be connected with, influenced by, and in some measure due to insanity; but for the purpose of the argument insanity is not necessary to the exoneration from blame of the vicious person for the display of his vice. It is the ignorance that exonerates, and the exoneration is the same whether the ignorance proceeds from insanity or from mere inexperience.

Or the vice may be due to a general deficiency in self-control, and this deficiency may be innate, or may occur subsequently to birth, either by the invasion of insanity or otherwise. General deficiency of self-control accompanies the general deficiency of other mental qualities in idiocy, imbecility, and lesser degrees of feebleness of mind; but there is also a general deficiency of self-control that stands alone, or, at any rate, may be accompanied by a high development of other mental faculties. There is a large class of persons who are often of acute and nimble intelligence, who are in general ability equal to or even above the average, who are of an active, bustling disposition, who are always busy, but are utterly devoid of industry. For by industry we mean steady persistence in a continuous employment, in spite of monotony and distastefulness; an employment that is followed, at the cost of present gratification, for the sake of the future benefit to be derived from it. Of such self-sacrifice these persons are incapable. They are always busy, but their activity is a recreative activity, in the sense that it is one which is congenial to them, and from which they derive immediate gratification. Upon no other terms will they exert themselves. As soon as they tire of what they are doing; as soon as their occupation ceases to be in itself grateful and attractive, it is relinquished for something else, which in its turn is abandoned as soon as it becomes tedious. Such people form a well-characterised class of human beings, and their characteristics are as follows:—They are clever; they readily acquire accomplishments that do not need any great application for their mastery; and agreeably to the recreative character of their occupations, their natures are particularly well developed on the artistic side. They draw, they paint, they sing, they play, they write verses, they make various pretty things with easy dexterity. Their lack of industry prevents them from ever attaining mastery of the technique of any art; they exhibit plenty of artistic taste and feeling, but they are always amateurs. Their productions, relatively good though they may be for amateurs, are never really good. Before they can be commended, allowance must always be made for this and for that. In games and sports, to which they devote a disproportionate amount of their time, they are always proficient, but never excellent. With the vice of busy idleness they display other vices. The same inability to forgo immediate enjoyment, at whatever cost, exhibits itself in other acts. They are nearly always spendthrifts; they are usually drunkards; they are often sexually dissolute. But, next to their lack of industry, their most conspicuous quality is their incurable mendacity. If in other arts they never rise above the status of amateurs, as liars they take professional rank. Their readiness, their resource, their promptitude, the elaborate circumstantiality of their lies are astonishing. The copiousness and unimpeachable efficiency of their excuses for failing to do what they have undertaken to do would convince any one who had no experience of their capability in this direction. Withal they are excellent company, pleasant companions, good-natured, easy-going and urbane. They have but one peculiarity which seems to indicate actual disorder of mind. Their self-conceit is not only inordinate, but remains undiminished in spite of their repeated failures in the most important affairs of life. The normal effect of failure, especially in important matters, is to depress; it produces dejection, diffidence, lack of self-confidence, of self-reliance. But the people here described are continually failing; their lives are one long record of failure; they are turned out of employment after employment. They see themselves fall immeasurably behind those who are admittedly inferior to themselves in cleverness. They are always in debt, and always sinking deeper into debt. The means that they possess they cannot administer with ordinary judgment and prudence. They take no thought for the morrow. They will starve on an income on which an ordinary man could live in comfort. And yet they are not only cheery and content, but their confidence in their own powers, in their business aptitude and general superiority to other people, remains undiminished. Such characters are common enough, and there are few people who do not number some among their acquaintances. The lack of self-restraint is plainly an inborn character, for it may shew itself in but one member of a family brought up in precisely the same circumstances and on precisely the same rules as other members of the same family who do not shew any such peculiarity. In this respect the innately vicious person is in precisely the same position as the moral imbecile, to be described later on. He is born with one important mental faculty missing or defective, as another may be born with hare-lip, cleft-palate, spina bifida, or other physical incompleteness.

General defect of self-restraint may, however, be due largely, perhaps entirely, not to congenital defect, but to failure of cultivation. Mental qualities, as well as physical qualities, are developed by exercise, and atrophy from disuse. Neglect to exercise self-restraint, in the presence of an alternative, facilitates a similar neglect on a subsequent occasion; and every such neglect renders more difficult the exercise of self-restraint on every subsequent occasion. At length, by repeated failure to exercise the faculty, its efficacy diminishes to the vanishing point. But even when it has been cultivated to a high pitch, the faculty of self-control may be greatly impaired by an attack of insanity. In most cases of insanity it is in some way impaired, as we have seen, but it is impaired in very different ways. There are forms of insanity in which the spirit of self-sacrifice is exaggerated to a very high degree, so that the subject of this disorder refuses self-indulgence of every kind—will not eat palatable food, or will not eat at all; will not sleep in a bed; attempts self-injury and even self-destruction. But these are almost always acute cases, and when the insanity is of long standing, the ability to forgo immediate gratification is almost always diminished and vice of some kind is indulged in.

The most important, because the least formally recognised, form of morbid vice is that in which self-restraint is temporarily removed or overpowered by some paroxysmal craving or nervous crisis. The condition is not very uncommon, is very well characterised, and is clearly morbid. A man of normally clean, sober, and chaste life is attacked from time to time, at intervals usually of a few months, with an overpowering desire to kick over the traces. In some cases the desire is merely for drink, in others it is for irregular sexual indulgence. In others again, it is for neither of these modes of dissipation, but merely for a life free from the conventions and restraints of civilisation. Whatever the particular craving may be, it comes on very rapidly, and is so powerful as to be in most cases irresistible. The person who experiences it plunges into wild and gross sensuality. He usually recognises the loss of reputation that he will suffer if his vagaries become known, and he leaves his home and resorts to some haunt—usually the same, time after time—in which his identity is unknown, and here he gives way to wild and often frantic dissipation. He drinks to enormous excess, often in solitude, and for the mere sake of drinking; or he lives in a brothel and indulges in unbounded excesses; or perhaps he wanders about the country as a tramp, dirty, unkempt, the associate of gipsies and thieves. The paroxysm is short-lived. After a few days, a week, rarely more than a fortnight, his excesses become abhorrent to him. He regards them with the same loathing with which they are regarded by all decent people. He abandons his squalid surroundings and returns to decency and propriety, and lives an exemplary life until, after an interval of a few months, the fit seizes him again. When they are closely examined, such people often deny, and there is no reason to doubt the denial, that they have, when they break out, any pronounced craving for drink or for sexual indulgence. What they experience is a horrible, ill-defined, voluminous, indescribable, and unbearable sense of uneasiness; and if they drink to great excess, as they usually do, they drink from no craving for drink, but because in drunkenness they find relief from the intolerable misery that they suffer. In some cases the sufferer from these paroxysms has the fortitude, with or without assistance, to resist his desire, and refuse the relief that alcohol gives him. He remains sober throughout the crisis; but it is evident to the observer that he suffers intensely, and at the end of a few days, when the attack passes off, he is left prostrate and exhausted, and almost as much a wreck as if he had had recourse to his usual palliative. To call the periodical occurrence of this intense uneasiness a nerve-storm or nerve-crisis does not help us to elucidate its nature, but it marks the occurrence as morbid—as bringing the conduct into the domain of disease rather than of morals. What the nature may be of the bodily process that underlies the change, we can scarcely even conjecture. It presents no close similarity with any other known manifestation of disease. It resembles epilepsy in being an affection of the nervous system, and in its periodical occurrence; but these similarities are too general and too vague to allow us to infer any community of nature. The onset of the paroxysm has not the suddenness of epilepsy, and the duration is very far greater. If I allowed myself to speculate on the nature of the paroxysms, I should rather suppose that they were due to the periodical discharge into the blood of some chemical substance, perhaps originating in a ductless gland. I have elsewhere ("Presidential Address to the Medico-Psychological Association, 1908") given reasons for supposing that the supply of chemical substances in the blood to the nervous centres is the physical basis of Desire, and the occurrences in question seem more akin in nature to the periodical appearance of certain desires than to any other mental change. In certain cases the paroxysmal outbreaks occur at longer and longer intervals and so die out; in others, which are more numerous, the attacks become more frequent and last longer, at the same time, however, becoming milder, and the morbid condition is more settled and occupies a larger part of life. It is not difficult to afford relief and help the patient over a paroxysm if he will consent to apply for aid when he feels the first warnings of the attack. Sedatives are, in such a case, of the greatest service; bromides, bromural, and apomorphine being the most useful. The paroxysm is usually of such brief duration, at any rate in the early years of its occurrence, that there is little fear of establishing a habit; at the same time, there is always a danger that the patient may substitute some new drug for the alcohol to which he is accustomed, and it is the best practice never to let him know what he is taking, never to give him a prescription, or allow him to administer the drug to himself. Cases such as have been described are most frequent in the male sex, but they are by no means confined to the male sex. Not only are there women who are paroxysmal drunkards, but there are women of good social position, and customarily virtuous habits, who break out at stated intervals and go upon the streets for a few days or weeks, or, without yielding to the desire, yet suffer acutely from it, and have periodical and mysterious illnesses which are really paroxysms of this nature. Such cases as these can scarcely be compared with the ordinary week-end drunkard, in whom the vice is indeed periodical, but the periodicity is plainly determined not by internal crises but by external circumstances.

Wrong-doing and Insanity.—Crime is a technical legal term, meaning a certain limited class of serious offences. Wrong also, under its legal title of tort, is a term already appropriated and strictly limited. In order to deal with the whole of trespasses against others, I use, therefore, the term wrong-doing, which I can define in any sense I please. As already explained, I define it as the wilful infliction of intentional harm for the gratification of the actor. To make an act wrong, it must also be unprovoked, or insufficiently provoked, and the actor must know and appreciate, not only the "nature and quality" of his act, but also the circumstances in which the act is done. The act must be *wilful.* It must be willed. It must carry with it the assent of the actor and the dominance of his will over his bodily movements. A movement which inflicts harm by accident is not wrong. Harm inflicted during sleep is not wrong-doing. But even in these cases the act may become wrong if the accident could and ought to have been foreseen, if the circumstances in which sleep was courted were such that sleep was likely to be harmful. The law provides punishment, criminal or civil, for many cases in which harm is done by preventible accident; and not only is the sentinel or the watch at sea punished for sleeping at his post, but in Germany a woman is punished for overlying her child. Ordinarily, however, acts that are unwilled carry no responsibility, and responsibility attaches most closely and most unhesitatingly to those acts in which will is most manifestly concerned. For the degree in which acts are governed and prompted by will varies very widely indeed. Such acts as throwing the arms forward when falling forward, as throwing the arms up when falling backwards, as starting at a sudden noise, as flinching from a sudden unexpected assault, are not voluntary, depend on no exertion of the will, and cannot be prevented by any voluntary effort. At the opposite end of the scale are instances of conduct wherein an end is pursued by a long series of acts, each leading up to the next, and each requiring a direct exertion of will, as when a sale or a purchase is effected through advertisement, through negotiations with agents, correspondence, visits, inspections, legal settlements, and so forth. Between these extremes there are all possible intermediary degrees; and it is evident that the longer and more elaborate the chain of acts leading up to a certain end, the more complete is the responsibility for the attainment of that end, for the more numerous are the occasions on which the will interposes for the purpose. If we compare two cases of homicide, in one of which death is caused by a single blow of the fist instantly following a gross insult or an assault, and the other by the slow action of poison secretly obtained and cautiously administered in many doses over a long period, it is manifest that the latter is much more wrong, and it is more wrong because of the more frequent and more deliberate exercise of the will in bringing it about. Impulsive crimes are never so reprobated, nor ever so severely punished as deliberate crimes, for the more impulsive an act, the less do we regard it as wilful—as the product of volition.

By an impulsive act is to be understood an act which is unpremeditated, which is undertaken on the spur of the moment, and without any, or with the minimum of, balancing of advantage and disadvantage to be obtained from the act. Impulsive acts are commonly the acts of those who have little power of self-restraint, and for this reason as well as for their usually unprofitable character, they are regarded with reprobation. The more impulsive the act, the less premeditation and consideration of its probable results with which it is undertaken, the more likely, obviously, is it that these results will be disadvantageous; and acts of extreme impulsiveness are exhibited by the insane only. In insanity such extreme impulsiveness is not infrequent. An insane person will suddenly start up from a state of quiescence, and even of lethargy, and commit a violent and unprovoked assault or act of destructiveness without warning, and apparently without premeditation.

It has not infrequently been pleaded in defence of a person charged with crime, that the crime was committed under the stress of an irresistible impulse, and this defence has been urged in cases in which the act was not impulsive at all, but was deliberate, and manifestly premeditated. Such a defence must be extremely difficult to establish. Even if it is proved that the act was impulsive, the fact that it was irresistible cannot be known except to the actor. All that witnesses can observe is that it was not in fact resisted. They can never know if all the reserves of the actor's self-control were called up to resist it.

Every act that appears to be voluntary must be presumed to be due to an effort of will; but this presumption may be rebutted, for every such act is not of necessity voluntary. The mandates of the will may be interfered with in their descent from the brain to the muscles, and the will itself is subject to certain obscure disorders which seem to diminish or remove the responsibility of persons so affected for their acts. Movements may be effected without any exertion at all of the will, as in convulsion and spasm, but as in no case do such movements rise to the status of acts, they need not be considered here. But, in chorea and in tabes, movements initiated by voluntary effort may be so modified and interfered with during the passage of the efferent current from the brain to the muscles, as to produce acts that are unwilled. A choreic person may scald another by upsetting boiling water over him, or a tabetic may stamp violently upon another person's foot; but since in neither case was the harmful action willed, no responsibility for it will attach to the actor. The choreic person willed to move the saucepan, but not to upset it; and the tabetic willed to put his foot down, but not on the other person's foot, nor to stamp violently.

The will, however, may be interfered with at its source, while the channels from the brain to the muscles are unobstructed and otherwise normal. Disorders of the will are twofold. Antithetic to the tendency to impulse, already considered, is the opposite condition in which the normal period of hesitation is unduly and unreasonably prolonged. Normally, the period of hesitation that precedes an act depends upon four factors—the importance of the act, and the number and complexity and balance of the considerations that determine its advantage or disadvantage. There are cases in which persons hesitate over an unimportant and simple act for a time that would only be justified if the act were important as well as the advantages and disadvantages evenly balanced. Such persons will hesitate, for five or ten minutes or more, whether to put on first the right stocking or the left; whether to cross the road; whether to smoke a cigarette or a cigar. Such a degree of hesitation is clearly morbid, and though it has never, as far as I know, given rise to an act harmful to others, it is quite conceivable that it might do so, and if it did, the actor should be held irresponsible.

In order that an act may be one of wrong-doing, not only must it be willed, but there must be in the mind of the actor an intention of harming some one else. Intention differs from will on the one hand, and from motive on the other, and it is very important to distinguish between these three states of mind. Every wrong act must be wilful. Many criminal acts must include intention as an essential ingredient. It is sometimes said, and more frequently assumed, that the law takes no account of motives; but this is not correct. The law never charges motive as an ingredient in crime, but in the course of trials nothing is more frequent than an attempt to shew that the prisoner had a motive, and what the motive was. In every case in which the perpetrator of a crime is unknown, the search for him proceeds upon the principle *cui bono*? Who would benefit by the crime? In other words, who had an apparent motive for committing the crime? And when the case comes to trial, and the proof is obtained by circumstantial evidence, no small part of the duty of the prosecution consists in shewing that the prisoner had a motive to commit the crime. We have seen that will is the active exertion of the self which immediately precedes, and as far as we can judge, causes an act. Intention is the desire to produce the consequences which immediately result from the act—what are called the natural consequences, that is to say, the immediate consequences, which it is known by experience are likely to follow at once upon the act, and must have been in the mind of the actor at the moment the act was done. The intention of an act is the answer to the question—What consequence of the act was most prominently in the mind of the actor at the moment the act was done? If A fires a pistol in the direction of B, the wilful part of the act was the pulling of the trigger. If A is experimenting upon a new mode of igniting a cartridge, the desire most prominent in his mind at the moment the act is done is the explosion of the cartridge, and this is the intention with which the trigger is pulled. That B happens to be in the line of fire is a circumstance which was not taken into account, and the wounding of B forms no part of A's intention. If, however, the desire most prominent in the mind of A at the moment of pulling the trigger was that B might be struck by the bullet, then the intention of A was to wound B. It is often said that an actor must be presumed to intend the natural consequences of his act, but it is clear, on consideration, that there are many natural consequences of an act that an actor cannot be said to intend, and that he may intend to produce results which are not the natural consequences of his act. The test of intention is not whether the consequence is or is not natural, but whether the actor, at the moment of acting, had or had not in his mind the desire to produce the result. Whatever result he then desired was his intention, and whatever result he did not then desire was not his intention, whether or no it was the natural consequence of his act. If A points a gun at B, and pulls the trigger, desiring, when he does so, the death of B, then A's intention is to kill B. But if the gun is not loaded, the death of B is not the "natural consequence" of the act, in any reasonable sense in which those words can be used; yet the absence of this natural consequence does not operate to remove the intention. If, however, the gun is loaded, and the death of B is the result, there ensue many other natural consequences of the act, which A may or may not have foreseen, but which he cannot be said to have intended. It is a natural consequence, in the circumstances in which the act is done, that the bullet shall pass through B and hit C. It is a natural consequence that D shall faint, and E shall strike A on the head. It is a natural consequence that there shall be a coroner's inquest. It is a natural consequence that the office that has insured B's life shall lose money. But none of these natural consequences entered into the intention of A, and their absence from the intention was not because they were not natural consequences, nor because they could not have been or were not foreseen; but because, at the moment of pulling the trigger, there was no desire in the mind of A to produce them. His mind was concentrated upon the one effect—the death of B. This result alone was in his mind when the act was done. This was then his desire, and this therefore was his sole intention.

Motive resembles intention in being a desire for a certain consequence to flow from an act, but differs from intention in not being present in the mind of the actor at the moment the act is done. The willed act is a means of bringing about the intended result, and thus satisfying the desire that we call intention. The intended result is a means of bringing about some further result, and thus satisfying a further desire that we designate motive. In the order of performance, therefore, the willed act precedes the intended result, and the intended result precedes the satisfaction of motive. But the order of origination is the reverse of this. Some more or less primitive desire exists in the mind as a motive; and, to satisfy this desire, some one course of action is chosen, and forms the intention, which subsequently becomes the basis of a voluntary act; A desires to be revenged on B for some injury, real or fancied. The desire for revenge is A's motive to action. He may revenge himself by injuring B in reputation, or in pocket, or in person; and out of these methods he chooses the last. The desire to injure B is now narrowed down to a desire to injure him in person. Again a choice presents itself. A may injure B by beating him, or stabbing him, or shooting him; and out of these modes he chooses shooting. The desire to shoot B becomes the motive to a series of intentional acts. A gun and ammunition are purchased. The desire to shoot B is the motive of the purchase; the desire to purchase is the intention with which he enters the shop, for at the moment of entering the shop the matter most prominent in his mind is the purchase of the gun. The desire to shoot B is pushed back for a time. It is swamped by other considerations—of price, bore, number of barrels, and so forth. Having procured the gun, he sets out to waylay B, which is now become his intention, the underlying motives of injuring B, of injuring him by shooting, being for the time subordinate—sub-latent, implicit, and overshadowed by the desire to reach the appointed place in time. Arrived at his post, all his preliminary intentions have been fulfilled, his preliminary acts performed, and now the motive to which they were preliminary—the shooting of B—emerges and comes to the front. There is now but one intermediate act interposed between the desire of injuring B and its satisfaction, and this desire accordingly ceases to be the motive to other intended acts, and becomes itself intention. But it may be that A desires to kill B, not from revenge, but in order to rob him, and thereby secure a sum of money to enable A to leave the country and escape the consequences of other misdeeds. In this case the series of acts and of motives does not come to an end with the death of B. When A pulls the trigger he has in mind not merely the death of B, but the desire of rifling his pockets. This desire thus forms his intention, the motive being escape; and as soon as the intention is carried into effect, and the money secured, this motive becomes intention, and he flees to the railway station with the intention of effecting his escape. Thus, as each intention is accomplished, the immediate motive which prompted it becomes an intention, to be superseded in its turn, until the ultimate motive of the whole chain of acts is at last satisfied.

Generally, in all acts of wrong-doing, the intention is to inflict harm on others, the motive is to obtain satisfaction for the wrong-doer, and unless both these conditions are satisfied, the act is not wrong. In the normal, motive and intention are harmonised, so that the end desired in the intention is a step towards the satisfaction of the motive, and the motive prompts an intention whose satisfaction contributes to that of the motive. It may be, of course, that choice is at fault—that among the alternative means that appear practicable for attaining the end desired in the motive a wrong means is selected, so that the act done does not realise the intention, or the intention satisfied carries the actor no nearer the satisfaction of the motive. A may pull the trigger with the intention of shooting B, but the gun may not be loaded, or if it is, A may miss his aim; or it may be that A succeeds in killing B with intent to rob him, only to find that his pockets are empty. In these cases, the attainment of the end—the satisfaction of the motive—is frustrated by external conditions, by want of foresight, want of precaution and so forth; but there is no discrepancy between motive and intention. Granted that the circumstances were, as the actor wrongly, and perhaps on insufficient grounds, believes them to be, the intended result would contribute to the satisfaction of the motive. There are, however, conditions of disease in which intention and motive are not harmonious, but antagonistic; in which it is manifest that the act intended cannot possibly satisfy the motive which prompts it; or in which motive and intention are in direct antagonism, so that the act intended is a violation of motive. The difference between the two cases here put is mainly this—that in the first, the intention may be in harmony with an immediately preceding motive, though it is antagonistic to an ultimate motive, whilst, in the second, the intention is antagonistic to the immediately precedent motive, so that there is an intimate conflict of opposites. In every case of voluntary action we may follow the chain of motives back from the intention until we come to one of a comparatively small number of crude desires, which are primitive, and beyond which we cannot go. They are primary endowments of the human mind, and do not appear to be derivative, although it is possible that they may in fact all be resolvable into one primitive desire—that for the preservation and prevalence of the stirp. If so derivable, they can be derived only by inference, and for practical purposes each of them is an ultimative motive or spur to action. To take the case of the nefarious A, he purchases his gun in order to fire it at B; in order that he may thereby kill B; in order that he may obtain from B's pockets the means of flying beyond the sea; in order that he may escape the hanging that he has rendered himself liable to by some previous crime. Further back than this we cannot penetrate into his motives. We are arrived at a motive—the desire for self-preservation—which is, for practical purposes, primitive; and does not admit, as every other link in the chain we have examined admits, of derivation from a more fundamental, more primitive, motive. We are now at the end of the chain, and are come to the staple. In the same way, we may take any other act whatever, and trace it back step by step, through a larger or shorter series of motives, to some simple fundamental instinctive desire—the desire of self-preservation, the desire of race-preservation, the desire of common-preservation, the desire of preserving others.

Normally, these desires prompt to acts that tend to the satisfaction of the desires; but in disease the place of these acts may be taken by others which are either futile, and utterly incapable of satisfying the desire, or by acts that are directly subversive and contradictory of the desire. An early subsidiary of the desire of self-preservation is the desire for nourishing food; but for this desire may be substituted one for wholly innutritious substances, such as chalk or clay. Or instead of acts preservative of life there may be acts destructive of life, a desire of self-destruction being substituted for the desire for self-preservation. Similarly, the desire of race-preservation exhibits its crudest and most fundamental phase in the desire for sexual intercourse; but for this desire there may be substituted a desire for quasi-sexual proceedings with persons of the same sex, or with young persons not yet virile or nubile; or, instead of normal sexual desire there may be a total repulsion from all sexual proceedings, and even a desire for self-mutilation, which may be expressed in acts that make reproduction impossible. In all these cases, the motive and the intention are in harmony, and the disorder resides, not in any conflict between motive and intention, but in the substitution of an abnormal for a normal motive. The disorder, the change, is in the motive, and not in the intention. But this does not express all the facts. Motive is undoubtedly disordered, and a new and morbid motive becomes dominant and operative, but the old motive is not always destroyed. In many cases we can recognise a conflict between the normal motive and the morbid one, and sometimes one, and sometimes the other, gains preponderance. The normal desire of self-preservation is often thus brought into conflict with the morbid desire of self-destruction, and the conflict may extend over weeks, months, or years, sometimes one and sometimes the other gaining the domination.

Somewhat distinct from morbid desires are morbid aversions, which also can be traced to disorder of primitive instincts. Morbid dreads of things not in themselves dangerous, morbid repulsions from things not in themselves repulsive or even non-existent, are not infrequent among disorders of mind. Such aversions are claustrophobia, the horror of being in an enclosed space; agoraphobia, the horror of being in an open space; mysophobia, the horror of uncleanliness; and so forth. In these cases there is a morbid and irrational horror which is well known by the subject of it to be morbid and irrational, but which yet dominates conduct. The fear of being in an open space or in a closed space, as the case may be, is a derivative of the instinct of self-preservation. This primitive instinct or desire gives rise to many aversions. It directly prompts the aversions to the crude pains of bodily injury, and to all those situations and circumstances which threaten bodily injury. It gives rise to that indescribable feeling of unpleasantness which most people experience on looking down from a great height, even though they may be quite efficiently guarded from falling. There is no danger of falling; there is an insurmountable railing at the edge of the precipice, which is manifestly a completely efficient safeguard, and yet it is impossible to approach the edge voluntarily, and if we are compelled to approach the edge a horrible feeling is experienced which cannot be suppressed or reasoned away. The sufferer from agoraphobia or claustrophobia experiences precisely similar uneasiness in circumstances which are different indeed, but are only superficially different. In all three cases the same feeling of horror and impending destruction is produced by circumstances that are quite well known to the sufferer to be safe and innocuous. The claustrophobe who is shut up in a railway carriage, the agoraphobe who is in an open space, is as thoroughly and completely aware of his safety, and of the irrationality of his terror as the person on the edge of a cliff who is safeguarded by a stout railing from falling over, but none the less is he incapable of utilising his knowledge to suppress his panic. In such cases the sufferer performs intentional acts apparently without motive. The giddy man shrinks away from the edge of the cliff, well though he knows he is in no danger of falling over; the agoraphobe clings to a tree or presses his shoulders hard against a wall, well though he knows there is no danger in crossing the space before him; the claustrophobe straps his legs together to prevent himself from jumping out of the window of his carriage, well though he knows he is in no danger if he sits still. In such cases there appears to be an hiatus between motive and intentions. The act is intended, undoubtedly. The intention of the first is to get away from the edge of the precipice; of the second, to keep near an upright surface; of the third, to compel himself to sit still. And the ultimate motive in all the cases is the same—self-preservation. But between the motive and the intention there is a great gulf fixed, for it is known at the time the intended act is done that it will not contribute to the satisfaction of the motive. If we say that the motive is the prevention or removal of the dreadful feeling that the circumstances inspire, there is no clear distinction between this motive and intention, for this is the very desire that is in the mind of the actor at the moment of acting.

The cases just described, in which an intended act is done without a motive, stand intermediate between the ordinary case in which an intended act is done in pursuance of a motive, and the following case in which the motive and the intention are positively antagonistic. These extremely bizarre and puzzling acts are done under the influence of what is called obsession. A woman of irreproachable character and strong religious feeling applies to her doctor for aid against an impulsion which she loathes and recoils from with horror—to utter obscenities and blasphemies. At frequent intervals in the narration of her trouble, she pauses, to whisper rapidly and almost inaudibly, and when her whisperings are audible they are found to consist of oaths, obscenities, and blasphemies. The utterance of such expressions fills her with horror and dismay, but she declares that she cannot help it, and the truth of her declaration is manifested by the anguish of mind that she evidently suffers, and by the very fact that she implores for relief with agonised persistence. Another woman feels an urgent desire, whenever she has a knife in her hand, to cut her child's throat. So urgent is the desire, and yet so strong is her horror of it and dread that she may satisfy it, that she always takes the precaution of placing her child in the care of a neighbour before she ventures to use a knife. She begs her husband to lock the knives up before he goes out. She seeks medical aid to assist her to overcome her horrible intention. She may even apply to the police to restrain her. She declares, and her conduct bears out her declaration, that she loves her children, and would not hurt them for the world, but yet, when she feels a knife in her hand, she is urged by some internal compulsion to wound them. Commonly she is successful in resisting this desire, but if she did yield to it, there could be no doubt or question about her intention. The desire that would be predominant in her mind at the moment of using the knife upon her child would be that of wounding it. And this desire, this intention, would be in direct antagonism with the love she bears it, and the desire to safeguard it from injury which she exhibits by sending it out of the house when she is about to cut its bread and butter. Her underlying and more permanent desire—her motive—is for the welfare and safety of her child. Her immediate desire—her intention—is to do the child a deadly injury. Motive and intention are, then, in deadly conflict. For this state of mind we have no explanation. We must be content to recognise its existence as a not unquestionable event, and the problem that we have to solve is how far the unfortunate woman would be responsible, that is to say rightly punishable, if she were to yield to her impulse and carry out her intention.

There seems prima facie to be a clear difference in degree of responsibility between acts that are done on the prompting of such perverted instincts as homosexuality and geophagy, and acts done in obedience to the prompting of agoraphobia and claustrophobia. Acts of the first class seem more clearly to be rightly punishable than those of the second. Yet both classes may be regarded as perversions of instinct, since in both the instinct prompts to acts that cannot subserve its purpose, and may even defeat it. In the first cases the motive is the satisfaction of a desire, in the second the satisfaction of an aversion ; but a difference so shadowy and unessential does not appear a sufficient foundation for a difference of responsibility, nor can such a distinction be founded upon a different degree of morbidity in the several cases. Any difference in the responsibility that may be apportioned would seem to rest rather upon this—that in the case of homosexuality, and in less degree in that of geophagy, the perverted instinct is satisfied by a series of acts, each of which necessitates an exertion of choice and a determination of will, whilst the acts that are prompted by agoraphobia and claustrophobia are single, isolated, and usually impulsive acts, not preceded by premeditation.

Acts done under the prompting of obsession stand in a different category. In them there is, *ex hypothesi,* no motive. They are done with intention, it is true, but beyond the desire to produce the single effect present in the mind of the actor at the moment of acting, there is no further desire to be satisfied by the act, but the contrary. The person who is impelled by obsession to utter obscene and blasphemous expressions derives no gratification, satisfies no desire, by the utterance, beyond the completion of the intention. Any motive that there may be behind the intention is in the opposite direction. It is said by Sir Fitz-James Stephen that the law takes no account of motives, but looks to intention only. It has already been shewn that this is not strictly true. In any case in which the perpetrator of a crime—for instance, a murder by poisoning—is unknown, proof of motive is a very important part of the case for the prosecution, and if it is proper in any case to shew that the prisoner had a motive, it is surely proper to shew that he had not. In cases of obsession the absence of motive for the crime, and the presence of a motive against it, are surely important factors in the determination of the degree of responsibility that is to attach to the crime. If a person applies to the police to be restrained from committing a crime to which he is impelled by an obsession, if he seeks medical advice to counteract the obsession, if he enters an asylum and submits to its restrictions from fear that he may be impelled to commit the crime, and if in spite of all these precautions voluntarily taken circumstances occur which render the crime possible, and he commits it, it seems clear that he ought not to be held fully responsible for it. Obsession is by no means an infrequent occurrence. The number of persons who apply to alienists for relief from this malady every year is by no means neglectable. Their distress is very real, and their desire to do the deed is often very strong; and yet I know of no case in this country in which any person has ever been tried for a crime which appeared to be committed under the influence of obsession; and of the many cases that I have seen in which the obsession was towards violence, I have never known one in which the intention was carried out into action. Obsession to the utterance of objectionable words appears, however, to be much less under control, and does often issue in action. Whilst, therefore, there is no case on record in this country in which obsession has been pleaded, it seems likely that sooner or later a crime will be committed by an obsessed person, and if the existence of obsession is proved, it seems right and just that it should go far to remove the responsibility of the obsessed person.

The conditions of responsibility thus far laid down are, therefore, that will, intention, and motive must all concur in the act. The actor must will the act, intend to harm another, and desire to obtain by the act some ulterior gratification for himself, which forms the motive. Certain other aspects of motive need consideration. From time to time crimes are committed for which no motive, or no motive that appears, to the ordinary man, adequate, can be assigned. A husband murders the wife with whom he has always appeared to be on affectionate terms. A man assaults without provocation an unoffending stranger. A woman murders the child to whom she has always appeared devotedly attached. A wealthy woman turns shoplifter. A wealthy man steals the spoons from the table at which he is a guest. And many crimes are committed every year from motives which appear to the onlooker utterly inadequate. The first comment to be made on such cases is that it by no means follows that, because the motive is undiscoverable, therefore no motive existed. Especially between persons who are very closely associated, who live together in the same house, or are associated in the same business, if they are reticent and self-controlled, the most malignant animosity may exist without betraying itself, even to the servants. An unoffending stranger may be attacked and killed by mistake for some enemy whom the murderer desires to destroy. No one but Sir Piercie Shafton understood the motive of the deadly animosity that prompted him to seek the life of Halbert Glendinning. The Newgate Calendar gives many instances of murders committed on very intelligible motives by persons who appeared to be on most affectionate terms with their victims. Wealth does not necessarily confer honesty upon its possessor. Many wealthy persons have obtained their wealth by dishonest means; and it by no means follows that ability to purchase an article destroys the desire to obtain that article without paying for it. Of course, when absence of motive can be positively proved, such absence has a very important bearing upon responsibility; but, although it is by no means as difficult to prove a negative as the old logicians contended, it must be extremely difficult in any specific case to prove a total absence of motive for an act. Yet, where motive can be excluded, it seems that responsibility is abolished, not necessarily because the criminal is insane, but because, sane or insane, he does not fulfil the conditions of responsibility in acting for his own gratification. There are cases in which the act is of such a character as scarcely any motive can be considered adequate to account for. A considerable number of murders are committed with such an excess of brutal and revolting violence as seems to establish a prima facie case of insanity in the murderer, and some of these are committed by persons known to be epileptic. In such cases the victim is not only killed, but mutilated or battered almost out of semblance of humanity, and whereas no conceivable motive would prompt such an act in an ordinary person, the motive assigned for such crimes of excessive violence is often of the most paltry description. In a recent case a woman was murdered, her head and arms cut off, for the sake of the value of her umbrella and some of her clothes.

To render an act of wrong-doing punishable, or to invest the actor with responsibility for it, the motive must be a self-regarding motive. The act must be done for the gratification of the actor. It is important to notice that this condition is true of wrong-doing only. It does not apply to treason in any of its forms. Attacks upon the government, upon religion, and upon custom are often undertaken with no prospect or hope of benefit to the actor. His action is often undertaken with the certainty that it will lead to his destruction, usually with the probability that it will bring down upon him obloquy, reprobation, and other painful results. Such acts are usually undertaken from public spirit, and with a desire of benefiting the community at large, even against the will of its own members. To treasons, therefore, the following considerations do not apply, but to make an act of aggression wrong, it must be done for the gratification of the actor—the motive must be self-regarding. Intentionally to destroy the property of another is wrong if the act is done for the gratification of the actor, either by the satisfaction of his spite or to gain advantage in any other way, as by removing an obstacle to his enjoyment. If I see a man with a goblet to his lips, about to drink its contents, I do him wrong if I dash the goblet out of his hand and break it into fragments. I do wrong, at least, if I do this to annoy him, to prevent him from satisfying his thirst, or to injure him by destroying his property for my own gratification. But if I believe that the cup contains poison, and that he is wilfully about to drink poison, I do him no wrong by dashing the cup to the ground; and the wrongness of the act is removed by removing the self-regarding motive. Similarly, I do wrong to stick a knife into a man from a motive of revenge, but if my motive is to open an abscess, I do no wrong; and yet again, if, behind my motive in opening the abscess, there is the motive of displaying my skill and anatomical knowledge, I do wrong or right according as the self-regarding motive is or is not predominant. If the act is done primarily for the relief of the patient, then it is not wrong, even if I obtain gratification by the display of nerve and skill; but if the act is done primarily for the display of my own qualities, and only secondarily for the welfare of the patient, then the act is clearly wrong, and is made wrong by the substitution of a self-regarding for a non-self-regarding motive.

Even if an act, harmful to others, is purely self-regarding, it may be divested of wrongness if the proportion of harm done is very small in comparison with that of the benefit obtained. It is wrong to steal, but if my child is perishing of cold, I may, without wrong, steal a match to light the fire which will save its life. It is wrong to damage the property of another, but if I see a man hanging, I may cut the rope, even though it does not belong to me.

Leaving now the subject of motive, we find that responsibility for wrong acts depends, to a great extent, upon the presence or absence, the adequacy or inadequacy, of provocation. In all systems of jurisprudence provocation is taken into account in awarding punishment, that is to say, in fixing responsibility. It is prima facie wrong to break a man's arm intentionally; but if the man is murderously assaulting me, I may break his arm in self-defence, and my action will not be wrong. It is permitted to repel an attack by any means that are not more than sufficient to prevent my own injury, but having succeeded in preventing injury to myself, I am not justified in pursuing my adversary and inflicting further injury upon him. In so repelling an attack, knowledge, motive, intention, and will are all such as to satisfy the definition of wrong-doing, and yet the act is taken out of the category of wrongs by the presence of provocation. Such a case as has been instanced presents no difficulty either in law or ethics; but difficult cases may easily be constructed. If a man is following me down the street with foul abuse, am I justified in assaulting him to make him leave off? and if I am not then justified, am I not justified if he pollutes with obscenity the ears of the lady who is walking with me? The fact that the law will interfere and punish my assailant for using "language calculated to provoke a breach of the peace" seems to indicate that the law regards my retaliation as a natural, if not an inevitable, result of the provocation.

Finally, to render an act punishable as wrong, it is necessary not only that all the conditions above dealt with should be satisfied, but that the actor shall know and appreciate, not only the immediate consequences of his act, but also the circumstances in which the act is done. The immediate consequences of an act, as far as they are known to the actor, must be desired by him at the time of acting, and constitute his intention. If they are not known, not anticipated, by the actor, then he has no intention of producing them; and then, if the intention forms part of the offence with which he is charged, he is clearly not guilty. If A discharges a gun at B, fully believing the gun to be unloaded, or loaded with blank cartridge, and if the gun is nevertheless loaded with ball, which hits B, A neither desired nor anticipated this immediate consequence of his act, which was, therefore, not his intention. So far there is little difficulty, and will be little difference of opinion.

But, in order to attach responsibility to a harmful act, it is not enough that the act shall be willed, intended, unmotived, and void of provocation. There must be the further condition that the actor knows and appreciates the circumstances in which the act is done. Want of knowledge of the immediate consequences of the act deprives the act of intention to produce these consequences, but this want of knowledge may, and often does, depend on want of knowledge of the circumstances. The mother who allows her child, peeling after scarlet fever, to play with other children, does wrong if she knows that the child is infective, but does no wrong if she has not this knowledge. The woman who seizes an innocent bystander and gives him in charge, does no wrong if she *bona fide* believes that he has picked her pocket. If I take my neighbour's umbrella instead of my own, I do no wrong if I really thought it was my own. Every mistake excuses the mistaken person for any harm he may do, provided the act he does would not have been wrong if the circumstances had been as he mistakenly believes they were. This principle is the foundation of the answer of the judges to the fourth question put to them by the House of Lords in 1843. "If a person under an insane delusion as to existing facts commits an offence in consequence thereof . . . we think he must be considered in the same situation as to responsibility as if the facts with respect to which the delusions exist were real." That is to say, if the facts he imagines would justify his act if they really existed, then he is excused; but if the state of things that he imagines would not, if it were true, justify his act, then he is not to be excused. This is a logical position, and to the first part of it no exception can be taken; but few are nowadays inclined to admit that a deluded person should never be excused if his delusion is not of an exonerating character. Such a view presumes delusion to have precisely the same effect upon conduct as some mistake; and this presumption is erroneous. If it were correct, the course which a deluded person will take with reference to the subject matter of his delusion could be predicted with as much confidence as the course which a mistaken person will take with respect to the subject of his mistake; but this is never possible. Deluded persons are constantly betrayed by their insanity into acts which have no discernible reference to the delusions they entertain. When a person holds the delusion that the whole city, the whole country, or the whole world belongs to him, it might naturally be expected that he would be apt to act in accordance with this delusion, and appropriate things that do not belong to him under the belief that they do: The delusion is a frequent one, but the logically consequent conduct is not at all frequent. Now and then, indeed, a case occurs, but, commonly, the persons who entertain delusions of this character respect with complete propriety the possessions of other people. The belief that all that they see belongs to them coexists with the knowledge that other people have a sole right to their property.

Impossible as this coexistence appears to a sane person, it is but one instance of the contemporaneous existence of contradictory beliefs which is so frequent in insane persons. Not only does the deluded person not carry his delusions into their logical effect, but he very frequently does, in consequence of his insanity, acts with which the delusion appears to have no connexion. Believing that he is a man of boundless wealth, he cuts his throat. Believing that he is entitled to the throne, he tears up his clothes. Believing that he has a rat in his stomach, he smashes the windows. The influence of delusion upon conduct is far less simple than appears to be assumed in the doctrine that the deluded person is to be treated as if he had made a sane mistake; and if this is true of delusion, still more is it true of insanity in general, of which delusion is but one, and a comparatively unimportant feature.

It is settled law that every one is assumed to be sane until the contrary is proved, and the burden of proving that an offender is insane lies upon him who asserts it; but an offender having been proved to be insane, the burden of proving that his insanity had no connexion with the offence with which he is charged should lie upon the prosecution. I think that this would better serve the ends of justice than the present method, by which the defence must prove not only that the prisoner was insane at the time the offence was committed, but that the insanity was of such a character as to deprive him of the knowledge of the nature, quality, and wrongness of the act. Even, however, if the suggested rule of law were to be substituted for that in existence, it would be far from covering all cases; for many offences are committed by persons whose minds are temporarily disordered, but who are not insane in any but a technical sense, which would probably not be admitted in a court of law.

In the case supposed, of a man who points a gun at another and pulls the trigger in the firm belief that the gun is unloaded, the harmful result of his act arises from his want of knowledge or misappreciation of the circumstances in which he acts. He does not know that the gun is loaded, and acts in the belief that it is not. Similarly, if he takes another man's watch in mistake for his own, his act is not stealing, for he acts in misappreciation of the circumstances. It is true that he acts without intention to steal, and his defence might well rest on this ground, but in this case and in many others intention can be inferred from the state of his knowledge, and it is easier to prove want of knowledge than want of intention. In the great majority of cases in which crime is committed by insane persons, there is a want of knowledge and a misappreciation of the circumstances in which the act is done, which should go far to relieve the actor from the penal consequences of his act. It very often happens that there is no such exonerating quality in the delusions of the criminal, if he entertains delusions at all, as would bring him within the benefit of the answer of the judges; and yet he so misappreciates the circumstances as to shew that he is not capable of judging fairly of the rectitude or turpitude of his act, and though he may in one sense know that his act is wrong, and may know pretty clearly that it is illegal, yet his state of exasperation, from fancied wrongs, may be so great that at the worst he should be regarded as a person acting under extreme provocation, and when to this is added a general obfuscation of intellect, which renders him unable to reason logically or appreciate properly the relation of his victim to himself, it is clear that he ought to be judged on a footing entirely different from that of a sane man. It is especially, though by no means solely, to cases of paranoia that these considerations apply. The man who believes that he is the victim of a plot, by which he is continually being injured in mind, body, and estate, and who is haunted by the notion that the plotters are all around him, though he cannot identify them, may surely receive some consideration if he turns upon one of his neighbours and assaults him, even though he has no reason to, and may not, associate his victim in any way with the plot. We cannot dive into the illogical mental processes of the insane. It is impossible for us to recognise, difficult for us to realise, that what appears to us the wildest conjecture is to them the most convincing reason. But when we see a person holding simultaneously two contradictory beliefs, when we see that he honestly believes that he is enormously wealthy, at the same time that he begs for a few coppers, we cannot but realise that the whole realm of motives must be different in him from what it is in sane people, and we must make very liberal allowances for his conduct. At the same time it is to be remembered that, in the majority of the insane, there is a large area of conduct in which they are sane; and if, in this area of their conduct, they commit offences, they may fairly be judged by the standard of the sane, saving only that it must be a lenient standard, for we can never be quite sure how far the penumbra of the insanity extends.

Much has been written about instinctive criminals, and there is a school of writers, mainly Continental, but with a few American and English members, by whom it is held that criminality is an innate quality, in almost the same degree as blue eyes or albinism; that it is recognisable by physical peculiarities; and that those who are thus distinguished are *ipso facto* irresponsible, in the sense that they ought not to be punished. Of this doctrine it must be said that at present it is entirely wanting in proof, and is not likely to be accepted generally in this country (*vide* p. 1020). Some even of its adherents have been repelled by the extravagances of its more enthusiastic advocates. Whilst, however, the doctrine is destitute of proof, or even of probability, as far as the vast majority of criminals are concerned, there is very plausible reason to believe that a few persons do now and then appear sporadically, in whom the ordinary restraints of morality seem never to be developed. They are born with a congenital inability to become moral, just as others are born with a congenital inability to grow to adult stature, or to develop a complete palate or lip or spinal canal. Such persons may be born of families, every other member of which is normal. Their brothers and sisters may be honest, industrious, moral, self-restrained, and decent law-abiding members of society. But, in this family of normal honest persons, there is one who from infancy has failed to recognise the obligations of morality. He has been a thief, a liar, an impostor, and a fraud since he became able to talk and to use his hands. No severity of punishment deters him; no prospect of disgrace has any effect on him; no feeling of affection for others sways him in the least; he is innately criminal. Sometimes his criminal propensities disappear at, or after, adolescence; sometimes they remain through life; and I have not yet been able to discover any criterion which would enable us to predict in childhood which of these results will follow. It seems, however, that the majority of the cases do recover, for of the many children who have been brought to me as a precaution, in order that when they fall into the hands of the police and become the subjects of a criminal charge, I may be called to bear witness to their morbid character, the proportion of those in whom this course has been followed is insignificantly small.

Those who are said to be born criminals because they are born with sexual desires directed towards their own instead of towards the opposite sex are in my opinion wrongly so-called. They are born with such a constitution that the sexual desire, when it appears, is directed into a morbid channel. Their desire is morbid; but there is no more reason why they should not control this morbid desire than why a person with normal desire should not control that desire. There is no evidence whatever that the desire is morbidly excessive, or that self-control is congenitally weak. Since the illegitimate gratification of the normal desire is regarded as a proper object of punishment, there is no reason why the gratification of the morbid desire should be regarded in any other light. The case is otherwise when the morbid desire is not congenital, but appears at an advanced age, toward the close of a long life, throughout which desire has been normal, and after this normal desire has died down with the advance of age. It sometimes happens that, at this time of life, sexual desire undergoes a rapid and morbid intensification, and receives, at the same time, a morbid direction towards immature children or towards members of the same sex. The morbid character of such cases is twofold, and is, it is clear, so pronounced as to go far to exonerate the criminal from responsibility.

For a third kind of criminal, irresponsibility is often claimed, namely women of good position and ample means who steal from drapers' shops. In a small proportion of these cases the women come of insane stock, and have exhibited signs of nervous disease; but in much the larger proportion there is no such history, and no reason to believe that they are other than thieves whose furtive habit has been started by temptation and opportunity, and confirmed by impunity. There is, however, a class of thieves for whom partial or total irresponsibility may fairly be pleaded. These are the thieves who steal articles of one kind only. One such thief stole fans, and had a large collection of stolen fans of all degrees of value. Another stole carpenter's tools, far beyond what he could use. A third, whenever she was drunk, invariably stole pieces of bacon; and the list could be extended indefinitely. It is important, with reference to these cases, to bear in mind that the cyclical recurrence of the same mental and nervous state is a well-established phenomenon in disease. It is very rare for the epileptic fits of different persons to be identical or indistinguishably alike in character, but every epileptic fit of the same person begins in the same way as every other fit of that person. When the fits of an epileptic are followed by post-epileptic automatism, the automatic act is usually the same after each fit; and, if not the same, is similar—is nearly allied in character. In recurrent insanity the beginning of each attack closely resembles the beginning of every other attack in the same person, and usually the attacks are alike, not only in the beginning, but throughout. The attacks of one patient always begin by his upsetting his bath, the attacks of another by his buying pigeons; the attacks of another by acts of indecency; of another by writing libellous letters; of another by getting drunk; and so forth. It is difficult not to associate these cyclical occurrences with the cyclical thefts of similar articles, especially when the articles are not used by the thief, are not turned into money, but are merely accumulated, or are perhaps given away, or even thrown away.

CHARLES A. MERCIER.

The ordinary bibliography of criminal responsibility is much too voluminous to be referred to, but all that is essential is contained in the admirable summary by Dr. Orange in Hack Tuke's *Dictionary of Psychological Medicine*, in Fitz-James Stephen's *History of the Criminal Law of England*, in the *Report of the Select Committee on the Homicidal Law Amendment Bill*, 1874, and that of the Capital Punishment Commission, 1875. The works of the Continental school of criminologists headed by Lombroso, Garofalo, Féré, etc., may be read, but should not be accepted. Those of Krafft-Ebing and his imitators may be enjoyed by the connoisseur in pornography. My own book on *Criminal Responsibility* treats the subject from the psychological point of view.

C. A. M.

IDIOCY AND IMBECILITY

By G. E. SHUTTLEWORTH, M.D., and FLETCHER BEACH, M.B., F.R.C.P.
Revised by G. E. SHUTTLEWORTH, M.D.

Etiology.—Corresponding with the broad classification of the forms of mental imperfection into those dating from birth and those produced by causes acting on the child afterwards, the principal causes may be grouped as pre-natal and post-natal. The former include hereditary influences, mental and physical; whether traceable through several preceding generations, or personal to the parents themselves. The most notable of the pre-natal causes are a family history of phthisis, of insanity, imbecility, epilepsy, or other neurosis, and a parental history of intemperance, syphilis, ill-health, accident, or nervous shock affecting the mother during gestation. The chief causes coming into action at the time of birth (which might be designated *natal*) are the results of pressure during prolonged parturition or in forceps delivery: premature birth is another cause referable to this group. The post-natal class comprises such causes as eclampsia (convulsions during teething), epilepsy, sunstroke, cranial injury, severe mental shock, inflammation of the brain and its membranes, and exhausting febrile illnesses—such as whooping-cough, scarlatina, measles, and small-pox. Experience teaches that it is rarely scientific to assign one specific cause to a particular case; when it is possible thoroughly to investigate the history, many contributory factors may usually be discovered. The medical man, therefore, must not too readily accept the proffered explanation of parents that the cause was purely accidental, or due exclusively to some illness during childhood; these may, indeed, be regarded in some instances as immediate causes, but the bias to mental breakdown is innate. Children of highly neurotic ancestry are often incapable, by reason of nervous and mental instability, of tiding safely over the crises of development; and such cases have been described by J. Langdon-Down as *developmental.*

From a series of statistics, collected by ourselves at a large pauper and at a large charitable institution respectively, embodying in the aggregate the history of 2380 cases, a few figures bearing upon the causes of idiocy may be quoted. Of pre-natal causes the most prominent was a family history of phthisis, noted in no less than 28·31 per cent of our cases. In 21·38 per cent hereditary mental weakness (insanity or imbecility) was recorded; and in 20 per cent epilepsy and allied neuroses were noted. Parental intemperance gave a percentage of 19·57 at the pauper asylum, and of 13·25 at the charitable institution (16·38 being the average of the two); the difference being probably due to the different social plane from which the patients were derived. Much stress has been laid by teetotal advocates on the paramount influence of parental intemperance on the procreation of a mentally deficient progeny; and although our figures (which have been collected with considerable care and discrimination) do not warrant the exaggerated views which are sometimes put forth, they are nevertheless sufficiently impressive to point this moral. Inherited syphilis was traced in no more than 1·17 per cent of our cases; and Sir Jonathan Hutchinson states the result of an inspection of the patients at Earlswood to be that he found "only a very few who could be reasonably suspected of being syphilitic." It is probable, however, that the subtle influence of inherited syphilis, even when not denoted by its more obvious stigmata, may be responsible for certain cases of mental and physical breakdown at the period of the second dentition or puberty which have been described under the name of "developmental general paralysis." Syphilis has been traced in 80 per cent of such cases (Mott).

Abnormal maternal conditions during pregnancy were recorded in 29·87 per cent of our cases; and, although it may be doubted if the mental causes assigned, such as shock, worry, and depression (amounting to 25 per cent), were in all cases adequate to interfere with due fetal development, there can be no reasonable doubt that the ill-health of the mother during gestation, or a serious accident—such as a fall downstairs or out of a carriage—not infrequently produces disastrous effects. The question of the influence of consanguinity of parents may here be referred to, though statistics do not throw much light upon the subject. It would appear that the proportion of idiot children who are the offspring of cousins is not much in excess of the ratio of consanguineous marriages to marriages generally; and that the influence for evil of such marriages is solely brought about by the intensification in the offspring of some morbid proclivity common to both parents.

Passing now to causes operating at birth, there is no doubt that protracted pressure during parturition is a frequent cause of mental impairment. In no less than 27·25 per cent of Dr. Beach's cases there was a history of protracted parturition, forceps delivery having been effected in 3·98 per cent only. J. Langdon-Down estimated the frequency of *asphyxia neonatorum* at 20 per cent amongst idiots generally; and at 40 per cent amongst those who are first-born children. Our joint figures give a lower percentage; but considering the pernicious effect on the brain of the condition which is the result of protracted pressure, the timely application of forceps in such cases is clearly indicated to the skilful obstetrician.

Infantile convulsions (eclampsia) give a percentage of 27·39 per cent in our combined cases; but it is open to question whether, in the majority, an innate morbid impressionability of the nervous system be not really the predominant factor. Epilepsy was assigned as the cause of the idiocy in about 12 per cent of Dr. Beach's cases; but the same observation applies to this as to the previous class. Dr. Shuttleworth's figures give 4·75 as the percentage in which mental enfeeblement was attributed to epilepsy as well as to inflammation of the brain and its membranes in early life, including acute hydrocephalus.

Infantile paralysis was recorded in only 0·92 per cent of our cases; and sunstroke in 0·54 per cent only. The influence of febrile illnesses as factors in mental impairment is noted in 5·96 per cent; acute brain symptoms were intercurrent with the majority, whilst in the rest atrophy of brain tissue resulted from the exhausting character of the illness. Injury (post-natal) figured in 6·17 per cent only of our cases.

We repeat, then, that the causes contributing to a given case of idiocy are usually complex. The figures which have been quoted above give the percentages of frequency of the various factors most commonly recognised; more than one being for the most part noted in each particular case.

In 1904 M. W. Barr attempted to assign in a table drawn up on the same lines as our own a single etiological cause for each of 3050 cases culled from the records of the large Pennsylvania Training School for Feeble-minded Children. For the reasons just stated we do not regard this as a satisfactory method, but it may be of interest to note that of the 3050 cases, 1978, or 64·85 per cent, are set down as due to pre-natal causes; 89, or 2·92 per cent, to causes acting at birth; and 983, or 32·23 per cent, to post-natal causes. In no less than 27·38 per cent of the first group of cases, a "family history of idiocy and imbecility" has been traced; that of insanity figures in 7·08 per cent of the cases, and of pulmonary tuberculosis in only 7·57 per cent, together with "scrofula" in a percentage of 1·18. Difficult labour and "accidents" in birth account each for 0·59 of the natal cases, and instrumental delivery is only recorded in 0·5 per cent. Amongst post-natal causes, "injury to head from falls" occupies the first place with a percentage of 6·26, epilepsy the second (5·90 per cent), and "acute disease" the third (4·46 per cent), though this last percentage might be doubled were the effects of the exanthemata also included.

Types.—Idiocy has been well defined as "mental deficiency, depending upon imperfect development, or disease of the nervous system, dating from birth or from early infancy, previous to the evolution of the mental faculties." Imbecility is a milder form of mental incapacity. The cases which come before us may be represented by eleven principal types, each of which has characters of its own; and on inquiring into the family history we find that some cases date from birth, whilst others have evidently been produced by causes which have acted on the child afterwards.

The most important types of idiocy will now be described.

1. First, there is the *simple congenital* idiot, who has no marked defect of the head or limbs. Such a patient, though below the average height, usually has a head of normal shape; the expression of the face, however, is vacant, the features coarse and heavy, the palpebral fissures deficient, the lips thick and held more or less apart, often with saliva flowing from them, the jaw heavy and sometimes badly hung, the palate highly arched, the teeth irregular and decayed, and the ears large, implanted far back, with a defective or adherent pinna or lobule. The sense of hearing is sometimes dull; an imperfection exaggerated by deficient attention. The child does not follow moving objects with his eyes, which remain fixed or wander restlessly; and the inco-ordination between the muscular efforts of the lips, tongue, throat, and chest explains the discordant or badly-modulated voice, and the more or less imperfect articulation. The patient walks with a clumsy, shuffling gait, and the hands perform even simple acts with difficulty. The body may be inert, or swing automatically backwards and forwards. The circulation is feeble, and the hands and feet are frequently cold. The ability to observe is often fairly developed, but the power of attention is defective and the memory weak.

2. The second type is the *mongolian*, thus designated by J. Langdon-Down in 1866 from likeness of the facial features to those of the Mongolian race. Imbeciles of this type are fairly frequent in the institutions of this country and of America; at the Royal Albert Asylum, Lancaster, they constitute more than 5 per cent of the total admissions: at Earlswood, 2·6 per cent; at the New York State Institution for Feeble-minded, 8·2 per cent. In at least 50 per cent of 350 cases studied by one of us (G. E. S.) they are the last-born children of a large family, the

age of the mothers in many cases verging on the climacteric. A family history of pulmonary tuberculosis seems commoner than in other types of imbecility; but parental syphilis, which Dr. G. A. Sutherland suggested as frequently bearing a causal relation to mongolism, has been but rarely noted by other observers. Some cause impairing the mother's vigour during gestation may usually be ascertained; and consequently fetal development does not proceed regularly, so that the child at birth may be described as "unfinished." The physical characteristics of this type are brachycephaly with flattened occiput; more or less obliquely-placed palpebral fissures; broad features with usually a flattened bridge of nose and epicanthus; sometimes outstanding pitcher-shaped ears; fissured lips and a coarse tongue, transversely fissured as the child grows older, and apt to be protruded; for the most part wiry hair with a tendency to the growth of down on the forehead, often a furfuraceous condition of skin and tendency to blepharitis. Strabismus is frequent and nystagmus occasionally seen. The stature is below the normal; there is a remarkable laxity of joints and delayed epiphyseal ossification. Hernia (especially umbilical) is not uncommon. The hands are broad and the fingers short and thick, though somewhat tapering towards the tips, and the thumb and little finger are apt to be abnormally short as compared with the other digits. The feet are also characteristic, often presenting an abnormal cleft between the great toe and the next, recalling those of the chimpanzee. Growth proceeds but slowly, and puberty is usually deferred. The circulation is feeble, and Dr. A. E. Garrod has described 6 cases of congenital heart-malformation associated with this condition, an association which, however, is comparatively rare in mongoloid patients surviving the age of three years, and therefore not often noticed in institutions. Such cases, however, are specially liable to chilblains, and to atonic inflammatory affections of serous and mucous membranes, and prone to the invasion of tuberculosis, a large number dying at or before puberty of some form of tuberculous disease—tuberculosis figuring in as many as 93·8 per cent of all mongoloid deaths at Earlswood Asylum. The voice is apt to be gruff and the speech indistinct.

As regards mental characteristics, a tendency to imitativeness and even mimicry is usually well marked, and the disposition is for the most part placid, though in some cases there are occasional ebullitions of temper. There is often a sense of humour and comicality of conduct which run in the direction of make-believe and play-acting. There are various degrees of mongolism, but the more superficial cases react favourably to suitable training, and acquire educational attainments of an imitative character, such as writing and drawing, without much difficulty, and even become fair readers and reciters. There is, however, but slight power of abstraction or of originality, and although a measure of manual aptitude may be obtained, and simple routine occupations acquired, the lack of initiative interferes with the development of practical industrial usefulness.

3. The *Cretinoid* Type.—Children so affected bear a remarkable

resemblance one to another as regards their bodily conformation, facial physiognomy, mental characteristics, and the symptoms generally. They are of diminutive stature, and have a dolichocephalic head, flat at the top, spread out at the sides, and broad behind. The hair is sparse, coarse, and dry, like horsehair; and is usually of a brown or black colour. The forehead is low and depressed laterally. The eyelids, of a pale bluish hue, are swollen and infiltrated with a solid oedema which does not pit on pressure. The subcutaneous tissue of the hands and feet is similarly affected. The nose is broad, the cheeks full and flabby, the mouth large, the lips thick and slightly apart, and the ears large but not malformed. The neck is short and thick, and usually presents no sign of a thyroid gland. Well-developed fatty swellings are seen in most cases on each side of the neck, and occasionally in the armpits; they sometimes disappear before death. The abdomen is large, distended, and contains in its walls a quantity of subcutaneous fat; frequently there are umbilical and occasionally inguinal herniae. The arms and legs are short, and the hands and feet broad, thick, and sometimes dry and scaly. The skin of the body, as well as of the upper and lower extremities, is thick, easily separable from the muscles, whitish in colour, dry and rough, owing to the almost entire absence of perspiration; on the face it is of a yellow or waxy sallowness. The voice is rough, harsh, or squeaky, the gait clumsy and waddling, and the attitude inert. The reflexes generally are slow, reaction time being much retarded. Speech is limited to a few words, often monosyllabic. These children are placid and good-tempered, and

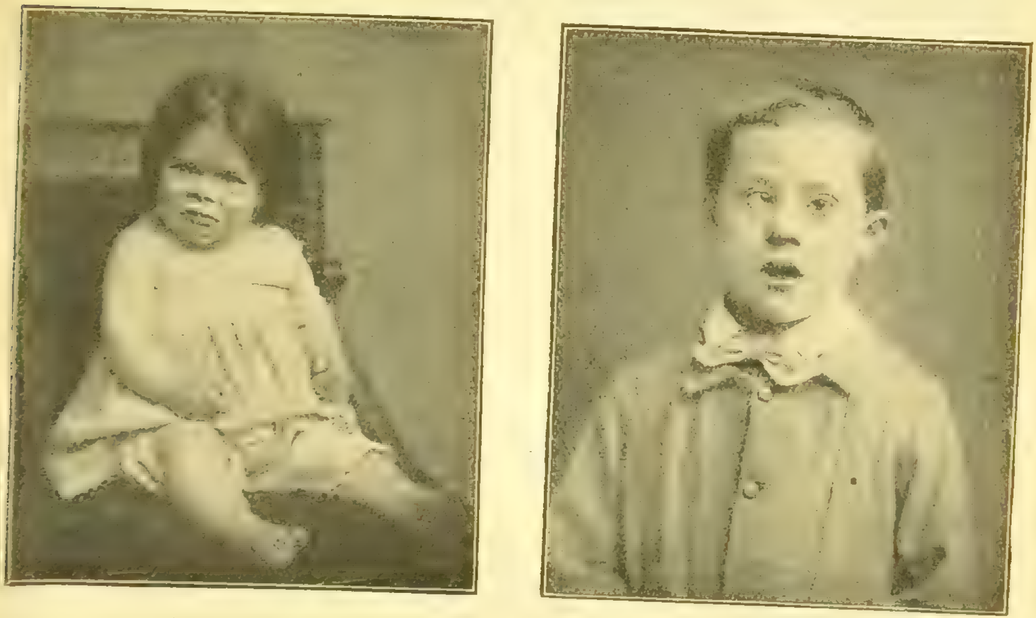

Fig. 26.—Photographs of mongolian and cretinoid imbeciles for comparison. Cretin girl aged 22 years; mongol boy aged 12 years.

fond of those who attend upon them. Death is due to bronchitis, convulsions, pneumonia, diarrhoea, or simple exhaustion. The thyroid treatment of cretinism, which is described elsewhere (Vol. IV. Part I. p. 341), must be kept up as long as the patient lives; otherwise he will relapse into his former condition.

Diagnosis of Mongolism and Cretinism.—Cases of the cretinoid and of the mongolian type are apt to be confused, especially at an early age, and a correct diagnosis is of importance to save parents the disappointment of expecting amelioration from thyroid medication prescribed for mongolian imbeciles presumed to be cretinoid. In passing it may be said that, except in *mixed* cases (which occur but rarely), thyroid treatment is ineffective in mongolism, and whenever a marked effect is obtained the presumption is in favour of cretinism, though this is a much rarer form of imbecility than the former. In tabular form the characteristic differences may be set forth as follows:—

Mongolism.	Cretinism.
1. Characteristics noticeable from birth.	1. Characteristics often not noticeable till sixth or seventh month.
2. Skull brachycephalic; contour rounded or short oval; longitudinal and transverse diameters nearly correspond.	2. Skull dolichocephalic; narrow in front, flat at top (fontanelles close late), expanded laterally, broad behind; often asymmetrical.
3. Forehead usually smooth.	3. Forehead usually wrinkled.
4. Palpebral fissures "almond-shaped," and more or less oblique upwards and outwards. Frequent epicanthus. Strabismus common. Ciliary blepharitis frequent.	4. Palpebral fissures horizontal, but appear small owing to pseudo-oedema of eyelids. Strabismus and ciliary blepharitis less common.
5. Cheeks chubby, often florid; complexion mottled.	5. Often circumscribed malar flush; complexion ashy or waxy.
6. Lips often transversely fissured; lower lip may be pursed up over upper.	6. Lower lip often everted. Mouth open. Drivelling common.
7. Tongue large and coarsely papillated, if not fissured. Tongue recurrently protruded and drawn back.	7. Tongue large, but not coarsely papillated or fissured. Tip of tongue thickened and constantly protruding.
8. Skin smooth in infancy, but furfuraceous later; not redundant or "baggy."	8. Skin dry and scaly; forms folds here and there, being redundant and "baggy."
9. Hair "wiry," often "mouse colour," sometimes blonde. Downy growth common on forehead and cheeks.	9. Hair harsh, coarse, and scanty; usually of darkish tint (Bourneville says brown); scalp often eczematous.
10. Thyroid gland palpable to greater or less extent.	10. Thyroid gland impalpable to most thorough examination.
11. No fatty tumours in the posterior triangle of the neck.	11. Fatty tumours (pseudo-lipomas) frequently found in the posterior triangle of the neck, etc.

Mongolism.	Cretinism.
12. Long bones somewhat shorter than usual, but slender.	12. Long bones shortened and thickened; in some cases bowed.
13. Hands broad; thumb and little finger short, the latter often incurved. Fingers taper at ends.	13. Hands broad, thick, and stumpy, with wrinkled skin. Fingers square at tips.
14. Feet large and flat. Wide cleft between great and next toe.	14. Feet squat. Skin redundant about ankles and dorsum of foot.
15. Abdomen often distended; occasional umbilical hernia; often inguinal hernia.	15. Abdomen very bulky and prominent, with folds of skin; umbilical hernia common.
16. Expression more or less vivacious and mobile; observant and imitative; more or less active.	16. Expression dull and immobile; unobservant and apathetic; all reactions slow.

Similarities in the Two Forms.—Deficient stature; flattened bridge of nose, with expanded alae; late and irregular dentition, deferred closure of fontanelles, retarded puberty.

4. The *microcephalic* type represents cases in which the head is smaller than normal, usually 17 or 18 inches in circumference, and occasionally 15 or 16 inches only. The smallest we have ever seen was that of a girl, aged twelve years, whose head measured only 12 inches in circumference; and her brain, after death, was found to weigh only 7 ounces. This girl led a vegetative life, but learnt to recognise those around her, became cleanly in her habits, and made some attempt to articulate. The head in these cases is oxycephalic in shape, and the occiput is flattened. The forehead is narrow from side to side and often recedes, but the features are usually shapely, the eyes large, and the nose aquiline. In some cases they have a bird-like aspect; in others, the face is large in proportion to the cranium. Though the lowest class of these patients have scarcely any power of observation, and will sit still all day, the higher class are active and lively. They are restless, imitative, and fond of music, but have little power of attention; their capacity for improvement is limited by their cranial deficiency.

5. In strong contrast with the above class are the *hydrocephalic* cases, with large heads. According to Meynert, in the congenital form the lateral ventricles are extended in their long diameter; whilst in acquired hydrocephalus the ventricles are increased in their transverse and vertical diameters. The educational treatment is the same for both these classes, but it cannot be put into operation until the disease ceases to be active: then, as experience shews, some improvement can be made, unless a large amount of fluid cause wasting of the grey matter of the brain. The head is rounded in shape, so that the antero-posterior and transverse diameters are nearly the same; the widest circumference is often at the temples, where there is sometimes a perceptible bulging above the usual place of greatest width around the superciliary ridges. The fontanelle is raised, thus differing from rickets in which it is depressed; and in the

latter affection the head is elongated in the antero-posterior diameter. Patients of this kind do not improve much, but are usually good-tempered, of a contented disposition, affectionate, and fond of those who attend upon them. The most intelligent hydrocephalic patient under our care, after seven years' training, learnt to read, to write, and to keep simple accounts, and became a useful farm-labourer; the remaining evidence of his imbecility being of a moral character.

6. A type with which the above is sometimes confounded is the *hypertrophic*, in which the brain is larger than normal. This and the types afterwards to be considered belong to the non-congenital class. The diagnosis between the two forms—the hydrocephalic and the hypertrophic—was described some years ago by Dr. Fletcher Beach, and rests on the following points. In hydrocephalus the increase in size of the head is most marked at the temples; in hypertrophy, above the superciliary ridges: in hypertrophy the head is square or perhaps elongated in shape; in hydrocephalus it is rounded: in hydrocephalus there is sometimes elasticity over the lately closed fontanelle; in hypertrophy there is none, and there is occasionally a depression in that situation: in hydrocephalus the distance between the eyes is increased; in hypertrophy this does not occur; moreover, the hypertrophic head does not attain so large a size as that of chronic hydrocephalus. This is a comparatively rare form.

7. Another important type is the *traumatic*; in this form the defect is due to a fall or blow on the head; or results from injuries to the head during birth, caused by a narrow pelvis and prolonged labour. In these cases the degree of imbecility produced varies with the extent of destruction of the nervous tissue, and the presence or absence of hereditary predisposition; and the same remarks apply to the amount of improvement which will result from training. These patients have usually well-shaped heads and limbs, and good features. They can walk steadily and see and hear perfectly; but as there is often great difficulty in fixing their attention, they do not speak, nor take any notice of what is said to them. In other cases more improvement is made, and the children make good progress under training.

Closely allied to the above are the *eclamptic* cases, in which convulsions come on soon after birth, continue some years and then cease, but with so injurious an effect on the brain that the child becomes imbecile. An illustrative case of this kind is that of a girl who was mentally sound at birth and was a lively child up to the age of two years. She had convulsions when cutting the teeth at the age of seven months; and the fits continued until she was five years old. She became very excitable and later very quiet. When under observation she was so listless that, if allowed to do so, she would sit still for hours. She would not speak, but would amuse herself by singing or humming tunes at various times. Although kept for a considerable period under training she learnt nothing, as her attention could not be gained. In other cases of this kind the excitability remains, and then there is constant movement, and the child is perpetually engaged in mischievous acts.

These patients have a bright expression, but the eyes wander restlessly and the attention cannot be fixed. They have full use of their limbs, but cannot bear restraint and often fly into a passion; during this excitement, if they cannot inflict pain on others, they will bite their own arms or hands, or stamp with their feet on the floor.

8. Then there is the *epileptic* type, which represents some patients who recover under treatment, others who remain stationary, and others again who go from bad to worse, and pass at length into dementia. The first sub-class are bright, well-made children, who progress at school, and take an interest in their work, whether educational or industrial. The second are also well-formed children, who are very listless, but can talk and take an interest in what goes on around them. They usually make fair progress; but, when doing well, a succession of fits comes on and throws them back, so that for a time they become lost and dazed. The third sub-class have a more animal type of face, are dull, and, in consequence of the frequent fits, make no progress whatever. They are often addicted to masturbation, and gradually become quite lost and demented. Cases of this type are very numerous, as at least 25 per cent of all idiots and imbeciles suffer more or less from epilepsy.

9. Another important type is the *inflammatory* or *post-febrile* type. Imbecility in these .cases supervenes after some illness, such as measles, whooping-cough, enteric or scarlet fever; these diseases cause inflammation of the brain and its membranes (or are complicated with it), not sufficiently grave to be fatal, but serious enough to cause mental impairment. As Ireland truly says: "The amount of damage to the intellectual powers must be mainly dependent upon the intensity of the morbid process." This we seldom have an opportunity of measuring, as the child does not come under our care until the disease has passed away. Of course, if one of the diseases we have mentioned should occur in a child previously disposed to imbecility, this affection is more likely to follow it. Many of these patients do remarkably well under training. In a girl, who was under our care, the imbecility came on after an attack of measles when she was four years old. For some time afterwards she remained excitable, passionate, and uncontrollable; but when she came under observation she was well developed and nice-looking, with good use of her limbs, in full possession of her senses, and able to speak and talk. She made considerable progress, both educationally and industrially, and made herself useful in every way.

10. A type frequently met with is the *paralytic*. The paralysis may come on before or after birth, but the majority of cases belong to the latter category. Generally these patients make fair progress mentally, if there be no fits, and the injury to the brain be not great; but little can be done for the paralysed limbs. In cases of birth palsy, on the other hand (in which athetosis is often present), considerable improvement results from training. Patients included in the latter class, however, in whom the imbecility and paralysis have come on after repeated fits, cerebral apoplexy, or atrophy of the brain, do not make much progress,

either physically or mentally, for the fits interfere with improvement or arrest it.

There are other types of less importance, such as the amaurotic, syphilitic, choreic, scaphocephalic, which need not receive detailed description here. (For "Amaurotic Family Idiocy" see p. 468.)

Pathology.—The pathology of idiocy will be considered in respect of certain differences and exceptions.

Hypertrophy of the brain is occasionally met with, and is of interest because it is often mistaken for chronic hydrocephalus. It is due to increase of the neuroglia of the white matter of the brain; there is not only mere growth, but the nutrition of the cerebrum is modified as well. The brain substance is usually white and hard, and cuts like cheese; generally it is anaemic on section, but there may be congestion of the membranes and increase of the subarachnoid fluid, and of that in the lateral ventricles. On removing the calvaria the brain often springs up as if under pressure; and on closer inspection the sulci will not be noticed, as the convolutions are flattened and pressed together. The parts affected are chiefly the white matter of the two hemispheres, the corpus striatum, and optic thalamus. Death usually occurs from convulsions, or from an intercurrent disease, such as pneumonia, bronchitis, or diarrhoea. The weight of the brain is considerably increased; in one case under our care, a boy aged fifteen years, this organ weighed 62 ounces; a weight of from 49 to 53 ounces, which is much above the average in childhood, is fairly common. As a rule the head in this disease is not so large as in chronic hydrocephalus; it is square, or somewhat elongated in shape, whereas in hydrocephalus it is rounded. In hypertrophy there is no increase in the distance between the eyes, a condition commonly noticed in hydrocephalus. There are other differences, but the above are those on which we lay most stress.

The opposite condition is *atrophy of the brain*, which is often seen; it is present at birth, or comes on some time afterwards. The most common form of the congenital class is the microcephalic brain, in which there is incomplete development. The deficiency affects chiefly the anterior, upper, and occipital convolutions; the two former are often very simple in arrangement, whilst the insufficient occipital development causes the brain to be shortened, and the cerebellum to be to a great extent uncovered. The ganglia of the base are not affected in the same proportion, and the cerebellum retains its normal size. The cause of the disease is obscure; it was formerly attributed to premature synostosis of the cranial sutures, but this condition exists in exceptional cases only.

In the acquired form there is a loss of nerve-structure from inflammation of the brain, meninges, or skull, occurring either before birth or supervening in infancy. Cases of unilateral atrophy of one hemisphere, with coexistent atrophy of the limbs on the opposite side, are seen very frequently. On making a necropsy we find a thickened cranium on the affected side, opacity and increased thickness of the membranes, effusion of fluid into the subarachnoid space or into the ventricles, and atrophy of one hemisphere of the brain, with atrophy of the cerebellum and spinal cord on the opposite side.

Sclerosis of the brain is either diffuse and affects large portions, or multiple and disseminated in patches. The former kind is the more common of the two. It involves a considerable part of one hemisphere, and is not distinctly circumscribed; the medullary substance is chiefly affected. The frontal, ascending frontal, ascending parietal, and occipital convolutions are those which are mostly implicated. The white matter is hard and looks on section like white of egg, though sometimes there is a honeycombed appearance. The increased hardness and density are due to an overgrowth of the neuroglia, which compresses the nerve-fibres, and finally causes their disappearance. The disease is due, no doubt, to a chronic inflammation of the membranes; and we find, on examination, increase and distension of the blood-vessels, infiltration of the perivascular sheaths with leucocytes which sometimes make their way into the surrounding tissue, and occasionally an increase of the fibrous tissue around the vessels. In disseminated sclerosis patches are found scattered through the cerebrum, cerebellum, basal ganglia, pons, medulla, and spinal cord. The convolutions of the brain are usually exempt. The patches are circumscribed and tough, and in the spinal cord vary in size from a pin's head to a hazel-nut; they are usually larger in the brain matter itself. The meninges of the brain and spinal cord may be healthy, or there may be signs of congestion or chronic inflammation. The cerebrospinal fluid, which is increased, is sometimes cloudy, and the lateral ventricles are dilated.

Porencephalus is a condition closely allied to chronic hydrocephalus, as fluid is found in the place of brain substance. The defect is either congenital and caused by arrest of development of the brain, or it may be an acquired condition. Usually, there is a cavity on the surface of the hemisphere which may communicate with the arachnoid sac, or more commonly with the ventricular portion of the brain. Sometimes, and especially in the acquired form, small cysts are found which contain clear straw-coloured or gelatinous fluid. The third ventricle, the lateral ventricles, the occipital lobes, and the convex surfaces of the right frontal regions are the parts chiefly affected. These cavities may be the result of antecedent haemorrhage into the substance of the brain.

Softening of the brain is either general, or limited to certain portions of it; it is usually due to a feeble circulation, or to a diminished supply of blood. The part involved is white in colour. The chief causes are an effusion of blood into the ventricles and subarachnoid space, chronic hydrocephalus, thrombosis of the middle cerebral arteries or superior longitudinal sinus, and tuberculous meningitis. The central white matter of the hemisphere, the corpus striatum and optic thalamus, fornix and septum lucidum are the parts chiefly implicated.

Tumours of the brain are generally tuberculous in origin, but occasionally are of a gliomatous nature. Sometimes tuberculous masses are found scattered through the substance of the brain, or gliomas of the size of a walnut or small apple may be met with, causing flattening of the convolutions over them. Occasionally psammomas are found attached to the choroid plexuses; and sometimes long masses growing from the dura mater make their appearance.

Simplicity of the convolutions is often noticed. Sometimes we find, on examination, convolutions of half an inch or even one inch in width. Of course, in these cases the arrangement is as simple as possible. As a rule all are more or less affected. Occasionally the convolutions of both sides are equally involved.

Asymmetry of the hemispheres and convolutions, an altered relation between the grey and white matter of the brain, thickening of the arteries, and thrombosis of arteries and veins are sometimes found.

Diseases of the membranes of the brain, affecting chiefly the convex surface, are often seen, especially in the chronic form. On examination, we find congestion and thickening of the dura mater; thickening and opacity of the arachnoid membrane; adhesion of the dura mater to the skull, of the membranes to each other, and of the pia mater to the brain; increase of the subarachnoid fluid, which may be clear, turbid, or purulent in character; and an excess of serum in the ventricles, which are dilated, with their lining membrane rough.

The acute disease takes chiefly the form of tuberculous meningitis, and in the cases we have seen it ran a lengthened course. The miliary tubercles are found in the meshes of the pia mater; especially over the longitudinal sinus and the Sylvian fissure, the middle cerebral artery, pons, and medulla. At the base the membranes are thickened and opaque, and the meshes surrounding the optic commissure and Sylvian fissure are filled with yellow exudation. The lateral ventricles are dilated and filled with fluid, and the central parts of the brain are softened.

The most common *diseases of the cerebellum* are atrophy, tumours, and cysts. Atrophy has already been mentioned in connexion with atrophy of the brain, and requires no further description. Tumours are chiefly tuberculous in character, and in one case we have seen several cysts.

The diseases of the spinal cord most usually seen are acute poliomyelitis—giving rise to infantile paralysis, which, when it comes under our notice, has assumed the chronic form; congenital arrest of development of the pyramidal tracts, or descending sclerosis of the pyramidal tracts, both of which cause spastic paraplegia; unilateral descending sclerosis in connexion with disease of the brain; and chronic myelitis. The last form may be transverse myelitis, extending so as to cause destruction of the ganglion-cells of the anterior horns. Sometimes the transverse form ends in softening, or is associated with ascending sclerosis of the columns of Goll and descending sclerosis of the pyramidal tracts of the lateral columns. External spinal pachymeningitis, internal hypertrophic pachymeningitis, and tuberculous spinal lepto-meningitis are occasionally found.

Affections of the Cranium.—After removing the calvaria and holding up the convex surface to the light, it is sometimes seen to be diaphanous in places; especially in the region of the anterior fontanelle, which occasionally persists. The sides and arch of the cranium are in a few cases thickened and sometimes asymmetrical; the former condition occurs when there is atrophy of the brain on one side and the skull is thickened to compensate the wasting; the latter, when one hemisphere of the brain is smaller than the other, or when the brain grows toward the side where there is least resistance. In a few cases the sutures of the skull become closed soon after birth, and then a microcephalic skull is formed; this form of skull is often characterised by a sloping away of the frontal part where the temples are situated,. when an oval brain is formed. The scaphocephalic, conical, and other abnormally shaped skulls are caused by too early ossification of the sagittal, lambdoidal and coronal, or other sutures. The differences between the chronic hydrocephalic and hypertrophic skulls have already been mentioned. At the base there is often found in cretinism, as well as in other forms of idiocy, premature synostosis of the sphenoid with the basilar part of the occipital bone.

The cells of the brain in idiocy are generally rounded or pear-shaped and deficient in angles and processes. In 1879 Dr. Bevan Lewis drew attention to the frequency, in certain forms of amentia associated with epilepsy, of incompletely-developed nerve-cells, resembling those which exist normally in the second and third layers of the cerebral cortex of the ape. Dr. Tredgold has remarked that in primary amentia the nerve-cells of the cerebral cortex are characterised by the following conditions as compared with those of the healthy brain:—(1) numerical deficiency; (2) irregular arrangement; (3) imperfect development of individual cells; the pyramidal layers shewing the most obvious abnormalities, including in the deeper layer pigmentation of the cells at one angle. Wilmarth noticed overgrowth of neuroglia in 25 per cent of the hundred brains of the mentally defective children carefully examined by him.

Treatment and Training.—Indications for training vary with the different forms of mental deficiency; consequently a knowledge of the leading characteristics of the several types is essential in determining that which is most appropriate in a particular case. The inert, impassive idiot, for example, requires rousing, stimulating treatment; the child of restless, nervously excitable temperament is the better for soothing influences, with such exercises as will tend to bring its irregular outflows of energy under control. For successful treatment co-operation between patient, physician, teacher, and nurse is required; and in this article we propose to consider briefly various ameliorative measures under the heads of:—

A. *General management.* B. *Medical and surgical treatment.* C. *Educational, industrial, and moral training.*

A. *General Management.*—The home training of a mentally deficient child cannot begin too early. In the words of Séguin (*Idiocy*, p. 88): "As soon as any function is set down as deficient at its due time of development, the cause must be sought and combated; if external, removed; if seated in the nervous apparatus, counteracted by the earliest course of training and hygienic measures." Unhappily the mothers of weak-minded children are, as a rule, not themselves sufficiently strong-minded to carry out any system; but a judicious nurse may do much to check purposeless movements (apt to become automatic), and to promote habits of cleanliness. It is the duty of the medical attendant to point out the importance of persevering efforts, in spite of small and tardy results. Regularity of the bodily functions should by every means be encouraged; and gentle discipline will end in the formation of cleanly habits, of infinite importance in the after-life of the child. The "laissez-faire" system, which is content to treat the growing imbecile as a perpetual infant, to swathe him in napkins like a baby, and to inure him to the contact of wet and soiled clothing, is a mistake. The child should be encouraged to indicate his wants, and to avail himself of the opportunities of relief regularly afforded him. Cleanliness of body is to be promoted by frequent warm baths and frictions of the skin, the exhalations from which are apt to be offensive. Amongst other hygienic measures, sufficient exercise in the fresh air is of the first importance. This is apt to be neglected; sometimes from a shrinking from exposure on the part of parents, sometimes from difficulties arising from the physical infirmities of the patient. The superior facilities for outdoor life is one recommendation of a residence in the country rather than in town; and in view of the proclivity of idiots and imbeciles to tuberculous affections, a dry gravel soil and a bracing atmosphere are desirable. The dwelling must be bright, cheerful, well ventilated, and well warmed. A *diet* comparatively rich in carbohydrates is needed, as in these children thermogenic powers are deficient, and the extremities prone to chill. Clothing likewise must be warm and appropriate. The body should be encased in wool, the underwear being of varying thickness according to the season. The cut of the clothing should be such as to conceal and not to accentuate the bodily defects. Whilst some modification may be required to meet peculiar habits (such, for example, as the provision of slavering-bibs in some cases), it should assimilate as far as possible to that of ordinary children of the same age. As a rule, the wearing of india-rubber urinals, and other means of avoiding "accidents," militate against care in improvement of the habits.

B. *Medical and surgical treatment* consists, of course, in the application of general principles to the special necessities of the idiotic class. We must content ourselves by referring to a few of the conditions most commonly met with. In the first place, feeble-minded children are, as a rule, feeble-bodied; and mental improvement depends to a considerable extent upon physical amelioration. The improvement of the general nutrition, of the quality of the blood, of the nervous powers, and of bodily infirmities is therefore essential. Cod-liver oil, glycero-phosphates, Parrish's chemical food, iron and quinine in their various combinations, are often serviceable; and in loss of nerve-power and wasting of muscles, electrical treatment and massage may be usefully tried. The mucous diarrhoea which is frequently met with, especially in the mongolian class, is best combated by an emulsion of castor oil with small doses of opium; in the fermentative variety with fetid stools, minute doses of carbolic acid, or of sulpho-carbolates, have proved useful. Enuresis may be dealt with by diminishing the amount of fluid imbibed at the evening meal, and by increasing doses of belladonna or atropine combined with bromides. Various discharges from eyes, nose, and ears may be treated by astringent lotions, boric or perchloride washes, and scrupulous cleanliness. A spongy condition of gums is often met with in this class, and calls for strict attention to the hygiene of the mouth, and the administration of chlorate of potassium. For chilblains, so common with idiots of feeble circulation, prevention is, of course, better than cure; and, as prophylactics, cod-liver oil and the painting of the toes with iodine are frequently of benefit. To diminish irritation (and consequent scratching) bathing with dilute solution of hydrogen peroxide is often serviceable. Frictions of the feet and legs are also called for, and in frosty weather the wearing of wash-leather socks both by night and day is an advantage. Where epilepsy complicates idiocy bromide treatment may be tried, but not to an extent as to depress the patient. In some cases borax and sodium bromide in combination appear more efficacious than bromide alone; and the diet, although nutritious, should not be rich in nitrogenous elements. The treatment of cretinism is described elsewhere (Vol. IV. Part I. 341).

As regards surgical treatment it may be said that, as a rule, idiots are bad subjects for operations, which should only be undertaken when urgently necessary. Craniectomy, which in former years was practised as a remedy for microcephaly, proceeds on an irrational basis, and the results have been eminently unsatisfactory. Bourneville, indeed, has adduced the evidence of necropsies to shew that in the long run such excisions tend to diminish (instead of increasing) the cranial capacity, as exuberant growth of bone is thrown out to replace that which was removed by the surgeon. Post-nasal growths (adenoids, etc.), frequently met with in abnormal children, should be removed, as they tend to intensify stupidity.

C. *Educational, Industrial, and Moral Training.*—Under educational we include physical as well as mental training, inasmuch as the two are more or less interdependent. Starting with exercises calculated to improve and to co-ordinate the muscular powers, we endeavour by the simplest forms of drill—such as clapping the hands in time to music—to strengthen the child's powers of attention and of imitation. Breathing exercises are also useful, special care being taken to promote nasal breathing with closed mouth. We note also any sensorial deficiencies, and endeavour to remedy these by appropriate exercises. Following the lead of nature in the spontaneous education of the normal infant, we proceed to place before our pupil contrasting substances, which he is encouraged not only to see, and if necessary to smell and to taste, but also to handle—the sense of touch serving to correct the impressions through the other senses. By the persevering application of such methods the intellectual activity is at length aroused, and exercises, at first purely imitative, are performed with discrimination. It is impossible, in the compass of an article like this, to give a detailed account of the various devices by which the above-described processes are effected: we can merely mention *bean-bags, peg-boards, size and form boards* as some of the apparatus found useful for the purpose. From these preliminary exercises the pupils readily pass to the occupations of the Kindergarten; and of these we may mention bead-threading, macramé-work, and paper-weaving as specially serviceable not only for training the fingers but also for exercising the intelligence. Speech is, as a rule, more or less imperfect in mentally deficient children, and when the imperfection is not due to structural defect, much may be done to improve it by lip and tongue gymnastics, followed by systematic lessons in articulation. Music, which has special attractions for the mentally deficient class, is often a good incentive to speech; the words of nursery rhymes, repeatedly sung to attractive tunes, being gradually acquired. Some idiots have a remarkably correct sense of time and tune; and in consequence music is an excellent auxiliary to drill, which should be arranged so as to be not merely an imitative exercise, but to call forth the pupil's powers of attention and prompt action. Of what is regarded as ordinary school work we can only say here that all instruction must be illustrated as far as possible, and that every step in calculation must be objectively demonstrated. Mere learning by rote is of little avail; and in the education of mentally feeble children parrot-like repetition should be carefully avoided.

Industrial training is really of more importance to this class of pupils than literary acquirement, and Kindergarten occupations form an excellent introduction to manual work. Later, instruction in sloyd, chip-carving, and carpentry may be given to those who shew a taste for mechanical work; whilst others may prefer tailoring, shoe-making, or mat-making. For adolescent boys outdoor work in the garden, or at a farm, is of special value; for girls, sewing and work in the house, kitchen, or laundry, are most likely to be useful, and patients so employed will lead much happier lives. Care must be taken to vary the monotony of training as much as possible; and the necessity of frequent recreation must not be lost sight of.

Moral training must proceed together with the intellectual; and good discipline must be maintained by kindly firmness. The aid of religion is valuable in promoting truthfulness, honesty, and straightforwardness of conduct; and the duty of doing to others as one would be done by must be steadily enforced.

In conclusion, we may point out that such training as that above described can hardly be carried out satisfactorily at a patient's own home; to obtain the best results the varied resources of a specially equipped school or institution are desirable, and even mentally deficient children are the better for such emulation as judicious classification affords. For those who desire to follow up the sociological aspects of the subject, apart from those strictly medical, we would recommend reference to the very comprehensive *Report of the Royal Commission on the Care and Control of the Feeble-Minded* issued (in 8 volumes) in 1908.

G. E. Shuttleworth.
Fletcher Beach.

REFERENCES

1. Andriezen, W. L. "The Pathogenesis of Epileptic Idiocy and Epilepti Imbecility," *Brit. Med. Journ.*, 1897, i. 1081.—2. Barr, M. W. *Mental Defectives, their History, Treatment, and Training*, Philadelphia, 1904.—3. Beach, Fletcher. *Treatment and Education of Mentally Feeble Children*, London, 1895.—4. *Idem.* "The Etiology of Sporadic Cretinism," *Brit. Med. Journ.*, 1896, ii. 620.—5. *Idem.* "Clinical Lecture on Mental Deficiency in Children," *Clin. Journ.*, 1897, x. 227.—6. *Idem.* "The Mentally Feeble Child and How to Train Him," *Pediat.*, 1897, iv. 490.—7. *Idem.* "Insanity in Children," *Journ. Ment. Sc.*, 1898, xliv. 459.—8. Bevan-Lewis. *Textbook of Mental Diseases*, London, 1899.—9. Bourneville. *Recherches cliniques et thérapeutiques sur l'épilepsie, l'hystèrie et l'idiotie*, Paris, 1895 to 1903.—10. Carswell. "The Care and Education of Weak-minded and Imbecile Children in Relation to Pauper Lunacy," *Journ. Ment. Sc.*, 1898, xliv. 474.—11. Cunningham, D. J., and Telford-Smith, T. *The Brain of the Microcephalic Idiot*, Dublin, 1895.—12. Fennell, C. H. *Journ. Ment. Sc.*, London, 1904, l. 32.—13. Garrod, A. E. "The Association of Congenital Heart Disease with Mongolian Type of Idiocy," *Trans. Clin. Soc.*, London, 1898, xxxi. 316.—14. Hammarberg, Carl. *Studien über Klinik und Pathologie der Idiotie*, Upsala, 1895.—15. Hill. "Pathology of Mongolism," *Quart. Journ. Med.*, Oxford, 1909, ii. 49.—16. Ireland, W. W. *Mental Affections of Children*, London and Edinburgh, 1898.—17. *Journal of Psycho-Asthenics*, published quarterly at Faribault, Minnesota, U.S.A.—18. Looft, Carl. *Kliniske og aetiologiske Studier over Psychiske Udviklingsmanger hos Born*, Bergen, 1897.—19. Mogridge, G. "Imbecility and Mental Deficiency in Children," *Omaha Clinic*, 1894-95, vii. 450-453.—20. Mott, F. W. *Arch. Neurol.*, Lond., 1899, i.; 1903, ii.—21. Peterson, F. "The Stigmata of Degeneration," *State Hospitals Bulletin*, Utica, N.Y., 1896.—22. *Proc. Amer. Assoc. Medical Officers of Institutions for Feeble-Minded*, etc., published annually.—22*a*. *Proc. Roy. Soc. Med.*, 1909, ii. (Clin. Sect.), 187-194.—22*b*. *Rep. Soc. Study Disease in Children*, London, 1900-1907, i. 3, 65, 126, 157; ii. 218; iii. 150; v. 21; vi. 16; vii. 101, 108; viii. 210, 276, 278, 407, 460.—23. *Report of the Departmental Committee on Defective and Epileptic Children*, London, 1898.—24. Sachs. "Amaurotic Idiocy," *New York Med. Journ.*, 1896, lxiii. 697.—25. Sante de Sanctie. "Mongolismus," *Eos*, 1909, v. 82, *seq.*—26. Séguin. *Idiocy*, 1866.—27. Shuttleworth, G. E. and Potts, W. A. *Mentally Deficient Children—their Treatment and Training* (3rd edition), London, 1910.—28. Shuttleworth. "The Industrial Training of Imbeciles," *Journ. Ment. Sc.*, 1898, xliv. 531.—29. *Idem.* "New Medical and Surgical Methods in the Treatment of Mental Defects," *Medical Annual*, Bristol, 1895, 324, *seq.*—29*a*. *Idem.* "Mongolian Imbecility," *Brit. Med. Journ.*, 1909. ii. 661.—30. Spiller, W. G. "A Contribution to the Pathology of Imbecility and Idiocy," *Phila. Med. Journ.*, 1878, i. No. 11.—31. Still, G. F. *Common Disorders of Childhood*, chapter xxxvi., London, 1909.—32. Telford-Smith, T. "The Paralytic Type of Idiocy and Imbecility," *Pediat.*, 1898, v. 541.—33. Thomson, John. *Clinical Examination and Treatment of Sick Children* (2nd edition), London and Edinburgh, 1908.—34. Tredgold, A. F. *Mental Deficiency*, London, 1908.—35. Voisin, Jules. *L'Idiotie*, Paris, 1893.—36. Weygandt. *Psychiatrie*, Munich, 1902.

G. E. S.

MANIA

By G. H. SAVAGE, M.D., F.R.C.P.

VARIOUS names have been given to the several forms of maniacal excitement which need no special mention here.

Some forms have been named from their causes, as for instance alcoholic mania, which, however, resembles ordinary mania; though the alcoholic factor may give it a delirious aspect.

Ephemeral mania may follow epilepsy, childbirth, or any toxic influence.

Senile mania is often erotic in tendency, and leads rapidly to exhaustion; in these cases early and careful feeding is essential.

Chronic mania is in some cases chronic from the outset; in others it is secondary to previous attacks of insanity.

Recurrent mania occurs as a rule in neurotic persons who, having had several attacks of acute mania, pass into a state of permanent instability, so that they are never long without recurring attacks of excitement, which, as a rule, resemble one another almost in every detail; such attacks may recur during thirty or forty years, leaving the patient, between the attacks, sane and with little mental defect. In such cases asylum treatment is almost essential; though some can be managed in private care when the periods of excitement are short and the patients physically feeble.

ACUTE MANIA.—**Definition.**—A form of mental disorder depending on altered nutrition of the highest nerve-centres, marked by loss of control of the higher psychical processes, which gives rise to mental excitement and disorder in the conduct of the individual. This defect of control varies in degree as well as in aspect, the variations depending on the heredity, education, and general surroundings of the patient, as well as on the remote and proximate causes of the disorder.

Forms of Mania.—Acute mania is seen in two very distinct forms—the one approaching delirium, the other resembling hysteria. We shall consider these two different groups of symptoms apart, as they are in most respects utterly unlike each other; though the chief characteristics in both consist in defect of the higher nervous control with restless excitement.

The following general remarks hold good of both forms. Mania may arise from disease of the brain itself: thus it is common as an early symptom of general paralysis of the insane; it may depend on general bodily disease affecting the nutrition of the brain, and may thus be met with in various blood disorders; it may arise in conditions of defective blood-supply to the brain, or be due to blood poisoning, as in alcoholism, uraemia, plumbism; or to general sepsis, as in puerperal states.

Mania may be part of a morbid mental process, or it may be the whole. Thus there may be maniacal stages in *folie circulaire* and in some chronic forms of mental disorder; or there may be a maniacal outburst during general paralysis, or with epileptic insanity.

Mania may alternate with periods of mental depression, and is now described as a special form of mental disorder under the name of maniacal depressive insanity. Mania rarely arises without some prodromal symptoms.

Simple Mania without Delusions. (Simple Hysterical Mania.)—This form of disorder is most frequently met with in persons who come of neurotic families. Such persons have more than ordinary mental ability, but with lack of persistence. They are often attacked for the first time at or about puberty; and the attacks are generally associated with some slight exciting cause, such as emotional upset, physical illness, disappointment or the like. A young man or woman, after some emotional or physical disturbance, becomes sleepless, and then complains of vague uneasy pains in the head; various neuralgic discomforts are felt: change of scene is recommended, possibly with temporary relief; then more complaints are made, of a very hypochondriacal nature, the patient being restless, discontented, and almost always stirring up family troubles: this stage may last for a few days, or for weeks together, then a spirit of opposition arises so that everything seems to be done out of perversity. These patients have a marvellous power of upsetting homes and friends. They make frivolous complaints, exaggerate events, and often tell untruths without any design. They say and do what comes first into their minds. They rapidly become less and less conventional. Thus a lady will smoke, talk slang, or be extravagant in dress, and will declare her intention of doing as she likes. At this stage love affairs and like complications are common. In men sexual and alcoholic indulgences generally follow; whilst in women masturbation or unseemly language and gesture are present. The sleep is broken, the appetite large or capricious, the pulse often rather rapid and weak, temperature normal, the general condition feeble, irritable, and anaemic.

There is, moreover, general perversion of sentiment and of feeling. In the early stages it is common to meet with patients who change their religion. As a rule with each recurrence a similar religious version takes place; the Protestant seeks advice from a Romanist, and the Dissenter turns to the Church.

At first the general aspect of the case is very hysterical; a girl will receive the doctor in bed, dressed in the most fanciful of bedgowns, with her hair, her flowers, her books and nick-nacks all arranged for effect. The pupils are generally dilated and mobile; the tongue tremulous and moist, with perhaps some white fur; bowels generally confined with some gastric symptoms; urine often abundant and of low specific gravity; menstruation often profuse or irregular. The mental disturbance is worse at the menstrual periods. A patient in this stage may never get beyond home control, though she is often very hard to bear with in a private house. The milder cases are rarely certifiable, unless some foolish act betray the disease, or the patient refuse food, or threaten suicide; yet removal from home at an early date is the essential means of cure. In some cases, however, the loss of self-control becomes more obvious, and no doubt remains about the necessity for legal control. This simple hysterical mania may be but an early stage of ordinary acute mania with delusions, or it may be the early stage of acute delirious mania. If the attack follow its ordinary course, after some weeks or months the patient becomes dull, complaining frequently of symptoms like those with which the attack came on, such as neuralgia or weakness; this period is one of anxiety, for suicidal attempts are then not uncommon; the sensitive neurotic has discovered his tendency to insanity and dreads the future. With complete rest, followed by quiet travel (in men a sea voyage is often useful), the normal level is once more reached. Such an attack may be completely recovered from, and there may never be a recurrence; on the other hand, if a fresh and similar attack occur within a year or eighteen months, I think the prognosis is very bad; the prospect being that the patient will be liable to recurrences which will tend to mental weakness or to more permanent mental instability, recurrent mania, recurrent melancholia, or partial mental weakness with periodical outbursts of excitement. Another not uncommon termination is for the patient, after several attacks of mental excitement, to die of nervous exhaustion, or of some secondary lung trouble with which there is not nerve power to contend.

The *treatment* of this form of disorder is removal from home and relations (in many cases there is no necessity for certification), simple diet, abundant exercise in the open air, abstinence from alcohol; in the cases of girls it is well, if possible, that a nurse should occupy the same bed to check masturbation. Saline purgatives will probably be required. It is certain that sexual excitement must be avoided. I have known marriage suggested, and even tried, as a cure, with the most disastrous results.

As far as drugs are concerned I do not approve of the continual use of bromides or chloral, singly or combined; at times they may be useful, but conditions of life are more efficient than drugs. With sexual excitement I give dram doses of extract of salix nigra, with small doses of bromide thrice daily. Hydrotherapy is useful.

Just as at puberty these cases of hysterical or emotional insanity without delusions may occur, so in the decline of life similar disorders may arise; men who, till they were fifty-five or sixty, had led exemplary lives, lose self-control and do foolish things; such attacks frequently pass off in the same way as in adolescents, leaving a depression of spirits which is regarded by friends as proper contrition. However, similar attacks may recur, and not infrequently lead to moral and financial ruin before their true nature is recognised. In nearly all these senile cases there is a recrudescence of lust which leads to foolish or immoral connexions, senile marriages, and, occasionally, to sexual crimes or misdemeanours. The general termination of these cases is in mental enfeeblement, though the outbreaks of excitement may still recur.

Though these cases are common towards the end of life, they may also occur as the first stage of general paralysis; or at any age they may be met with in states of nervous exhaustion following bodily disease.

Impulsive Mania.—These are cases of sudden maniacal excitement without a stage either of depression or of exhaustion. They may be epileptic in origin, either preceding, replacing, or following an epileptic attack, whether of the major or minor form (*vide* art. "Insanity and Epilepsy"). These attacks, regarded as simple explosive discharges of nervous force, may be very sudden, violent, and destructive; fully formed and apparently purposive acts may be performed, and yet the patient may have no memory of any part of the attack; in this respect he differs from those suffering from ordinary mania, in whom there is almost always a memory of the illness, though in the hysterical this is often denied. I have met with sudden ephemeral attacks of mania associated with chronic mental weakness and with nervous exhaustion, especially if the exhaustion were due to sexual excess or masturbation; it is also worthy of notice that some chronic cases of delusional insanity seem to present sudden impulsive outbreaks of mania—the truth being that such patients have suffered for weeks or months from mental troubles which they have concealed. Obsessions and impulses have been looked upon as parallel. In the one case there are irresistible ideas, in the other irresistible actions.

Simple acute mania may depend on brain disease or on bodily disease which affects the nutrition of the brain, and may lead to loss of higher control. Though most common in early and middle life it may occur at any age; it occurs nearly equally in both sexes. It is favoured by neurotic heredity and previous attacks; the immediate causes may be physical or moral. Any cause of delirium, such as alcohol or toxaemia, may set up mania.

The *symptoms* depend on defective control of the thoughts and feelings, which leads to incoherence in language and impulsiveness in act.

The earlier stages are marked by general malaise with dread of some impending evil; there is a restless, emotional state with disturbed sleep and painful dreams, capricious appetite, rapid circulation, disordered digestion, sallow skin, and lithatic urine. In women menstruation is often excessive or too frequent; and there is general ill-health of a vague nature with little or probably no increase in bodily temperature. Common sensibility may be affected, there being hyperaesthesia or local patches of changed or defective sensibility. The mind is equally disturbed; for, though power of perception and even memory are retained, there is want of power of application and attention, and the thoughts appear without control; there is a tendency to react to the first impression without consideration of any but immediate consequences. This is a kind of uncontrolled or exaggerated mental reflex. A sensory impression starts a train of thought which is at once diverted by the next sensory impression. In the same way a visual or auditory suggestion may give rise to a direct and unbalanced action.

In the above ways incoherence of language and impulsive action follow. A patient seeing a "ring" on your finger may at once say, "fling," "sing," "bring"; then as rapidly pass from verbal association to ideational, and say "gold," "bank," "robber." Or on seeing the ring he may begin as before; but as he is playing on the word "ring," a sound strikes his ear and he goes on from ring to "music," "ringing changes," "church bells," and so on.

It is interesting to note that some maniacal patients react more readily to impressions on one sense than they do to those on another; and in the same way the impulsive reaction will be greater on some lines than on others.

The period of malaise above described may be more profound, and may amount to a true melancholic stage in which all the ordinary symptoms of melancholia are present; this stage varies very greatly in duration, in some instances lasting only a few days, or even hours, whilst in some it may continue for many weeks or months.

Some cases of ordinary melancholia which have lasted for years may suddenly take on the characters of ordinary mania. The passage from the melancholic stage to the maniacal may be sudden or slow, simple or variable; that is, it may appear and disappear, again to reappear. In many cases the patient appears to wake up to the folly of his depression, and thinks that now he is quite well and that he must make up for lost time. This leads to restlessness and over-action. The period of transition may be very short, or may itself need consideration; for it is at this time when we hope—too often in vain—to check an attack. In patients who have had previous attacks, similar if not identical symptoms recur in the same order, and we are inclined to hope that the process may be modified by early treatment. I am very doubtful if such attacks can thus be cut short.

The first sign of acute mania is generally some outrageous or violent act. The nature of the attack will vary greatly, according to the sex, age, and social position of the patient. As a rule there is degradation along all the social lines; the patient says things without regard to the feelings of others, and, later, without regard to social customs. There is a strongly marked exaggeration of self-feeling, a feeling of independence and a consequent intolerance of interference; next to the increase of self-feeling, there is almost invariably increase or perversion of sexual desire, leading to immoral or indecent acts; increased appetite often with well-marked degradation, so that blood, raw meat, or filth may be eaten; the tongue is coated, the breath foul, the bowels generally confined and the stools offensive. In some cases marked gastric disorder is present, the face may be flushed or sallow; as a rule the bodily weight decreases; there is no real increase of muscular power, and there may be marked tremor of the finer muscles, and lack of staying power; there is constant restlessness, the urine may be of high specific gravity with an excess of lithates, but it rarely contains either sugar or albumin. Sleep is very defective, there being often constant restless chatter day and night, till the mouth and tongue become dry, and speech difficult to understand. Dreams and nightmares, however, often pervade the restless sleep. The senses are often unduly acute and memory may be good, so that a maniacal patient will recall words which have been spoken to him when he was maniacal. In some cases, though the special senses seem to be acute, common sensibility may be reduced; at any rate patients will suffer injuries or inflict them on themselves without apparent feeling.

The destructiveness of maniacal patients may be aimless; their acts may be simply impulsive, depending on uncontrolled reflexes; but often the destructiveness has a constructive idea at its origin, the patient, wishing to have new clothes or clothes of a new pattern, unpicks his coat, but, lacking the power of sustained effort, he fails in reconstruction.

Hallucinations and delusions may occur at any period of the disorder, but as a rule they seem to occur first during the restless nights, when the bewildering thoughts seem to take shape, appear as foes or friends, and are addressed in suitable terms. Hallucinations of sight are very common, especially in cases depending on any form of toxaemia. Gastro-intestinal trouble gives rise to notions of poisoning.

Illusions are very common at night, when they are the cursory and mistaken interpretations of the sensory impressions. Delusions, more particularly having reference to the patient and to his friends, are common; at first the feeling of buoyancy leads to self-appreciation, and later to the notion of undue interference.

The symptoms depending on loss of control become more and more marked, till the patient is restless, sleepless, ungoverned, and incoherent; an organism which is swayed by every influence and impulse and beyond all self-control. Such a state in which thought follows and obscures thought, and in which no "ego" persists, may run on to exhaustion and to death; or it may slowly pass by, leaving the patient in a state of exhaustion more or less profound. He may then have passed from a stage of melancholia through one of mania to one of dementia, with a sudden, or slow, or progressive transition. As a rule, the amendment follows natural sleep, and may seem to result from some bodily crisis or disorder. The recovery may be rapid or slow, and it is important to recognise that in many cases there is great danger of relapse during the post-maniacal stage; premature return to home and work are therefore especially dangerous in these cases. An attack of mania rarely passes off under three or four months. In a woman two normal menstrual periods should be passed before return home; and no man who has had a severe attack of mania ought to resume business responsibilities till he has been well for six months. There may be perfect and enduring recovery; or the recovery may be partial, the patient being no longer able to fulfil his business or social duties. Among the latter results we meet with cases of moral as well as others of mental defect.

An acute attack of mania may leave a man incapable of directing his conduct, in one way or in many. Recovery may seem to be perfect, but instability remains so that the patient becomes liable to recurring attacks. In some cases one attack of mania leaves a patient demented, and incapable of ever again taking his place in the world. Death may occur from exhaustion; in which case restless, sleepless excitement leads to a delirious state in which it is impossible for the patient to rest or to take food; and the result is rapid emaciation and death. In some cases of mania the end is quite sudden, the patient in the midst of restless excitement dies suddenly; such cases occur in the anaemic, and in persons with diseased vessels, or with an alcoholic history.

Death may occur from suicide, or more often as the result of accident. Maniacal patients not infrequently sustain accidents and injure themselves, and yet are unaware of the injury, and cannot give a history of its cause.

Treatment.—In the treatment of acute mania, the need of an asylum is almost inevitable; for though it is possible, where money is no object, to treat a patient in a private house, yet in my experience the patients do not recover so rapidly as if sent to an asylum.

In the early or threatening stage, we must be guided by general principles; and experience shews that home and the most devoted friends are not to be looked upon as suitable ministers. I do not think foreign travel is to be advised in the earliest stage. I believe that change to some quiet, healthy, sleep-inducing spot is better, the man being removed from business, and the wife from husband and family. General tonic and hygienic treatment and hypnotics will have to be tried, and little or no harm follows the brief or occasional use of bromide and chloral.

I prefer giving great freedom in the open air, abundant milk food, and no stimulants; while the bowels are opened freely with calomel or saline purges. Hot baths are also useful. I have seen very good results from prolonged warm baths, the patient being kept in the bath for six or seven hours daily. There is no good in shaving the head and applying blisters. I prefer to give little in the way of sedatives, but if there be restless sleeplessness I give sulphonal, trional, or veronal in moderate doses; if there be violence and mere weakness I prefer paraldehyde in dram doses, every two hours for three doses. In young, active, and very violent patients hydrobromide of hyoscine (subcutaneously), beginning with $\frac{1}{200}$ of a grain each hour for three doses, is useful. Great freedom, abundant food, and no more sedatives than necessary, produce the best results, and these are best carried out in an asylum; as a rule alcohol should not be given.

G. H. Savage.

REFERENCES

1. Drewitt, W. H. "Acute Mania," *Cincin. Lancet-clinic*, 1895, N.S. xxxiv. 719.—2. Fronda, R. "Contributo allo studio clinico della mania ricorrente," *Atti d. xi. Cong. Med. Internat.*, 1894, Roma, 1895, iv. 197.—3. Givens, J. W. "Some Sugges-

tions as to the Treatment of Acute Mania," *Journ. Amer. Med. Assoc.*, Chicago, 1894, xxiii. 153.—4. Mann, E. C. "Mania transitoria," *Maryland Med. Journ.*, Baltimore, 1894-5, xxxii. 59.—5. Richardson, A. B. "Hysteria, Cerebral Manifestation," *Amer. Journ. Obst.*, N.Y., 1895, xxxi. 635; also *Cincin. Lancet-clinic*, 1895, N.S., xxxiv. 695.—6. Rohé, G. H. "Mania," *Internat. Clin.*, Phila., 1895, 4th Ser. iv. 145.—7. Walker, R. "Ueber manische und depressive Psychosen," *Arch. f. Psychiat.*, Berlin, 1907, xlii. 788.

G. H. S.

ACUTE DELIRIUM

Syn.—*Acute delirious mania; Bell's disease; Délire aigu; Delirium acutum; Delirio acuto; Delirium grave; Mania gravis; Typhomania; Phrenitis; Collapsdelirium.*

By R. Percy Smith, M.D., F.R.C.P.

Definition.—An acute mental disorder of rapid onset, with severe affection of mind of the delirious type, associated with febrile disturbance, profound bodily exhaustion and prostration, and tendency to the typhoid state; ending not uncommonly in coma and death.

Introduction.—The position of acute delirium among mental diseases has been a matter of considerable controversy; some observers have regarded it merely as a stage or variety of acute mania, and others as an autonomous affection or specific morbid entity, of toxic or infective origin.

Kraepelin includes it under the head of "Infektionsdelirien," the symptoms and course suggesting an infective origin, the source of which cannot be always ascertained. Anglade considers it to be due to the action of infectious processes still undefined upon a nervous system prepared by hereditary or acquired weakness. He separates it from mania, from febrile delirium, and acute or chronic intoxications. Bianchi considers that the old problem as to whether it is a morbid entity or only an episodic syndrome of various morbid states is still unsolved. He thinks that acute delirium being regarded as an expression of a grave intoxication there is no difficulty in conceiving that it may be sometimes a disease *per se* and sometimes a complication of other diseases. Drs. Savage and Goodall class it under the head of insanity due to toxic influences. Paton classifies it under the head of "Psychoses which are probably in part the result of auto-intoxication." Dr. Lewis Bruce considers that it is inseparable from other forms of "maniacal excitement with confusion," which he includes in a group of insanities in which there is evidence of the presence of bacterial toxins in the blood. He says "the physical symptoms alone point to the fact that the disease is the same whether it lasts for a fortnight or a lifetime, whether it recurs every month or six weeks or never recurs at all, whether it occurs in youth or old age, whether the symptoms are so acute as to deserve the

KING'S COLLEGE HOSPITAL
MEDICAL SCHOOL.

term delirious or so slight as to be called subacute." He regards the term typhoid or delirious mania as a misnomer, as all the toxic insanities, whether of metabolic or bacterial origin, may terminate in typhoid or delirious mania. Dr. Craig classifies acute delirious mania under the heading of "States of Excitement," but is inclined to regard it as a separate disease. Dr. Stoddart regards it as a phase of intermittent insanity in which all the characteristics of acute mania are excessive, and there are also physical signs of acute febrile disturbance.

It may be said that whilst various forms of insanity have come more and more to be looked upon as the expression of an intoxication either of metabolic or bacterial origin, and whilst there may be transitional forms between the febrile or post-febrile deliriums, insanities following exhaustion or intoxications, collapse delirium, and acute confusional insanity, yet the rapid onset, urgent symptoms, and grave prognosis in typical cases of acute delirium render its separate description and consideration necessary.

Etiology.—Neurotic or insane inheritance is commonly found. The disease more commonly affects women than men, and occurs most frequently between the ages of twenty-five and forty. Very often before the onset of the disease there has been debility for a long time arising from such causes as prolonged intellectual strain or anxiety or physical fatigue from any cause, accompanied by insufficient rest and sleep, and want of care in maintaining the bodily strength. Debility from exhausting disease may have the same result, especially if severe emotional stress be added. It occasionally follows the specific febrile diseases, pneumonia, erysipelas or influenza, or other exhausting illness, such as cystitis, diarrhoea, or diabetes. Further it may occur after confinement, or as the result of alcoholic excess, head injuries, heat-stroke or sunstroke.

The question as to the dependence of at least some cases of acute delirium upon intoxication the result of the action of a specific bacillus is still under discussion. Bianchi and Piccinino discovered a bacillus in the blood which they considered specific, but other observers have either failed to find Bianchi's bacillus or have found other micro-organisms, such as staphylococci, diplococci or streptococci, or have failed to find any. Bianchi considers that there may be a bacillary and grave form of the disease (delirium acutum), and a coccic form which is less grave (delirio sensoriale acuto). Dr. Lewis Bruce (3) isolated from the blood of a case of excitement with confusion a pure culture of a small coccus, and found that the serum of the patient in a dilution of 1 in 30 agglutinated this organism in less than an hour. He found, as the result of an extended series of observations in such cases that hyperleucocytosis existed, leading him to the belief that the diseases known as mania are due to bacterial poisoning, the point of attack being almost certainly the alimentary canal.

Morbid Anatomy.—The brain and meninges are usually found after death to be congested. The cortex of the brain appears somewhat swollen, and the grey matter darker than normal, with occasionally punctiform haemorrhages. The perivascular spaces are dilated, and contain abundant extravasated leucocytes, which sometimes cause a naked-eye appearance of white lines along the vessels. There is usually excess of subarachnoid fluid; but, in patients who have become comatose as the result of prolonged intense excitement, and who at the same time have not received sufficient fluid nourishment, there may be an almost complete absence of fluid from the brain.

Microscopically, the changes noticed are principally in the blood-vessels and perivascular spaces. In addition to extravasated leucocytes there may be red corpuscles. The small arteries are full of blood. Leucocytes may be in excess in the ground substance of the cortex, and in the pericellular spaces. Chromatolysis is observed in the cells of the cortex and other parts of the central nervous system, with displacement or disappearance of the nucleus and interruption of cell processes and axis-cylinder, the myelin breaking up into droplets or globules. The neuroglia is increased, especially around vessels and degenerating nerve-cells. There is usually hypostatic congestion of the lungs; the heart substance is pale and friable; and the skeletal muscles may shew evidence of degenerative changes analogous to those met with in enteric fever.

Symptoms.—Acute delirium may begin suddenly, but more commonly there is a short period of depression with irritability, sense of impending illness, sleeplessness, exhaustion, inability to concentrate the attention or to collect the thoughts, associated with discomfort, heat, or pain or confusion in the head. Hallucinations quickly appear, and are commonly terrifying and associated with an anxious expression of countenance. The emotional state varies, but is rarely exalted and commonly one of fear. Delusions are not always definitely formulated and change rapidly. Consciousness becomes profoundly affected, and the patient loses mental grasp of his environment, and passes into a condition of complete mental confusion with constant delirious chattering and loss of control over bladder and rectum. Sleep is absent or very deficient. There is extreme restlessness and excitement, with a tendency to resist and struggle against feeding, or any necessary attention; the effect of this being to produce profound exhaustion. If the disease be not checked by treatment, and the nutrition maintained by abundant feeding, the muscles waste, the fluids of the body become diminished in quantity, the tongue becomes dry and brown, the bowels confined, and the pulse feeble and irregular. Finally, the motor restlessness ends in twitchings and purposeless movements; complete mental apathy, attended by low muttering delirium, supervenes, and coma with eventual failure of pulse and respiration may close the scene. In cases which do not end fatally there is loss of memory of the attack.

The temperature is usually raised, but without definite course, or may be paradoxical; in most cases varying between normal and 103° F., but in others rising as high as 106° F. In another group of cases (delirium asthenicum) the temperature may not be elevated, or only for a short time at the outset of the disease, or when death is impending. Albumin

LIBRARY

is occasionally present in the urine, which is deficient in chlorides and hypertoxic. Bed-sores are apt to form rapidly. In cases of alcoholic origin peripheral neuritis may appear. Short remissions may occur.

Prognosis. — Always grave, and generally unfavourable to life. Patients who come under treatment early, and are not profoundly exhausted, may recover quickly; but, if early treatment be neglected, death may ensue by the rapid onset of coma or as the result of some local complication not unfrequently in the first fortnight. The prognosis is better in women than in men. Recovery takes place in some cases after a prolonged period of secondary stupor, when excitement has disappeared; but in others, though life may be preserved, there may be mental death, the patient remaining incurably demented.

Diagnosis.—From ordinary acute mania by the more rapid onset, the greater severity of the symptoms, the presence of fever, and the tendency to end fatally in a short time. From meningitis and other cerebral affections the disease is distinguished by the absence of cranial nerve paralyses, vomiting, optic neuritis, and convulsions. There is less tremor, more febrile disturbance, more intense excitement, greater prostration, and more profound affection of consciousness than in delirium tremens. Acute febrile diseases, with delirium in the early stages, are of course finally distinguished by the occurrence of a specific eruption.

Treatment.—Rest in bed in a quiet and somewhat darkened room, under the care of skilled nurses, and the removal of all sources of irritation, are necessary. In the milder cases of this affection, and where the means are ample, the patient may perhaps be treated at home or in a general hospital; but in the majority of cases removal to an asylum is absolutely necessary, and the earlier the better. Many patients are not removed till too late, and when irreparable damage has been done. In an ordinary house it is almost impossible to avoid the constant holding of the patient, which so rapidly exhausts his strength; and feeding is apt to be very imperfectly carried out. The patient should be liberally fed; principally with fluid nourishment in the form of milk, eggs, beef tea, plasmon, or peptonised meat. Should food be refused, as is often the case, nasal or oesophageal feeding must be resorted to, without hesitation and without delay; and should be done three or four times in the twenty-four hours. Stimulants are almost always indicated by feebleness of the pulse, and at times they induce sleep. Stimulants are often avoided on the notion that the case being one of "brain fever," harm will be caused by their administration; but many patients will die if stimulants be not given to them. Abundant lavage of the intestine is important as assisting in intestinal disinfection and supplying fluid to the tissues.

Sleep always needs attention, and if it does not follow feeding, or tepid sponging, must be produced by drugs. Chloral, bromide of potassium, opium, and hyoscine are better avoided. Paraldehyde is perhaps the best drug for this condition, and may be given by the nasal tube when the patient is fed. Chloralamide or sulphonal is, however, useful. The hypodermic injection of ergotin has been recommended. During convalescence tonics and liberal diet are necessary, and prolonged rest from work, in country surroundings and without excitement, is essential.

CONFUSIONAL INSANITY.—Synonyms: *Hallucinatory confusion*; *Amentia* (Meynert); *Akute Verwirrtheit.* This condition is closely related to acute delirious insanity. The name is applied to a form of acute mental disorder characterised intellectually by confusion of ideas, slow and incomplete thought-processes, slow reaction to questions, want of grasp of environment, failure to recognise persons or mistakes in their identity, failure to record new impressions, and hallucinations of various senses. Memory for the attack is usually defective. Definite formulated delusion may be absent, though in some cases present and arising out of hallucinations. The speech generally gives evidence of the confusion of mind, in that it is slow and fragmentary, and sentences are incomplete and incoherent. The patient, when roused, appears momentarily to wake up from a dazed condition, but relapses into it before any train of thought is completed. The condition differs from delusional stupor, as in the latter the patient is less responsive to questions and appears to be more profoundly stuporous, yet on recovery he is frequently found to have been fully conscious of his surroundings. In confusional insanity there appears to be, as a rule, neither pleasurable nor painful emotion, but a neutral state; though there is often resistance to feeding, dressing, and other attentions, partly due to delusion and partly to want of comprehension of their meaning.

The milder cases most nearly resemble the condition known as "acute primary dementia," or "anergic stupor"; the more severe ones approach the delirious type. The disease is, however, a very curable one, unless there be permanent organic disease.

It may result from acute exhausting diseases, or any cause leading to exhaustion, the puerperal state, sexual excesses, severe emotional shock, surgical operation or injury, or auto-intoxication.

The physical condition generally depends upon the cause, the patient always being weak and poorly nourished; and there is frequently a tendency to dryness of mucous membranes, and to the accumulation of sordes on the teeth, tongue, and lips. The temperature, however, is not raised.

In fatal cases changes similar to those described in Acute Delirium but of less intensity may be found.

The treatment consists in abundant nourishing diet, with stimulants in some cases, attention to the bowels, and the administration of iron and other tonics. Rest in bed is necessary in the more severe cases; but in the milder ones the patient is able to be up, and indeed derives benefit from gentle exercise in the open air.

R. PERCY SMITH.

REFERENCES

1. ANGLADE, D. Ballet's *Traité de pathologie mentale*, 1903.—2. BIANCHI, L. *Trattalo di Psichiatria.*—3. BRUCE, L. *Studies in Clinical Psychiatry*, 1906.—4. *Idem.* Morison Lectures "On the Etiology of Mania," *Brit. Med. Journ.*, 1908, i. 347.—5. CRAIG, M. *Psychological Medicine*, 1905.—6. KRAEPELIN. *Psychiatrie*, 7th ed., 1903.—7. ORR, D. "The Pathology of Acute Insanity," *Brain*, London, 1902, xxv. 340.—8. PATON, S. *Psychiatry*, 1905.—9. ROBERTSON, W. FORD. *Textbook of Pathology in relation to Mental Disorders*, 1900.—10. SAVAGE and GOODALL. *Insanity and Allied Neuroses*, 1907.—11. STODDART, W. H. B. *Mind and its Disorders*, 1908.

R. P. S.

TOXIC INSANITIES

By G. H. SAVAGE, M.D., F.R.C.P.

TOXIC insanity depends on poisons either derived from without or generated within the body. All these poisons, in interfering with the healthy nutrition of the highest nervous structures, act alike; and their effect depends on the nature and concentration of the poison, the frequency of its use, and the power of the individual to withstand its effects. We shall see that in some instances the poison, by its volume and by its concentration, annihilates all mental functions at once; this is familiarly shewn in poisoning by alcohol, and by various gases. In other cases the poison is retained in the body, and produces what are called "cumulative effects." The collection and concentrations of poisons in this way are well seen in the effects produced by lead and arsenic; and it is generally true that the persistence of the nervous symptoms is closely related to the stability of the poison, or to the directness of its effect on nervous tissue. Thus, lead is a more stable poison than alcohol, and produces much more persistent effects; whilst the poison of influenza appears to have a specific noxious action on the higher nervous tissues.

Some poisons are specially injurious to the nervous tissues, such poisons being selective in their specific action; on the other hand, some persons are especially sensitive to one or more of the poisons: certain conditions, again, tending to nervous degeneration, render some persons peculiarly susceptible to particular poisons. In my opinion neurotic persons are, as a class, more open to infectious influences; and the degenerating, such as those suffering from senile, general paralytic and similar brain changes, are readily affected by such poisons as alcohol and narcotics. It may be, too, that in the neurotic the poison of syphilis selects the nervous system for its manifestations. Poisons, whether introduced from without or generated within the body, may affect different parts of the nervous system, and these in different degrees; and consequently the resulting symptoms will differ enormously. Almost any form of mental (and nervous) disorder may arise from poisons; disorder extending from slight social incapacity to absolute and permanent dementia. Bianchi maintains that sensory insanity, that is organised delusional insanity, of various types, is due to endogenous intoxication and infection. He has found streptococci, staphylococci, and diplococci in the blood in these cases, particularly in the earlier stages. Probably, too, some poisons may arise from changes in the nervous tissues themselves, and I feel certain that some cases of acute delirious mania following on exposure which I have seen have had some such cause. Dr. Ford Robertson considers that the principal factors in the genesis of many acute and chronic mental disorders are various forms of gastro-intestinal infection.

There are several points of special interest which must be noticed before proceeding to other details. As a rule the poison is absorbed in small doses, and produces its first effects on the highest and most specialised parts of the nervous system. Loss of power of accommodation to special social conditions is seen earlier than loss of common mental faculty; at a later stage affection of the peripheral nervous system gives rise to various illusions and hallucinations of the senses, which may set up insanity with organised delusions; next, if its influence continue, the poison leads to permanent structural changes which weaken the will, the control of the emotions, and especially the recent memory. Another common characteristic peculiarity is the establishment of an organic need for the poisonous stimulant; a "habit" or craving which, in the case of the stronger poisons, can only be compared with lusts or irresistible passions. These desires lead to dishonesty and untruthfulness, which mark the strength of the poison and the weakness of the nervous system.

The poisons may act directly on the nervous tissues, affecting either the central or the peripheral parts first and chiefly; or the poisons may interfere with the general nutrition, and affect the nervous system mainly by altering the nature or amount of the blood-supply.

External poisons are chiefly absorbed through the respiratory or the digestive tract, though the skin or other surfaces may in some cases be the sources of infection.

Malaria, giving rise to ague and various intermittent and remittent fevers, may cause impairment of the nutrition of the nervous system; and though serious effects are not common in England, yet sleeplessness, neuralgia, mental depression, loss of power of application, doubt, suspicion, dread, fear of impending evil, are met with in such cases, and are relieved by such remedies as quinine and arsenic. I have met with acute mania and acute melancholia following ague; and permanent mental weakness, with some definite changes in the brain tissues, has been described as a result of prolonged exposure to malaria. Arrest of mental development of various degrees from idiocy to merely mental feebleness have been traced to malaria.

Influenza has a very marked effect on the nervous system. During the recent epidemics I have seen many forms of mental disorder following influenza, especially in predisposed subjects. Sleeplessness, neuralgia, neurasthenia, delirium, and peripheral neuritis are some of its earlier and simpler consequences; and though in the great majority of the cases these symptoms pass off, yet in a very large number actual insanity arises.

Influenza may give rise to insanity in highly neurotic subjects; it affects persons who have previously been insane; those who are at critical periods of life, such as that of childbirth or the menopause; and, still more commonly, those who from age, arterial degeneration, or alcoholism are already shewing signs of wear in their nervous systems. In comparatively few instances does the insanity follow the first attack of influenza; but the neurotic seem specially liable to frequent recurrences of the disease, recurrences which sooner or later produce serious injuries in the brain. Like alcohol, then, the influenzal poison disposes to fresh attacks, and tends to nervous degeneration.

Influenza may cause not only genuine insanity, but also diabetes, megrim, nervous deafness, epilepsy, various paralyses, and so forth. Though as a rule the mental symptoms following influenza are of the melancholic character, yet in some instances the febrile stage passes from delirium to acute delirious mania of a very grave kind. In most cases there is well-marked and distressing sleeplessness, accompanied or not by paroxysms of neuralgia; this sleeplessness is followed by waves of profound mental depression. In many instances the melancholia is simple; that is, without delusions; although the depth of despair and the paroxysmal nature of the symptoms frequently lead to suicide.

In many cases there are sensory disorders, and hallucinations are common; sensory defects, such as deafness and loss of taste and smell, are frequently met with. In patients who have already been insane, but who have been free from mental illness for many years, influenza may bring a return of their malady; and in chronic insanity recrudescences of mania or melancholia are common. Delusional insanity is frequently made worse, or started afresh.

Sometimes stupor succeeds the delirium, the epileptic fits, or the acute mental disorder which follows influenza. In some cases puerperal insanity begins after an influenzal attack. In a very few cases the febrile stage of influenza appears for a time to relieve the mental symptoms. Senile insanity of various forms may be precipitated by influenza.

In doubtful cases of general paralysis, influenza may clear up the diagnosis; thus, patients who have been losing some of their mental power, but who are not recognised to be suffering from this disease, after an attack of influenza become manifestly paralytic. In some cases of alcoholism influenza gives rise to marked symptoms of degeneration. Many patients are left changed, weakened, or aged by a nervous illness following influenza. Rest, sea travel, tonic treatment, and the free administration of quinine with some alcoholic stimulant are the most efficient means of cure.

Leledy of Bourges summed the matter up as follows:—Influenza may set up psychopathy. Insanity may come on at any stage of influenza; and this disease may set up any form of insanity. No specific symptoms result from influenza. The part played by influenza in the production of the insanity varies; it may take a nearer or a remoter place in the causation. In all cases there is acquired or inherited nervous instability. The insanity follows altered brain nutrition, possibly toxic. The onset of insanity is often sudden, and bears no relation to the severity of the influenza. The instability depends on general rather than on the special conditions The insane are less liable to take influenza than the sane (this is probably not true). Rarely, it has cured psychoses. There may be remission of symptoms during influenza. Influenza may lead to crimes and medico-legal issues. Bianchi writes "of the infective diseases influenza is certainly the most insidious as a cause of acute psychosis."

Insanity of various kinds has followed the **inhalation of poisonous gases.** The most common cases arise from exposure to *coal gas.* In these there is not infrequently complete insensibility, followed by a more or less pronounced state of stupor or acute dementia. In some cases the stupor is followed by a period of mental excitement of the hysterical or maniacal type; in my experience, it is more rare for mental depression or delusional insanity to arise.

In patients already degenerating, poisoning by coal gas may accelerate these changes, hastening the symptoms of general paralysis or those of senile dementia. Complete loss of memory of the accident, and of some interval after it, is common. Similar symptoms may follow poisoning by sewer air, if this be concentrated.

Carbonic acid gas poisoning leads to states of mental stupor, which may be very long, or even permanent.

Carbonic oxide has caused mental stupor with loss of consciousness and memory. In such cases very slow recovery takes place, and if the insensibility has been prolonged the recovery may only be partial.

Chloroform, ether, laughing gas, and other anaesthetics occasionally produce mental disorder, mania of hysterical character or stupor being the more common result. I have known one lady after chloroform, and another after "gas," behave as if they were drunk, a state which continued for some days (*vide* p. 921). In some neurotic patients the use of anaesthetics during labour seems to have contributed to subsequent insanity.

Enteric fever is water-borne as a rule, but the symptoms produced by it are like those following similar poisons borne in the air.

In any febrile state delirium may occur, and this is more likely to occur among the neurotic. Delirium may be the starting-point of acute mania; after the delirium, or after fevers apart from delirium, there may be marked evidence of mental weakness, as shewn by loss of recent memory, weakness of will, emotional weakness, and social incapacity. Moral insanity also may follow febrile disorders.

In some cases there is a direct connexion between the height of the fever, the degree of delirium, and the mental weakness; but in other cases no such relation exists. It may be mentioned that the joint-pains disappear with the delirium of hyperpyrexial rheumatic fever. I have met with mania of a severe delirious type occurring in pneumonia, and in several cases the crisis and rapid fall of temperature have been associated with acute mental disorder, generally characterised by hallucinations of sight and hearing. Such cases are very grave and require full stimulation.

Next we have to consider the **poisons which arise in the body** itself. Mental disorder may be the expression of disordered function of any of the excretory glands.

With *jaundice* depression is very common, and at times there are sensory hallucinations. I have myself met with only one (doubtful) case in which a jaundiced patient complained of seeing all things yellow. With hepatic trouble profound melancholia is not uncommon, and the interpretation of the miserable feeling will depend to some extent on the age, sex, and condition of the patient; the old man fears ruin, the middle-aged woman dreads dishonour. Gall-stones and their consequences are very common among the chronic insane in asylums, a proclivity dependent on their inactive life, good feeding, and torpid bowels.

With *albuminuria* there is a tendency in some patients to great irritability and to depression; and I have known suicides depend on this state. Worry and anxiety lead to kidney disease, and kidney disease often accentuates worry and depression. In some puerperal cases albumin may be present in the urine, but this is not a common occurrence. Eclamptic attacks, associated with albuminuria, occasionally give rise to mental disorder of various kinds. The convulsions of uraemia, if recovered from, may leave permanent mental weakness.

The relation of *excess of uric acid in the blood* to various neurotic states has been very fully studied by Dr. Haig. Though many authorities doubt his conclusions.

The old notions of suppressed gout and of gouty metastasis to the brain represent the fact that in some persons who are gouty, the cessation or arrest of a gouty attack may be associated with mental disorder, which is frequently melancholic. I have never met with greater mental misery than in such cases, and in them the tendency to suicide is very great. A return of the gout clears off all the melancholia. In some cases the suppressed gout is represented by hypochondriacal melancholia of a very harassing kind. Maniacal symptoms may come on before, during, or after an attack of gout; and the mania, as a rule, is violent and dangerous; the patients chatter without ceasing and rapidly exhaust themselves, and may die.

Furthermore, in such subjects epileptic seizures may occur, and lead to mental weakness and premature senility.

Sugar may be found in the urine of insane patients, and the present opinion is that this may occur as a passing symptom in some puerperal cases, and that it may be transient and in no way connected with the insanity. In certain other cases sugar may be present only during the insanity, and be related to the insane state, or it may occur only while the patient is sane and disappear while he is insane. True diabetes occurs in members of neurotic families, and it may alternate with other neuroses. Towards the close of diabetes, marked mental weakness may be evident; and some of the most extreme examples of insane hypochondriasis are met with in such patients. After the convulsions or coma of late diabetes marked dementia may come on. Sugar may be present in the urine of neurotic patients without producing any mental disorder.

Septicaemia produces a certain proportion of the cases of puerperal insanity, which generally arise within a few days of delivery; the thermometer must be the chief test of this, for acute delirious mania, which may also arise at the same time, is hardly to be distinguished from septic puerperal mania; but the temperature of the septic disease rises higher and is more variable than that of acute delirious mania. Septic puerperal insanity is generally maniacal in form, but it may be active melancholia or stupor. Though most of these cases are very grave, a certain number pass through the ordinary stages of the mental disease, and exhibit their septic nature only by some lung complication, or by some secondary abscesses. After severe injuries, such as railway smashes, I have met with a few instances of mental disorder which were in part attributable to septic troubles. Septic troubles may arise from large suppurating surfaces; and it is necessary to record the fact that iodoform applied as dressing to wounds may give rise to maniacal excitement, which rapidly passes off with removal of this dressing. There is nothing specific in iodoform mania; it may occur with the first dressing, or it may result from its prolonged use; it gives rise to restlessness, to sleeplessness, to irritability passing into mania; and the mania may rapidly give place to stupor or mental weakness. In some cases a degree of mental stupor follows the recovery from the acute disorder.

In a certain number of cases erysipelas precedes the outbreak of general paralysis, and the connexion between these diseases appears to be more than accidental.

Among the organic infections, syphilis has been already referred to (p. 904 and special articles).

Defective action of glands may so seriously affect the general nutrition as to cause insanity. Cretinism, myxoedema, and Addison's disease are examples of this; and I believe that in certain adolescents the imperfect action of ovaries and testicles give rise to defective general development. Such are certain conscientious, passionless youths with feeble circulation who seem unable to grow into vigorous social units.

Lastly, we have to consider the group of cases depending on poisons taken from without, usually through the mouth or skin.

Alcohol is the most important of these, and in England the increase of insanity is generally considered to vary directly as the increase of the consumption of alcohol. This subject is considered separately (p. 912), but I may sum up its effects thus: alcohol acts directly on the nervous tissues; it produces excitement, followed by exhaustion and decay; it affects neurotic more than non-neurotic persons; it affects more seriously the higher nervous systems of those who are already degenerating along their nervous lines; it acts most injuriously after injuries to the head and sunstroke in cases of general paralysis, and in senile degeneration. Immediately or remotely it may cause insanity. It may produce delirium tremens, delirium e potu, or acute delirious mania; and the delirium tremens may be prolonged, and even persist as chronic mania. It may produce sudden stupor, or more or less complete dementia. It may give rise to peripheral neuritis of various forms, which may start illusions, hallucinations, and delusions. The result of these sensory disorders may be one or other of the forms of systematised delusional insanity, persecution mania, insane jealousy, suspicions, paranoia, monomania of grandeur, and the like. It may give rise to epilepsy, and to its mental consequences. It may lead to defect of memory, or to insanity of doubt, due to loss of will-power. It may establish a habit of intemperance, chronic or recurring (dipsomania). It may assist in the production of general paralysis, and it gives rise to criminal tendencies and to crime; from the parent it may transmit to the child degeneration of mind, morals, or body. Dr. Mott has shewn that in the neurotic the nervous system suffers from alcoholic excess much more than the liver or kidneys. In fact, whilst in general hospitals, cirrhosis of the liver and kidneys is generally met with in alcoholic patients, this is not met with in alcoholic insane in asylums.

Idiocy, imbecility, epilepsy, neurosis, and crime are among the results of parental excess in alcohol.

I have met with a few instances of acute insanity following poisoning from *belladonna.* In these cases the delirium of belladonna poisoning passed directly into delirious mania. In all the cases there was strong neurotic inheritance. Atropine has in rare cases, even when dropped into the eye, set up temporary mental disorder.

Duboisine may set up mental disorder resembling that due to atropine.

To the effects of anaesthetics in producing insanity I have referred already. (*Vide* also p. 923.)

In England we have little experience of the poisoning produced by *Indian hemp* (Cannabis indica). (*Vide* article "Hasheesh Poisoning," Vol. II. Part I. p. 965.)

Pellagra, due to use of diseased maize, occurs in Italy; Greece, Russia, Egypt, and other countries in which maize is a common article of diet. (*Vide* art. "Pellagra," Vol. II. Part I. p. 892). It produces nervous prostration which may pass into any form of melancholia. There are two forms, acute and chronic; in both there is mental enfeeblement tending to dementia. Remissions are not infrequent, but unless prompt change of diet and surroundings are secured the tendency is to dementia and death.

Morphine is next to alcohol the poison most frequently regarded as a cause of insanity (see Vol. II. Part I. p. 946). In referring shortly to the subject here I would premise that as the neurotic are more sensitive to pain, and less tolerant of discomfort, they fly the more readily to sedatives, and suffer more from their use. Unfortunately, medical men are, of all persons, the most given to the abuse of morphine. It gives rise to insane habit (morphinomania), loss of moral sense, of truth, honesty, and all altruistic feelings. It gives rise to sensory illusions and hallucinations, which may originate various forms of delusional insanity, resembling those produced by alcohol. Suspicion, jealousy, fear of detectives are common in such patients. Visions of a terrific kind may occur by night or day. Distaste for food, with ideas of poisoning, may follow. Affections of common sensibility may set up ideas of unseen or electrical influence, hypnotism, spiritualism. Various defects of knowledge of time and space, somewhat similar in those occurring with alcohol, may appear. During the period of indulgence, or more often on the attempt to break off its use, mania or active melancholia of a very violent or suicidal type may arise. Vomiting may cause further exhaustion, and the free use of stimulants may be required. I believe in the sudden and complete withdrawal of the drug, and I do not believe in the gradual reduction. If the whole of the drug is withdrawn, and a full dose of sulphonal (or paraldehyde) is given, the patient being kept under chloroform for an hour till the milder sedative is at work, the worst will be got over, if the vomiting be not very severe. Rectal feeding and the administration of chloral hydrate by the bowel may be necessary.

Cocaine may produce acute delusional insanity in persons strongly disposed to its influence; but as a rule the hallucinations and ideas of persecution rapidly pass off. In my experience it produces hallucinations and delusions chiefly in those who take it in the hope of reducing the suffering while they are breaking off the morphine habit: in these cases the nervous system has already been seriously affected by the morphine, and the cocaine acts only as the exciting cause of the insanity. General systematised delusional insanity follows, which rapidly diminishes on the removal of the drug. In a few cases the nervous system has no power of recovery, and chronic delusions or chronic mental weakness results.

Sulphate of sparteine is used by some morphine takers, and atropine by others, to conceal their habit; and those alkaloids may assist in the nervous breakdown, and may colour the mental symptoms which arise.

Chloral hydrate, if taken in very large doses, may cause stuporose dementia or profound melancholia. In continual doses it leads to some moral perversion, and may give rise to delusions of persecution. It may start sensory illusions and hallucinations, but these are not very common. Stupidity, slowness of reaction, doubt, and suspicion are the most common symptoms produced by its prolonged use. In some instances the symptoms resemble those of general paralysis.

The use of *antipyrin* and *phenacetin* may become a habit, and even a craving, but I have not met with any cases of insanity following their use. With each addition to our list of sedatives and anodynes we shall increase the number of patients who suffer from their excessive use, but probably all the symptoms will be similar.

Lead, arsenic, and phosphorus may each give rise to forms of mental disorder. *Lead* appears to have a special power over the nervous system, and it may cause general or local nervous disorder. When taken by the mouth or absorbed through the skin, it may give rise to symptoms directly, or the result may follow an accumulation of the drug in the system. Acute mania may follow the taking of such a poison as plumbic acetate, or the absorption of lead from lotions. The mania may be of short duration, or having been set up by lead, may follow an ordinary course. Melancholia or stupor may result from the more slow absorption of the poison, but mental weakness is more common. The dementia of lead poisoning may be transient, or it may be part of a steady progress of degeneration leading to true general paralysis of the insane. General paralysis is started in a few cases by lead alone, or by lead and alcohol or lead and syphilis. Lead may give rise to peripheral neuritis and to palsies, which may be associated with delusions, illusions, or hallucinations chiefly connected with ideas of poisoning, persecution, and the like. Lead may cause epilepsy, and with the epilepsy there may be simple mental weakness; or any of the mental disorders associated with epilepsy may be met with.

Arsenic taking leads in some cases to a craving for it, and this may be associated with a loss of higher morality; but I have no experience of insanity due to the habitual use of arsenic. *Phosphorus*, however, may cause various forms of mental weakness, but I know of no special symptoms.

G. H. Savage.

REFERENCES

1. Bruce, L. C. *Studies in Clinical Psychiatry,* London, 1906.—2. Haig, A. *Uric Acid as a Factor in the Causation of Disease,* 7th edit., London, 1908.—3. Jacobsen, D. E. "Ueber Auto-Intoxications-Psychosen," *Allg. Ztschr. f. Psychiat.*, Berlin, 1894, li. 379.—4. Martel, C. *Contribution à l'étude de l'hystérie toxique,* Paris, 1894, p. 58.—5. Régis et Chevalier-Lavaure. "Des auto-intoxications dans les maladies mentales," *Semaine méd.*, Paris, 1893, xiii. 369.—6. Tatz, T. "Deux cas de folie hystérique d'origine infectieuse," *Ann. med.-psychol.*, Paris, 1895, ii. 376.—7. Urquhart, A. R. "On Insanity, with Special Reference to Heredity and Prognosis" (Morison Lectures), *Edin. Med. Journ.*, 1907, N.S., xxi. 204, 326.

G. H. S.

ALCOHOLIC INSANITY

By T. B. Hyslop, M.D.

In discussing alcohol as a factor in the causation of mental disease, there are several points of importance to be borne in mind. Some persons cannot maintain health and nourishment without the aid of alcohol, whilst others cannot tolerate its use even in small quantities. It may therefore be either beneficial or injurious. "One man's drink another man's poison" is not altogether a question of quality or quantity; much depends upon the peculiarities of the drinker. We have to consider—(*a*) the causes of alcoholism; (*b*) alcohol as a cause of insanity; and (*c*) the characteristics of insanity associated with alcoholism.

The **causes of alcoholism** are not infrequently overlooked in our estimation of cases of insanity. There may be a physical or a mental craving for alcohol, or a diminished power of resistance to its effects, the proclivity being the expression of an unstable inheritance; or, on the other hand, the nervous instability may manifest itself by a tendency to epilepsy, insanity, idiocy, imbecility, or other neurosis. This neuropathic diathesis, when due to alcohol, shews itself by an increase in mortality, diminution in the number of births, decrease of moral energy, and slowness in the rate of development of the intelligence. The majority of drinkers are disposed to drink by heredity. In 100 cases of alcoholic insanity admitted into Bethlem, no less than 67 had a neurotic inheritance; 32 had a family history of alcoholism: in 8 cases only was there a history of alcoholism without insanity in the parents. Alcoholism without insanity or other neurosis in the parents is more rarely followed by alcoholism in the offspring than when other neuroses in the parents are contingent factors. Alcoholism in the parents is, however, a very common factor in determining the occurrence of other neuroses in the offspring. Analysis of another series of 100 cases, in which intemperance in the parents was the sole ascertained cause, confirms the experience of Dr. Bevan Lewis that the alcoholism was almost exclusively limited to the father; in one case only was the mother addicted to drink.

The proclivity to drink may be manifested in several ways: (i.) it shews itself precociously, and the brain is more readily affected by alcohol; (ii.) alcohol may engender or determine a psychosis which is inherited, or reveal ideas or tendencies which normally were kept in subjection; (iii.) delirium tremens, mania transitoria, impulsiveness, tendencies to rash acts, and even epileptiform seizures manifest themselves under alcohol; (iv.) mental disorders may appear suddenly, due to causes other than alcohol; or sometimes even the sudden suppression of alcohol will determine an attack of insanity (*delirium e potu suspenso*); (v.) sensory perversions are more common, especially illusions and hallucinations of sight; (vi.) according to Legrain, suicidal attempts in these patients are planned and not impulsively carried out. This, however, is not invariable. In one case, marked amnesia, analgesia, and suicidal intentions carefully planned, were the main symptoms; the chief factor of causation being an hereditary disposition to drink. Further, in regard to heredity, there appears to be a greater tendency for the female children to be affected by insanity; they break down more readily, and from slighter causes, than the males. In a certain number of disposed persons a craving exists which may be quite independent of use of alcohol; thus, the heredity may be alcoholic and transmitted as a craving, or purely nervous, with alcohol as an accidental factor in the child as in the parent. I have seen three brothers who manifested the existence of hereditary cravings by the abuse of morphine, cocaine, and alcohol respectively.

The causes of drink-craving are therefore important, and the same causes may induce insanity or alcoholism, or both. Some conditions of exhaustion require alcoholic stimulation in order that the failing energies may be restored Out of 100 cases in which alcohol was an assigned factor of causation, in 11 only could it be said that alcohol was the sole cause. In all the other cases other factors were at work; such as syphilis, sexual excesses, bodily diseases, money losses, domestic griefs and worries, injuries to the head, intracranial diseases, or other conditions, which were either the causes of the habit, or at least as important factors as the alcohol itself in the causation of the insanity. In polyneuritic psychosis (Korsakow's disease), which is seven times commoner in women than in men, Dr. Mott finds that some infective condition, such as salpingitis, gonorrhoea, endometritis, syphilis, or pneumonia, is so often present that it is difficult to say how far the mental and neuritic symptoms are due to such causes, and not solely to the direct effect of alcohol on the nervous tissues. Syphilis, cranial injuries, sunstroke, malaria, and many other diseases render individuals very prone to suffer from the effects of alcohol; and in hot climates alcohol is more particularly dangerous to such persons.

Dr. Bevan Lewis finds that mental affections due to alcohol are far more prone to occur between the ages of twenty-five and thirty, and, again, from thirty-five to forty-five, than at other periods of life. The reason why the ages of from thirty to thirty-five are marked by special immunity is not explained. Children can directly inherit the tendency to drink; therefore it happens not infrequently that cases of genuine dipsomania occur at the early age of four or five years. Cohn, Madden, Moreau, and others have recorded cases of alcoholic delirium in children between the ages of five and eight years. Possibly alcoholism is more frequent in children than we at present imagine. When insanity supervenes the symptoms are nearly the same as in adults; but the prognosis is naturally more grave. In old age alcoholism may be symptomatic of brain decay; and the alcohol may tend to increase the failure of the mental faculties, and to hasten the cerebral degeneration.

Pathology.—The effects of alcohol upon the tissues and functions of the body generally have been described already (Vol. II. Part I. p. 901); and its effects upon the vascular and nervous tissues of the cerebrum have been dealt with by Dr. Mott (Vol. VII. p. 228), and so need not be reiterated here in detail. There is no constant or characteristic change in the brain as the result of alcoholism. The naked-eye and microscopical appearances in alcoholic pseudo-paralysis are quite unlike those of general paralysis of the insane. In alcoholic pseudo-paralysis there is not any disorganisation of Meynert's columns, and there is no evidence of lymphocytes or plasma-cells in the perivascular lymphatics. The only definite microscopical change is some neuroglial proliferation in the subpial and septal structure of the cortex, and replacement by it of the associational fibres in the tangential and supraradial layers. There is generally evidence of chromatolytic changes in the pyramidal cells and active proliferation of the young glial cells. In polyneuritic psychosis (Korsakow's disease) there is no infiltration of the perivascular sheaths and membranes with lymphocytes and plasma-cells. Some wasting of the tangential fibres, and some, but not advanced, subpial glial proliferation are found.

Symptoms.—The ordinary symptoms of alcoholism are too well known to need description (Vol. II. Part I. p. 922). The features of alcoholic insanity are varied and important both in a bodily and mental sense.

Motor disturbances are manifested chiefly by enfeeblement, incoordination, or paresis. Tremors, twitchings, spasms, or cramps may affect any of the muscular structures. The tongue, lips, eyelids, and fingers are usually tremulous; the limbs and even the intestinal viscera may be affected by muscular spasms; and the corrugators, orbiculares, scaleni, and sternomastoids are frequently affected by twitchings. Speech is often blurred, hesitating, and thick. The tongue may be not only tremulous or ataxic in its movements, but also protruded to one side. In acute cases, in which there is rise of temperature, refusal of food, sickness, and loss of weight and strength, the tongue may be furred, dry, and cracked. The speech resembles that of general paralysis, but in general paralysis improvement does not take place as in alcoholism. Similarly, the tongue tremors pass off more readily in alcoholic affections. Most drunkards display incessant movements and general restlessness; with also a characteristic tendency to impulsive violence, which may result in suicide or homicide. Epileptiform seizures may occur in varying degrees of severity, from mere dizziness, syncope, or *petit mal* to actual paralytic strokes. Hemiplegia and aphasia sometimes occur. Functional affections of the spinal cord are common, with abolition of the spinal reflexes. The knee-jerks may be absent, exaggerated, or unequal; and ankle-clonus may be present. The gait is sometimes ataxic or spastic. Impaired movement is often shewn by stolidity or loss of expression, or by asymmetry of the face. Inability to walk, with evidence of alcoholic neuritis, is much more frequent in women than in men (3 : 1), and occasionally there is loss of control over the sphincters.

The eye symptoms are of importance. Nystagmus has been observed rarely, and is apparently of doubtful significance. The pupils may be dilated or contracted, with or without failure of reaction to light and accommodation. Inequality is present in about 40 per cent of the cases; and irregularity of the margin is also comparatively frequent. Sometimes the cornea is duller in transparency, and the visual acuity may be diminished. Mere inequality of the pupils is not so significant as the absence of reaction to light. When there is failure of reaction to light, the pupils being either dilated or contracted, there is almost invariably a history of syphilis in addition to that of alcohol.

When bodily symptoms are present they require careful consideration. Alcohol alone seldom produces general paralysis; but not infrequently it so damages the brain tissues as to lead to a chronic delusional state, or to dementia. In cases with few or no physical signs the prognosis of the case will be based upon the progress of the mental state; and much will depend upon the restitution of memory, and upon the nature and persistence of hallucinations or delusions. The history of syphilis, sexual excess, stress and strain, both physical and mental, in addition to that of alcohol, renders the outlook more grave; and the case may end in stupor, dementia, or coma; or there may be persistence of hallucinations, aggravation of insane suspicions, moral perversions, suicide or homicide, or even the progressive paralysis of the insane. It must be noted that inequality of the pupils is not necessarily an unfavourable symptom, provided there be no history of syphilis or sexual excess. Inactivity of the dilated pupils to light, or dulness of cornea and inactivity of the contracted pupils, on the other hand, will probably prove unfavourable and portend the existence of a progressive lesion.

Much difficulty will be experienced in the diagnosis of the *pseudo-paralytic type*; and it is to be noted that in true progressive paralyses with remissions the pupillary changes may clear up much more readily than in the false forms. This, however, applies more to pupillary inequalities and irregularities than to conditions of reflex iridoplegia. In the false forms of paralysis the physical symptoms may follow epileptiform or apoplectiform seizures, or be consecutive to subacute alcoholism; but although the symptoms may be severe for the time they tend to improve rather than progress unfavourably.

The sensory perversions associated with alcoholism are numerous, and in many particulars characteristic. Sensations derived from the visceral system are frequent. Some patients complain of unusual feelings in the heart, as of fluttering or of "wind round the heart"; others derive their sensations from the lungs, stomach, or bowels. It is not, however, with the mere sensations that we have to do; the phenomena are almost invariably misinterpreted; and hence arises a grotesque and incomprehensible series of illusions and delusions. The illusions and delusions may take the form of poisoning, of magnetic or unseen agencies, or of torture of the viscera by internal agents, such as animals or devils. Most drunkards give a melancholic and suspicious interpretation of their feelings. Perversions of taste may arise in many ways, and are commonly associated with alcoholism. The perversion may take the form of a morbid exaggeration of all gustatory sensations, but much more frequently the sense of taste is diminished or absent. Parageusia is seen in nearly every form of insanity; and in alcoholism it usually takes the form of complaints that poison or filth is put in the food. Perversions of smell, especially when there is an olfactory stimulation proceeding from a disordered stomach, may take the form of offensive and foul odours, to which false objective significance is given. Perversions of sight are relatively more common than in other forms of insanity. The illusions or hallucinations are usually fleeting and terrifying. They are especially frequent in acute delirium tremens, and occur chiefly during the night time. Amblyopia and dyschromatopsia are also frequent. Galezowski found the chromatic anaesthesia chiefly pertaining to composite colours, especially yellowish- and bluish-greens. Auditory illusions and hallucinations are not so frequent in acute cases, but when there is a tendency to chronicity they become relatively more frequent. Perversions of tactile perception are very common, and the illusions and hallucinations arising in connexion with disordered cutaneous sensations are almost characteristic of alcoholics. In some cases hyperaesthesia is general, or confined to patches distributed indiscriminately over the cutaneous surface. The exalted sensibility may depend upon a too great sensitiveness to impressions of the sensory nerves, or a too acute perception by the nerve-centres of these impressions. This, however, does not tell us why patients suffering from tingling, numbness, burning, stinging, shocks, and the like, attribute their sensations to electricity, witchcraft, or mesmerism. Similarly conditions of diminished sensibility, either as local or general anaesthesia, or as hemianaesthesia, and the variations in the excitability of the nerves to pain and temperature can be partly explained from a physiological point of view; but we are quite in the dark as to the neural processes underlying normal perception; and there is as yet nothing to demonstrate the physiological or pathological reasons why the perceptive processes may be normal, and yet the interpretation of them so wide of the mark.

The outcome of persistent misinterpretation of sensory data is often disastrous, and patients of this sensory bent, from their common visceral or special sensory perversions usually pass into a chronic state of delusion. It must be remembered, however, that hallucinations of hearing and delusions of conspiracy and persecution occurring in alcoholic cases sometimes pass away, even when the patient has suffered from many previous attacks. Relapses are common, and the return of hallucinations may possibly be due to return of the disease of the same cells; in this case alcohol need not be the immediate cause of the relapse.

Delusions of grandeur and wealth occur in about 20 per cent of alcoholic cases. The conceit and optimism differ somewhat from the expansive benevolence of some forms of progressive paralytic dementia in that the delusions are characterised by fixedness and distrust in the drunkard, and are without the benevolence of the paralytic. No matter to what extent the drunkard carries his imagination, there is almost invariably a distrust and suspicion of those around him; and he is tenacious of his imaginary position and wealth.

Some cases are characterised from the first by dementia, and gradually the mental faculties are lost. Cases of this type are often deceptive, and extreme difficulty will be found in forming a prognosis. It is important to remember that alcoholics do recover from extreme depths of degradation; and slowly, step by step, regain the use of their faculties. According to some authors considerable amnesia is an unfavourable symptom; but even in these cases the memory is sometimes regained to an extraordinary extent, and the patient may be able to dispense with asylum care. It must be confessed, nevertheless, that many cases of continued amnesia do not end in recovery, but the patient remains in a condition of partial or complete dementia.

Not only do drunkards suffer from various degrees of amnesia, but they are also prone to be affected by various illusions of memory, termed "paramnesic" states. In these conditions the patient believes that his present circumstances, which are, in reality, new to him, have previously formed a part of his experience. Paramnesia occurs more commonly in alcoholic insanity than in any other form of mental disorder. A simple image may appear as a recollection. Thus, such patients with Korsakow's polyneuritic psychosis will give marvellous accounts of what they have seen and what they have done, although the accounts have no real foundation in fact. They state that they have just been out for a walk in the garden; whereas they have been kept constantly in bed. In some cases an illusion or hallucination may have been the initial factor in the production of the paramnesia; and the confusion has resulted from inability to distinguish between what was actually a false sensory perception and a perception founded upon an objective reality. When an illusion is revived, the fact that the primary vision was imaginary may be lost sight of, and the present revival appears to be based upon fact. In alcoholic cases the false memory usually refers to a visual image of persons or places seen, or to a motor or kinaesthetic image of actions preformed. It would appear reasonable to assume that the starting-points of the initial illusions or hallucinations were in the cells most immediately concerned with vision and kinaesthetic impressions respectively; but this would give no solution of the delusion as to the actual reality of the object seen or action performed.

Forms of Alcoholic Insanity.—It now only remains to mention briefly the chief forms assumed by alcoholic insanity.

(*a*) *Ordinary drunkenness* is attended by an increase in the frequency of the pulse and respiration. The arterioles become dilated, and there is, therefore, a lowering of the blood-pressure. The mental and moral functions are disturbed in various ways, and the higher forms of control are apt to be lost. The physical mechanism of speech and co-ordination of movements is also apt to suffer in varying degrees of intensity. Recovery usually takes place in the course of a few hours. Sometimes, however, death may occur from paralysis of the respiratory centre.

(*b*) *Mania a potu* is characterised by more intense excitement with more profound disturbances of the mental and motor functions. It occurs most commonly in those who have some congenital or neuropathic predisposition. The amount of alcohol necessary to produce the disturbances is often exceedingly small, and not in proportion to the effects it causes. In these cases recovery usually takes place in a few days; but in severer forms there may be apoplectiform symptoms, followed by coma and death, or tendencies to impulsive suicidal or homicidal acts. In some instances also there may be gross moral perversions, with consequent medico-legal difficulties.

(*c*) *Delirium tremens* occurs in the chronic alcoholic person, either as the result of a bout of alcohol or from some mental or physical shock. In most cases there is also profound disturbance of the digestive system and consequent auto-intoxication from defective metabolism. Most attacks begin during the night. The patient is restless, and disturbed by terrifying visions and fears of personal danger. Hallucinations of vision, hearing, and of common sensation are frequent; disorientation is usually present, and memory and cognition are profoundly disturbed. The physical signs are frequently very marked, there being in some instances a rise of temperature, furring of the tongue, constipation, and refusal of food. The urine is scanty, high-coloured, and sometimes contains albumin and casts. At first there may be contraction of the pupils, giving way later to dilatation. The superficial reflexes are usually diminished or absent. The knee-jerks are also usually diminished. By far the greater number recover; the few who do not recover either become chronically insane or die from cardiac failure or convulsions.

(*d*) *Subacute insanity* is characterised by mild melancholia or mania, with sleeplessness, dreams, nightmares, general restlessness, and hallucinations. It is generally of short duration, and with the return of sleep the patient recovers, usually within five or six days. Sometimes the symptoms resemble those of general paralysis of the insane, and to this type of case the term alcoholic pseudo-paresis has been given. The presence of epileptiform or apoplectiform convulsions, together with tremors of the tongue, face, and limbs, inequality of pupils, and alterations of the tendon-reflexes, may render the diagnosis difficult; but the pupillary light-reflex is seldom absent when alcohol is the sole cause. Sometimes the knee-jerk is absent owing to peripheral neuritis, and the symptoms may then simulate those of tabes dorsalis. In these cases recovery usually takes place in the course of a few months, and it is, therefore, essential that the condition should not be mistaken for general paralysis.

(*e*) *Polyneuritic psychosis* is characterised by mental disturbance associated with peripheral neuritis. The condition is sometimes known as "Korsakow's Syndrome." Korsakow described it first under the name of "psycho-polyneuritis" and later as "cerebropathia psychica toxaemica," and stated that its cardinal symptoms are irritable weak-mindedness, mental confusion, especially with regard to time and locality, and a form of amnesia in which memory for recent events is mainly affected, combined with peripheral neuritis. This syndrome has attracted much attention abroad, and has been investigated in this country by Drs. J. Turner and Ascherson.

(*f*) *Chronic hallucinatory insanity* due to the persistence of hallucinations of the special senses, and more commonly those of hearing and of cutaneous sensation. These hallucinations are followed by delusions of persecution.

(*g*) *Chronic alcoholic insanity*, which comprises the sensorial, motorial, amnesic, delusional, and demented forms already described.

Treatment.—For those who crave for alcohol preventive measures should be taken. The offspring of habitual drunkards should be removed from their vicious surroundings, and encouraged in some healthy occupation. The individual peculiarities of patients should always be well considered before prescribing alcohol, and it is also of great importance to ensure its discontinuance as soon as possible.

In acute cases of alcoholic insanity the diet should be nutritious and easily assimilated. Food should be given frequently, in small quantities. Some patients do well with food given at intervals of two or three hours.

Sleeplessness is a troublesome symptom, and it is often advisable to induce prolonged sleep by the use of hypnotics. The danger of hypnotics is that a craving for the hypnotic itself may take the place of that for alcohol. The bromides, chloral hydrate, sulphonal, paraldehyde, trional, veronal, chloralamide, or hyoscine may be given. Morphine should be avoided. Plenty of open-air exercise will sometimes induce sleep. Massage, the use of the dry pack and also of the wet pack, have in some instances proved beneficial. Many inebriates resort to so-called "specific" remedies, which unfortunately may be dangerous frauds. Institutions, therefore, which rely upon the efficacy of their proprietary "specifics" ought to be regarded with suspicion.

Suggestion during hypnosis is regarded as of considerable value in ridding a patient of the craving for alcohol. During hypnosis the patient is induced to believe that the desire for alcohol has left him, and that he even dislikes it. Sleep can also be induced by suggestion. In cases in which alcohol has been withheld, but in which its after-effects are still present, it is asserted by several eminent foreign observers that suggestion during hypnosis is capable of exercising a beneficial influence on various physiological processes, and that in this way it may prove a valuable ally.

Particulars as to Licensed Retreats and other details of the treatment of alcoholism will be found in the article on Alcoholism (Vol. II. Part I. pp. 901-937).

Theo. B. Hyslop.

REFERENCES

1. Andriezen. *Brain*, 1894, xvii. 569.—2. Ascherson, W. L. "On some Aspects of the Mental State in Alcoholism, with special reference to Korsakow's Disease," *Arch. Neurol. Path. Lab. Lond. County Asyl. Claybury*, London, 1907, iii. 483 (References).—3. Berkley. *Brain*, 1895, xviii. 473.—4. Binz. *Lectures on Pharmacology*, New Syd. Soc., 1895, 329.—5. Chapin. "Alcoholic Psychoses," *Rev. Neurol. and Psychiat.*, Edin., 1907, v. 172.—6. Clark, Campbell. *Mental Diseases.*—7. Cohn. *Berlin. klin. Wchnschr.*, 1888, xxv. 1043.—8. Hill, Alex. "Note on 'Thorns,'" *Brain*, 1897, xx. 133.—9. Korsakow. "Über eine besondere Form psychischer Störung kombiniert mit multipler Neuritis," *Arch. f. Psychiat.*, Berl., 1890, xxii. 609.—10. Kraepelin. *Lectures on Clinical Psychiatry.* Translated by Johnstone, 1904.—11. Legrain. "Alcoholic Insanity," Tuke's *Dictionary of Psych. Med.*, 1892, vol. i. p. 71.—12. Lewis, Bevan. *Mental Diseases.*—13. Moreau. *Ann. méd.-psychol.*, 1895, 8. sér., i. 337.—14. Morel. *Traité des dégénérescences humaines.* Paris, 1857.—15. Mott, F. W. "Alcohol and Insanity," *Arch. Neurol. Path. Lab. Lond. County Asyl. Claybury*, Lond., 1907, iii. 424.—16. Tamburini. *Riv. sper. di freniat.*, Reggio-Emilia, 1888, x.—17. Triboulet, Mathieu, et Mignot. *Traité de l'alcoolisme*, 1905.—18. Turner, J. "Twelve Cases of Psycho-polyneuritis in Women," *Journ. Med. Sc.*, Lond., 1903. xlix.—19. Wilks, S. "Alcoholic Paraplegia," *Lancet*, Lond., 1872, i. 326.

See also Huss. *Alcoholismus chronicus*, Stockholm, 1849-51. v. Magnan. *Alcoholism.* Translated by Greenfield, 1876. Marcet. *Chronic Alcoholic Intoxication*, 1862. Anstie. *Stimulants and Narcotics*, 1864. Masing. *Du rôle de l'alcool et des anesthétiques dans l'organisme*, Paris, 1860.

T. B. H.

INSANITY AND SURGICAL OPERATIONS

By Clinton T. Dent, M.C.

A few years since it was necessary to search closely through records of cases in order to find scattered reference to the occurrence of mental disturbance after surgical operations. Of late, however, the subject has attracted considerable attention, and it is now well recognised that insanity does sometimes, though rarely, follow as a direct sequel of the operation.

Etiology.—Without speculating on the precise causation, it may be noted that certain factors which may readily conduce to mental disturbance are present in any surgical operation of magnitude; and from the prophylactic as well as from the prognostic point of view, it is important to take these into account. Such are: the mental strain of anticipation; the actual operation, which may cause pain, bring about relief, or entail shock; and the mental reaction, even after an entirely successful operation. Among the possible after-effects, sepsis too must be reckoned. All these influences seem at first sight so likely to induce some degree of mental disturbance, that it is almost surprising that they are not more frequently the precursors of definite psychoses.

Surgical operations are now so common, and the public has come to regard the results as so uniformly good and devoid of risk, that the factor of anticipation has no doubt diminished of late years. Still it must be taken into account; for we are apt to forget that a patient can hardly be expected to steel himself to undergo an operation with the calmness that a surgeon can to perform it. As an instance of the factor of anticipation, Duplay cites the case of a man, aged seventy-four, in good health physically and mentally, who had a rather tight congenital phimosis. The prepuce was divided by an incision without an anaesthetic, and a small calculus removed. The man seemed to dread the operation, and made his will, and took analogous steps. On the fourth day following operation, mental symptoms devolved, and he became violent with delusions of persecution, and this was followed by melancholia. There was no mention of heredity in this case, but it is noted that he had albuminuria.

That insanity is a direct sequel of surgical operation is shewn by the remarkable fact that it occurs frequently in persons in whom there is no inherited liability to mental disorder. Making every allowance for the difficulty of obtaining a trustworthy history, the absence of any heredity in a large proportion of cases seems well established. In this respect the disorder has some affinity with instances of "Transitory Mania." It is true that instances have been recorded in patients who are the victims of marked heredity; and it has been asserted, chiefly by French surgeons, that there is almost uniformly such predisposition. With this view I cannot agree, for the absence of any hereditary taint, or even of any predisposition to psychosis, has been well established in a large proportion of the cases I have had the opportunity of seeing. In this country, at any rate, experience does not seem to bear out Duplay's rather sweeping dictum that "an operation is incapable of itself of causing insanity, and when it does occur there is predisposition." As cases in which undeniable heredity is present exhibit no special features, little reference will be made to them in this article.

The occurrence of a complication, such as insanity, after a particular case of operation, naturally leads to a search for instances of a similar character; and the conclusion is, on this account, apt to be too hastily drawn that the operation in question is one especially likely to be complicated with mania. More extended search tends, however, to confirm the conclusion that the nature of the operation has little to do with the matter. At one time it was supposed that operations involving the peritoneal cavity are more apt than others to be followed by insanity. Instances of insanity following operations on the female pelvic organs have been recorded; and it was at one time thought that operations of this class were particularly liable to be followed by psychoses. But the occurrence is certainly rare; and some surgeons who have had very extended experience in this particular branch of practice have never met with an instance of it. Mania has been known to follow operations of magnitude, such as amputations, excisions of joints, perineal section, ovariotomy, or hysterectomy: but it is recorded also after such slight surgical procedures as tenotomy and other minor procedures. The variety shews that no class of case is exempt, and none especially liable. As regards the nature of the operation, no one factor or combination of factors can be accountable. Instances of insanity have been noted after all the operations quoted, in none of which could the slightest tendency to heredity be traced. The removal of certain organs by operation is not infrequently followed by mental effects. As an obvious instance in point may be cited the mental changes in myxoedema following complete removal of the thyroid gland. Double castration, even when the testes are much diseased, may give rise to grave mental changes. Insanity has followed the removal of healthy testes—an operation at one time in vogue for the relief of enlargement of the prostate gland. But in the latter cases it is highly probable that other factors had to be taken into account, such as cystitis and secondary affections of the kidneys. For the purposes of this article, operations on organs having presumably an internal secretion may be excluded, as the discussion of the subject would lead us too far into speculative considerations.

Seeing that one form of puerperal insanity is held to be due to sepsis, and that surgical insanity has obvious points of affinity with that disorder, it is not surprising that some authors hold that insanity after an operation is due to failure to maintain an aseptic condition of the wound. It may seem bold in these days to assert that any sort of complication is unconnected with sepsis; but in my own opinion the insanity under consideration is commonly, if not always, quite independent of septic absorption. Insanity may occur where the wound heals absolutely by first intention throughout.

It is well known that certain drugs may produce insanity, as, for example, iodoform, morphine, belladonna, and eserine. Such drugs might be used either as surgical dressings, or in the after-treatment of a case: the toxaemia resulting from constipation produced by opium given in the after-treatment of an operation has been held to be the cause of insanity. But in many of the instances none of the agents commonly held accountable has been used at all; and the occurrence of mania with every variety of after-treatment shews that it may originate independently of toxic causes. Indeed, as already pointed out, the mania may occur while the wound heals absolutely by first intention, and when there can be little or no absorption of any drug, unless given by the mouth.

In all the cases there is a distinct period of quiescence after the operation, from the immediate effects of which the patient apparently recovers in a perfectly normal manner. The mania is thus to be distinguished from that produced by anaesthetics. In the latter instance the insanity is directly consequent on the operation, and the consciousness is affected or lost, possibly for weeks or months, from the time of the anaesthesia. The distinction between the two varieties of insanity is settled by the occurrence of mania after operations in which no anaesthetic was given.

The period of quiescence may vary from a few days to some weeks; the shortest period known to myself is two or three days, the longest eight weeks; the usual time being about one week. The complication may occur at any age, but it seems more prone than other forms of insanity to affect the young. I have seen well-marked instances in children of six, eight, and eleven years of age. My experience, therefore, differs from that of Hartmann, Broca, and others, who state that whilst they have frequently seen transitory delirium (*délires passagers*) they have never observed real psychoses in the young. But profound mental disturbance, associated with delusions and hallucinations lasting for many weeks, cannot be dismissed as a *délire passager*, and such a condition I have seen in several instances.

Symptoms.—The onset is insidious and gradual. Usually in children, and frequently in the case of adults, the first symptoms noted are fretfulness and a change of manner. The child conceives a dislike for the nurse or the mother. In young patients the disorder may not progress beyond this point, and in a few days or weeks the symptom passes off. The young often exhibit a repugnance to those nearly related to them which may amount to a horror; and older patients may exhibit entire indifference to, or even hatred of, those for whom they previously had much affection. This distressing feature disappears on recovery. The patients often become garrulous; they are usually dirty; in the young this is almost invariably the case. Sometimes they are restless, disturbing their dressings; but as a rule they pay little attention to the wound.

The temperature is usually raised, with a mean from 100° to 101° F. If the mania becomes chronic, the temperature may become subnormal. The increased temperature seems independent of the wound. Any surgical procedure necessary during the progress is, as a rule, well borne. If the wound is doing badly, and the diseased part can be wholly removed, the further operation will be more likely to benefit than harm the mental condition. If an anaesthetic be desirable, it is well to substitute one different from that originally employed. Generally speaking, when the patient is in a state of mania, chloroform answers best. If the wound progress satisfactorily, the patient's appetite continuing good, and the weight keeping up, recovery, both of mind and body, may be looked for with some certainty.

Acute mania is not very common, though it may occur. The chronic and the milder forms are most frequently seen. Delusions and hallucinations are frequent; in my experience the patients frequently become suspicious. The hair occasionally becomes coarse and stiff; and a return of the hair to its normal condition is often an early indication of recovery from the mania. The skin appears to shew little change. Anaesthesia or hyperaesthesia is not commonly seen; but occasionally, as in delirium, there is insensitiveness to pain in the wound. Though the mental condition of itself does not appear to exercise any injurious influence on the progress of the wound, if the operation has been a grave one, and the mania is acute, death may occur; but even in cases which end fatally the wound may progress in a perfectly normal manner.

As regards the **prognosis**, good hopes may be entertained of ultimate and perfect recovery if the mania be not very acute and the patient otherwise healthy. On the other hand, if the patient be the subject of grave disorder, and his constitution broken down by alcoholism, renal disease, syphilis, or the like, the mania is likely to persist and to run on into chronic dementia. Recovery, when once the mental convalescence begins, is tolerably rapid. In the young, recovery is almost invariable.

Throughout, the mental and the surgical aspects of the case can be considered rather apart, for the former affects the latter but little. The possibility, however, that some causes of irritation are present as the result of operation, leading to what is known as reflex insanity, must be borne in mind. A buried drainage-tube, or the irritation of an unabsorbed ligature, might be the persistent cause of mental disturbance, and recovery might depend on the removal of the foreign body.

On several occasions I have operated a second time on patients who have had attacks of this surgical insanity, without any repetition of the complication.

The stages of recovery are the same as in other forms of mania. The leading symptoms abate, delusions and hallucinations subside, and the patients have no recollection of events that happened during the period of insanity; as they improve further they become to some extent conscious of their own condition, and aware that something is wrong with them, and finally they recover their faculties sufficiently to realise fully that they have been out of their minds. But there is likely to be complete amnesia as to events and as to the lapse of time; and usually there will be no more memory of occurrences during the period of mania than there is in cases of concussion during the period of unconsciousness that follows the head injury. The amnesia, however, is not, so to speak, retrospective, as happens occasionally in concussion, in which the patient may have complete and permanent loss of memory of events that occurred some time previous to the actual injury.

The **treatment** is simple, and may be briefly dismissed. The great point is to feed the patient well. So long as the appetite continues good the case is hopeful. Drugs, such as iodoform, had better be discontinued if insanity occurs. Aseptic methods are now so universally followed that it is almost unnecessary to point out that the use of antiseptic agents, especially for washing or irrigating wounds, should be given up in favour of sterilised unirritating fluids. Insanity following operations is an unusual complication, and aseptic surgery is likely to make it still rarer. Hyoscyamus or hyoscyamine answers well in procuring sleep in many cases. Bromides are not to be recommended, as they tend to depress the patient. The dirty habits of the patients, while the insanity lasts, render them liable to bed-sores, and especial care must be taken to prevent this complication.

C. T. DENT.

REFERENCES

1. BATIGNE, P. "Prédisposition dans la genèse des troubles psychiques après les opérations gynécologiques," *Gaz. des hôp.*, Paris, 1899, lxxii. 573.—2. CHAGNON. "Psychical Troubles after Operation," *Montreal Med. Journ.*, 1898, xxvii. 958.—3. CROOM, J. H. "Psychoses following Pelvi-abdominal Operations," *Trans. Edin. Obst. Soc.*, 1900-01, xxvi. 98.—4. DENT, C. T. "Insanity following Surgical Operations," *Journ. Ment. Sc.*, London, 1889, xxxv. 1.—5. DUPLAY. "Folie post-opératoire," *Presse méd.*, Paris, 1899, i. 305.—6. ENGELHARDT, G. "Postoperativen Seelenstörungen," *Deutsche Ztschr. f. Chir.*, Leipz., 1900, lviii. 46, 194.—7. GARRATT, A. H. "Surgical Insanity following Simple Fracture," *Canad. Pract. and Rev.*, Toronto, 1908, xxxiii. 289.—8. GREKOW, J. "Postoperativen Psychosen," *Jahresb. f. Chir.*, 1901, 75.—9. KALLIONZIS. "Psychose post-opératoire," *Bull. et mém. Soc. de chir. de Par.*, 1908, xxxiv. 874.—10. LAPINSKI, M. "Psychosen nach Augenoperationen," *Allg. Ztschr. f. Psychiat.*, Berl., 1906, lxiii. 665.—11. MACPHAIL, S. R. "Postoperative Insanity," *Brit. Med. Journ.*, 1899, ii. 781.—12. MARCOPOULOS. "Troubles psychiques post-opératoires," *Bull. et mém. Soc. de chir. de Par.*, 1902, n.s. xxviii. 463.—13. PAGENSTECHER. "Delirium nervosum Dupuytren und Operationspsychosen," *Deutsche med. Wchnschr.*, 1900, xxvi. 591.—14. PICQUÉ. "Délire psychique post-opératoire," *Ann. méd.-psychol.*, 1898, lvi. 91.—15. PICQUÉ et BRIAND. "Psychoses opératoires," *Arch. de neurol.*, Paris, 1903, xv. 209.—16. *Idem.* "Psychoses post-opératoires," *Ann. méd.-psychol.*, 1898, lvi. 249.—17. PILCZ. "Postoperatives Irresein," *Wien. klin. Wchnschr.*, 1902, xv. 908.—18. SELBERGS. "Postoperative Psychosen," *Beitr. z. klin. Chir.*, Tübingen, 1904, xliv. 73.—19. URBACH, P "Psychosen nach Operationen am Gallengangsystem," *Wien. klin. Wchnschr.*, 1907, xx. 1465.

C. T. D.

THE EPOCHAL INSANITIES

By T. S. Clouston, M.D., F.R.C.P.Ed.

Attacks of mental disease are often determined by the great physiological epochs and "crises" of life. Certain remoter or more immediate causes act with especial force at such epochs. When an attack of insanity is so determined, its mental and bodily symptoms and accompaniments are influenced in such a way at each epoch that the cases have certain common characters, and may properly be classified accordingly. It is of the greatest interest and importance for the practising physician to look at his cases from this point of view; for it often leads him to found his treatment on physiological lines, to be more precise in his prognosis than he could otherwise be, and in many cases to take valuable measures for future mental safety after recovery. For the ordinary man or woman to pass with impunity of body and mind through the various epochs of life is perhaps rare; for the neurotic and the psychopathic to do so is still more uncommon. An attack of insanity forms the acme of the dangers incurred, and is the worst of all the risks, unless it be death itself; indeed by many men death would be faced more calmly. One of the chief points of interest in studying the epochal insanities is the opportunity it gives us of comparing the symptoms present with the normal physiological and psychological characteristics of the various periods of life; and of observing the degradation of physiological into pathological characters. That each epoch of life has its proper psychology is attested throughout our literature. Poetry, the drama, and fiction have all found their themes in the various thoughts, feelings, and actions characteristic of youth, of maturity, and of old age. Psychiatry, too, finds some of its most subtle and interesting studies in the mental and bodily symptoms, and in the prognosis and treatment of the insanities of puberty and adolescence, of pregnancy, of the puerperium, of lactation, and of the climacteric period and old age; for these are the chief epochal mental disturbances. It must be clearly understood that not all these are forms of insanity in a definite sense, as is general paralysis, for example; it is often impossible from the symptoms present in any one case for us to say definitely which form we have before us; and in mental and bodily symptoms, intensity, duration, and curability, individual cases of the same epochal form may also vary greatly. Yet in all these respects, as we shall see if we take 100 cases of each variety, there will be found so many characters in common as abundantly to justify this mode of classifying insanity, for which we are chiefly indebted to Skae of Edinburgh. For the physician to derive the full benefit from a study of the epochal psychoses he must correlate them with the epochal neuroses, and the other bodily diseases that are commonly called "critical."

THE INSANITY OF PUBERTY AND ADOLESCENCE (*Dementia precox*).—The normal process of the development of the human brain cortex in all its inconceivably delicate, subtle, and complicated functions, and the gradual establishment of the normal relationship of those functions to each other, are as "unthinkable" as the connexion of thought and organisation. Though our present knowledge in regard to these processes is very elementary, and very indefinite, certain facts are patent enough: the most definite of these is that the entering of sex and reproductive energy from the potential into the actual state at puberty accentuates and transforms the higher activities of body and mind. It also brings in a liability to diseases unknown before. A very little study of this all-pervading function and its effects shews us that this great process of vital transformation does not complete its work suddenly; it occupies, on the average, all the twelve years from puberty till full development about twenty-five. All this time steady progress is being made in acquiring new capacities, and new responses to feelings from within and to impressions from without. These impressions may come from the opposite sex, objectively or subjectively; from ambition; from morality; from religion; from art; from literature, indeed from the whole circumstances and conditions of life. An orderly progression of developmental changes in the emotions, the imagination, and the volition is seen, as time goes on, from the first onset of reproductive energy up to its completion and decay. In such an article as this, the normal physiology and psychology of puberty and its continuation in adolescence—this term being used to indicate the period from puberty till the fulness of bodily and mental development—cannot be entered into. The fact about adolescence that most concerns the physician is that it is a period full of the possibilities and the probabilities of arrested, perverted, and postponed mental and moral developments, and of the risks of diseases of many kinds, especially of nervous diseases. These are soon perceived to be to a larger extent the result of hereditary tendencies than the same class of diseases at later ages. Indeed evil heredity in certain respects then first comes into its fullest play, and decreases in its effects thereafter.

The insanity of adolescence and that of puberty are essentially the same. It is the most hereditary of all forms of mental disease; bad mental and nervous conditions being in fact traceable in ancestry and collaterals in twice the proportion of ordinary forms of insanity. In my personal experience I have rarely found any case, in which the family history was well known or accessible to me, in which there was not a record of mental disease, or one of the graver neuroses, in one or other of the patient's four grandparents, their collaterals or descendants. In 25 per cent of the whole, in which I could not find any such case of definite disease, I usually found the stock on both sides to be a highly "nervous" one; and sometimes I ascertained that for two or three preceding generations such neurotic stocks had intermarried, and so accentuated the nervous instability. As to the special symptoms and character of this form of insanity, I found, from a careful analysis of 320 cases, that in about 80 per cent of them the mental symptoms were those of mania; mostly of an active, lively, aggressive, motor character, tinged often with erotic, hysterical, or pugnacious ideas. The other 20 per cent of the cases were mostly melancholic in character, seldom being deeply suicidal, but often tending to be stuporose. In fact stupor, both in its melancholic and anergic varieties, is found much more frequently during the age of adolescence than at any other period of life. Whether maniacal, melancholic, or stuporose, one very marked character in the mental condition is the tendency of the symptoms to periodicity, remission, and relapse. This is common at all ages more or less; but in adolescent insanity it is especially frequent, few cases being free from it altogether. In different cases there would be a week, or a month, or two months of excitement, and then a remission, with a relapse in a few weeks. I explain this tendency as being the representative and pathological equivalent of the sexual and reproductive periodicity. As to the exact year or years of the twelve which cover the adolescent epoch, I find that the early years from fourteen to sixteen were very free from insanity; though they yield abundance of other developmental neuroses in the shape of chorea, somnambulism, and epilepsy. The next four years, from sixteen to twenty, produced a larger proportion of insanity than the earlier years, but still few compared with rather later years. It was in the next five years, from the twentieth to the twenty-fifth, that the great majority—68 per cent—of my adolescent cases occurred. These facts correspond with the physiological facts of brain growth and development. During its period of rapid growth in the womb, the great teratological defects and malformations and congenital idiocy arise. During the second period, from birth up to puberty, we have a long series of defects and diseases, from rickets and convulsions to certain forms of idiocy and epilepsy. During the adolescent period, we have in the earlier years, from fourteen to twenty while the motor centres are reaching perfection, epilepsy, hysteria, ugliness, and, as we have seen, little insanity; whilst in its later years, when the mental cortex and its functions, the crown of the whole organism, are reaching perfection, the great breakdown of insanity is most frequent. This coincides with the age when, in a normal organism free from neurotic heredity, perfect fecundity, the typical form and characters of man and woman, the moral qualities of self-control and altruism, and the intellectual and imaginative faculties are all approaching their full development. In persons who become insane, nature, unable to complete her work, attempts after her fashion thus to kill off a bad stock. A careful study of this period shews that in other hereditarily neurotic cases, adolescent psychoses of a more partial kind are apt to occur; such as stupidity, lethargy, indolence, unsocial practices, affective aversions, "incompatibilities" of temper, frothy religionism, sudden immoralities, perverted sexual acts, outrageous unconventionalities, dipsomania, and crime.

In the *treatment* of adolescent insanity we must take physiological considerations into account. To get healthy young men and women, sound-minded, self-controlled, moral, non-hysterical, muscular, and fairly fat, we provide during the whole period of development plenty of fresh air, muscular exercise, good non-stimulating food, little or no alcoholic drink, suitable employments, reasonable amusements, and usually also some outside control. Now these are the principles of treating adolescent insanity. We give milk, eggs, farinaceous diet, fruits, vegetables, and the lighter kinds of animal diet rather than too much stimulating animal foods. We want sound general organic development and not over-stimulation, for the neurotic and those hereditarily disposed to insanity have often a great difficulty in controlling and regulating their sexual and reproductive instincts during adolescence. Masturbation is common among them, and in them especially becomes dominant and hurtful. It is one of the complications of adolescent insanity. I give cod-liver oil also and the hypophosphites; and I try to keep up or increase the bodily weight during the attack. The patient should live in the fresh air, should do gardening, walking, and outdoor games. Under such suitable treatment about 60 per cent of the patients recover; but, unfortunately, in after-life half of these patients have relapses. Before perfect mental recovery takes place the signs of full organic development are usually seen; the form expands, the voice changes, the beard and sexual hair grow, the weight increases, and in women the mammae enlarge. Few cases end in death during the attacks; but unfortunately about 30 per cent sink into "terminal dementia," which constitutes the great mass of the incurable perennial insanity of our asylums. In these cases nature determines that the unfit shall not live, but the death is mental instead of bodily.

Within the past ten years the term *dementia precox* has been invented in Germany, and has come to be largely used there and in America as covering a considerable part of my "Adolescent Insanity." It has been divided by Kraepelin into the "paranoid," the "katatonic," and the "hebephrenic" varieties. It unquestionably represents a clinical type, but my objections to it are four, viz.: (1) that it is not founded as adolescent insanity is on a distinct and recognised physiological epoch; (2) that many of the cases are described as curable, and the word "dementia" should never be used to indicate a form of mental disease that can be recovered from; (3) that in the descriptions of *dementia precox* by the recognised authorities the great and characteristic symptom of periodicity is not given its proper place; and (4) that its so-called varieties often pass into each other, and cannot therefore properly be called even clinical entities. It has no pretence to any pathological basis (*vide* also p. 1006).

REFERENCES

1. Baillarger. "Accès de mélancolie débutant à l'époque des règles," *Ann. méd.-psychol.*, Paris, 1882, 6^e s., vii. 416.—2. Ball. "La Folie menstruelle," *Ann. de psychiat. et d'hypnot.*, Paris, 1892, iii.—2*a*. Campbell. "Modern Conception of Dementia Praecox," *Rev. Neurol. and Psychiat.*, Edin., 1909, vii. 623.—3. Clouston. *Clin. Lectures on Mental Diseases*, Lond., 1896; "The Neuroses of

Development, Puberty, and Adolescence medico-psychologically considered," *Edin. Med. Journ.*, 1881, xxvi. 5.—4. Hecker. "Die Hebephrenie," *Virchows Arch.*, 1871, lii. 394.—5. Jung. *Über die Psychologie der Dementia praecox*, Halle, 1907.—6. Kraepelin. "Dementia praecox," *Psychiatrie*, 1904, ii. Kap. v.—7. Lewis, Bevan. *Textbook of Mental Diseases*, 1899.—8. Skae and Clouston. Morison Lectures, *Journ. Ment. Sc.*, Lond., 1874, xix. 340, 491; 1875, xx. 1, 200; 1876, xxi. 1, 188.—9. Trömner. "Das Jugendirresein (Dementia precox)," *Samml. zwang. Abhandl. a. d. Geb. d. Nerven- und Geisteskr.*, 1900, iii.

The Insanity of Pregnancy.—Mental changes are common in pregnant women; insanity is very rare. The psychological changes that might almost be called "normal" in the pregnant woman commonly affect the emotions, appetites, and instincts. Sometimes very serious morbid cravings arise—for alcohol and opium, for example, or for unusual or indigestible and hurtful foods; but nature commonly shields the pregnant woman from actual insanity. Yet there are a few women who cannot become pregnant without becoming insane; these represent the acme of the tendency towards mental change. Then there are a few who are subject to the disease in special circumstances. The typical group of symptoms in the insanity of a pregnant woman are stupor, apathy, depression, conscious loss of affection for husband or children, and suicidal feelings; these may become very strong and dangerous, and should always be kept in mind as possible risks. In a very few cases I have met with acute mania; but this only occurs in those that have become very thin and weak, or who have been reduced by some severe bodily disease. The *treatment* must be largely expectant, with as few drugs as possible. Some patients recover before delivery, but this is rare; we have to wait till the confinement is over before the majority of these insane pregnant women throw off their morbid mental symptoms. Some of the patients who do not recover sink rapidly into complete dementia. There is usually a strong heredity in this form of mental disturbance, and the disease occurs most frequently in first pregnancies. I think that if the disease occurs before the fourth month of pregnancy the bringing on of abortion, after due medical consultation and with the sanction of the responsible relations, would be the proper remedy. This can now be done with little risk; and I think the woman is entitled to the extra chance this course would give her of regaining mental soundness, and of avoiding the greater risk of the mental death of dementia.

Puerperal Insanity.—No proximate causes of mental disease are, in certain cases, more definite than the physiological conditions of pregnancy, childbirth, and lactation. Childbirth especially stands out as bringing on mental disturbance of a severe character in 1 case out of every 400 labours; and as constituting 5 per cent of all the insanity in women sent to mental hospitals. Taken together, these three forms of insanity account for 10 per cent of all the asylum insanity in women. There is a certain uniformity in the symptoms of all three, especially in those of puerperal insanity; they are all very curable, and, as a medical man is usually in attendance on the patient at these times, it is most important that the early symptoms of the insanity then prone to occur should be recognised and treated; for many of the slighter cases, especially among the well-to-do classes of society, can be treated at home, and the attacks arrested at the outset. It adds much to the family distress if, at these times when grief drives away joy, the stricken parent has to be removed from home for treatment. The name "Puerperal Insanity" is technically limited to the mental disturbances that occur within the first six weeks after confinement. But as by far the majority of the cases—four-fifths of them—occur within the first fortnight after confinement, and one-half of them within a week, we commonly have to do with the bodily effects of the recent puerperium, mixed up with and complicating the treatment of the mental disease. We are not yet in a position to say decidedly what is the immediate cause of the very intense cortical disturbance that we commonly find. In some cases this is unquestionably direct sepsis from the uterus and passages; in others there is no direct evidence of this. The reflex disturbances of the brain from the womb, the sudden change in the requirements of blood-supply after delivery, the numerous new organic and functional accommodations that have to be suddenly made between the blood-forming glands, the womb, and the mammae, and the new and intense emotions aroused in the woman by pregnancy, childbirth, and nursing, may all upset the cortical working in those predisposed to mental disturbance. When compared with other forms of insanity, heredity is found to exist to about the usual extent. This is on the whole the most acute of all the forms of insanity, and the mean temperature I found to be higher than in that of any other form. The risk of death, too, is greater than in any of the curable forms of insanity, for over 8 per cent of the patients die during the attack. Every patient in whom the temperature rises above 103° F. runs grave risk.

The *symptoms*, in a well-marked case, are commonly as follows:—They come on very suddenly and run up to a high point of intensity within a very few days, sometimes within a few hours. The new mother, and the disease is most frequent in primiparae, changes in facial expression, looking self-absorbed and dull. At the most altruistic of all times of life she becomes egoistic; the time when the life of another human being absolutely depends on the mother, and the time of the intensest emotion seen in nature, that of a mother towards her sucking child, becomes a time of neglect, and even, in some cases, of homicidal impulse towards her helpless offspring. The mother does not notice the child, or ask for it. She does not readily answer questions; she does not eat; she does not sleep. She becomes restless, incoherent in speech, full of fancies, and expresses foolish dislikes to those about her; she soon begins to chatter or to scream, and in no long time her mental condition is that of an acute delirium. She needs to be held in bed, and exhibits great strength and great tendency to violence. The suicidal impulse is present in 40 per cent of the cases, and must be thought of and guarded against most carefully;

the nurses being warned of their responsibility. The bodily signs by this time are well marked; the temperature has risen to 100°, or, in the septic cases, even to 105° F.; the eyes are brilliant and feverish-looking; the lochia have stopped, sometimes becoming septic before disappearing; the pulse is small and thready; the face is haggard, though perhaps flushed; the skin is moist and clammy; the lips and tongue soon get dry. There are in fact combined together an intense mental disturbance, a great motor perturbation, and an organic state of dangerous exhaustion. Such a combination means much danger to life, and requires all the suitable nursing and treatment that can be instantly applied. The ways and means of keeping up the patient's strength have first to be faced; for usually she will not herself willingly eat or drink anything, urgently as she needs support. Liquid but strong nourishment, in any suitable form, we give every hour; such as liquid custards, strong soups, and alcoholic stimulants which I order freely; and I unhesitatingly feed the patient by the nose-tube if she cannot be got to take enough nourishment and stimulants otherwise. A custard containing two eggs, some sugar, and two glasses of wine, sometimes proves the best hypnotic and sedative. If the temperature be above 99° F. I always give quinine in from 5 to 15 grain doses, frequently repeated. The effect of this in reducing the temperature is in some cases instant. It is a good thing to keep washing out the vagina by antiseptic douches. If no sleep is got after the first night, it is well to try two drams of paraldehyde; but I am not in favour of the continuous or repeated exhibition of hypnotics or sedatives. Opium in any shape or form I avoid, because it tends to dry up still further the mucous membranes already too parched, and to accentuate the existing dislike to food. If called in at the stage when there is mere sleeplessness without excitement, I should give 20 grains of sulphonal. A never-ceasing supervision, plenty of nurses and constant warmth are absolutely necessary to give the patient the best chance. We expect some improvement to begin in a week or two, and recovery to take place within three months. A few patients recover at once, and completely, as soon as the initial period of fever and intense excitement has passed off; but in others, on the contrary, the disease runs on as subacute mania for six or even twelve months. When recovery has taken place it is usually complete and abiding; but the patient should have a change of scene before resuming her family duties, and she should not be exposed to the risk of another pregnancy for a considerable time. I always feel more sure of the recovery being complete when menstruation has returned. All the cases are not of the acute form I have described; a few are only mildly maniacal, a few delusional, a few melancholic, and a few confused and stuporose. These forms occur most frequently among the few patients in whom the disease comes on from the third to the sixth week after confinement. They need *treatment* on the same general principles as the acuter cases. My experience is that from 75 to 80 per cent of the patients recover. As might be expected, moral and mental causes may assist the direct effects of the puerperium in causing the disease. The disproportionate amount of the disease that occurs among primiparae whose offspring is illegitimate shews this fact very well.

Lactational Insanity.—The poor and hard-working are subject to mental upset during nursing in much larger numbers than the well-off. Four per cent of my female pauper patients are cases of lactational insanity; very little over 1 per cent of my richer patients are due to this cause. This throws much light on the causation of the disease; it means an exhausted anaemic brain without the means of treatment, without rest, and without the possibility of giving up suckling. It is much more frequently seen in mothers who have had many children in quick succession than in primiparae. The symptoms are far less acute in their character than in puerperal insanity. They are usually ushered in by the bodily symptoms of headaches, lassitude, neuralgia, giddiness or faintness; every one of which points to an exhausted anaemic brain. Then come sleeplessness and loss of interest in things; then depression of mind, which may soon pass into a subacute irritable maniacal condition. The mental control is lost, delusions come on, or suicide is attempted. The symptoms are rarely intense. The *treatment* of lactational insanity is that of brain anaemia. Immediately its preliminary or first symptoms appear, suckling should be stopped; the patient should have much fresh air, and a change. She should take iron, quinine, malt liquors, cod-liver oil, and good food. Recovery may be expected in 80 per cent of the cases in about three months. Recovery is always accompanied by a great increase in body weight, a better colour of skin, and a restoration of the number of red blood-corpuscles. Menstruation should not be stimulated or encouraged until the strength and fatness return. Very few of the patients die from the immediate effects of the disease; but tuberculosis must be looked on as a possible risk, for it occurs in a few of the cases. The nearer to the puerperium the first symptoms come on, the more acute and maniacal they are apt to be; the further away they are, the more subacute and melancholic in character will they prove.

REFERENCES

1. Aschaffenburg. "Über die klinischen Formen der Wochenbettspsychosen," *Allg. Ztschr. f. Psychiat.*, Berlin, 1901, lviii. 337.—2. Clark, Campbell. *Lancet*, 1883, ii. 97, 180, 277; and *Journ. Ment. Sc.*, 1887, xxxiii. 169, 372, 487.—3. Clouston. *Clin. Lectures on Mental Diseases*, 1904.—4. Fürstner, H. "Über Schwangerschafts- und Puerperalpsychosen," *Arch. f. Psychiat.*, Berlin, 1875, v. 505.—5. Lewis, Bevan. *Textbook of Mental Diseases*, 1899.—6. Macleod. *Brit. Med. Journ.*, 1886, ii. 239, 392.—7. Ripping. *Die Geistesstörungen der Schwangeren, Wöchnerinnen und Säugenden*, 1877.—8. Savage, G. H. *Insanity*, 1884.—9. Tuke, Batty. *Edin. Med. Journ.*, 1864-5, x. 1013.—10. Wigglesworth. *Liverpool Med.-chir. Journ.*, 1886, vi. 349.

Climacteric Insanity.—At the climacteric in both sexes there is in certain persons a special liability to nervous and mental breakdown. This is probably a partial representation of the physiological fact in nature that when the power to reproduce ceases, the whole vital energy is lessened, and begins to shew signs of decay. Reproductive death is, in fact, the first presage of actual death. In many forms of lower life the act of reproduction marks the end of the life of the individual. In the human being there is a normal psychology of the climacteric, and there is an accentuated and abnormal psychology which is called climacteric insanity. The normal mental changes consist chiefly of a lessening of the sexual desire and all that is implied therein; the affections largely change from the mate to the progeny, the imagination loses its former force and fire, poetry and action alike are less craved for. The instinctive feeling of sexual difference is lessened. There is less energy and less spontaneity and originality. Along with all these mental changes, the bodily changes, especially in women, of form, of the constitution of the blood, of gland and of trophic action, are coincident. These climacteric changes, mental and bodily, are seldom sudden; like adolescence, they cover several years. The moment of the menopause in woman is something of an accident, and is a mere signal flag announcing great physiological changes. The course of a case of climacteric insanity in a woman is usually this:—After experiencing many of the usual sensory neuroses that accompany the climacteric, such as giddiness, flushings, flashes of light before the eyes, or unusual feelings in the head, the woman, after, coincident with, or before the menopause, begins to feel less active in mind and body. Her courage, her spontaneity, her power of resisting and rising above the small worries of life, lessen. She often feels irritable. Then come groundless fears, conscious depression of spirits, want of interest in life and in her affairs, and lessened social instincts; meeting with her fellow-creatures is a strain rather than a pleasure to her; to live is no longer in itself an enjoyment. There are also usually well-marked bodily symptoms. The most common of these are loss of freshness of complexion, of weight, of appetite, and of digestive power. The skin looks muddy and pigmented, the bowels get costive, and the sleep is shortened or disturbed. In a few cases there is a sort of accentuated but irregular sexual excitation; or there are delusions as to the other sex, as, for instance, that some one is in love with her; or there are delusions of pregnancy; but all these cases are exceptional. The next stage is one of more pronounced mental disease. The morbid fears become formulated delusions of self-blame, suspicion, and fear. She affirms that something dreadful is certainly going to happen to herself, or to her husband or children. She has suicidal feelings, but they are not always intense. She becomes restless, cannot settle to work, moans and weeps. She rapidly loses weight, the alimentary secretion dries up, and there is troublesome constipation; the appetite is not only lost, but food is repugnant and refused; and there are often trophic affections and skin irritations. A stage of distinct toxic insanity comes, the source of this being alimentary. The melancholia becomes a markedly confusional state. This state may pass into the most excited melancholia, leading to exhaustion of strength, and death in spite of treatment; or the symptoms may all pass off gradually, the woman becoming for a time more healthy and strong than she had been for years before. I found that, out of 228 cases, 57 per cent of the female cases ended in recovery, whilst only 31 per cent of the males did so. There was a high proportion, but a low intensity of suicidal impulse. As to the age at which the disease occurred, about an equal number of cases in women occurred from forty to forty-five as from forty-five to fifty; whilst the male climacteric cases occurred chiefly from fifty-five to sixty-five. The chief difference between the female and the male climacteric case is that in the man the symptoms are more complicated with the lesser mental and bodily signs of senility. His "grand climacteric" is a less definite vital epoch. He feels the *taedium vitae*, he cannot work, he cannot enjoy life, he cannot settle to anything, and he tends to be hypochondriacal. Climacteric insanity is apt to last longer than most other forms, the average duration of treatment in the asylum cases of recovery being in 50 per cent less than three months; in 65 per cent less than six months; and in 91 per cent less than 12 months. At all the more advanced ages, both women and men, the number of recoveries diminished. There were some cases of mania among the many of melancholia.

The *treatment* of climacteric insanity is that of melancholia in general, but with special reference to an organism that has "turned the corner" of life. In the early stages I have found change of scene, with freedom from worry, and life in the fresh air especially effective. Careful dieting, frequent meals of stimulating as well as nourishing food, wines and malt liquors used dietetically, the natural mineral waters, quinine, iron, strychnine, the hypophosphites, arsenic, and other tonics are serviceable. A few cases in both sexes end in recovery after many years of melancholy and even of delusion.

REFERENCES

1. Berger. "Die Psychosen des Klimakteriums," *Monatsschr. f. Psychiat. u. Neurol.*, 1907, xxii. Ergänzungsheft, 13.—2. Christian. *Étude sur la mélancolie*, 1876.—3. Clouston. *Clin. Lectures on Mental Diseases*, 1904.—4. Conklin. "Climacteric Insanity," *Amer. Journ. Ment. Sc.*, Phila., 1871, lxii. 365.—5. Dreyfus. *Die Melancholie, ein Zustandsbild des manisch-depressiven Irreseins*, Jena, 1907.—6. Gages. *De la ménopause et de son influence dans la production de l'aberration mentale*, 1876.—7. Gaupp. "Die Depressionszustände des höheren Lebensalters," *München. med. Wchnschr.*, 1905, lii. 1531, 32.—8. Kraepelin. *Psychiatrie*, 1904, ii. Kap. viii.—9. Lewis, Bevan. *Textbook of Mental Diseases*, Lond., 1899.—10. Skae and Clouston. *Journ. Ment. Sc.*, Lond., 1874, xix. 340, 492; 1875, xx. 1, 200; 1876, xxi. 1, 188.—11. Tilt. *The Change of Life*, Edin., 1882.

Senile Insanity.—In old age the gradual decay and dissolution of energy and faculty extend especially to the functions of the brain cortex. Motor energy, speech, co-ordination, sensibility common and special, trophic power, and mind are all affected. The grey and white matter then shrink in bulk; cells and fibres gradually undergo demonstrable dissolution. Then vessels change in their coats and in their capacity to carry the normal blood-current, and to adapt themselves to the varying conditions of the intracranial pressure. Arteriosclerosis becomes marked and dangerous in its extent. Commonly the memory for names is the first mental faculty to fail, and then every faculty slowly diminishes in power. It frequently happens that we observe mind failure to take different courses in different individuals; in one memory, in another volition, in another emotion, and in another moral sense being the first to give way. The mental extinction of senile dotage must be considered a normal stage in the life of man, as much as his birth and his manhood. Looking to the immeasurable capacities of the brain cortex, to the strains it has frequently to bear, and to the manner in which bad heredity specially affects it, there is little wonder that in some cases the recession of mental function is not on physiological lines, but diverges into pathological states. It is not surprising that an organ, the normal action of which is so largely paroxysmal and spasmodic, and is so intensely sensitive to its proper stimuli from without and from within, should exhibit mental explosions in its decadence if it contains the seeds of hereditary weakness, or undergoes local irritations from gross lesions such as softenings or atrophies. Or, looking to the fact that the higher mental part of the cortex is by far the most complex product of evolution, it is scarcely surprising that in certain cases this product should fail earlier in life than the coarser elements of the brain and body; and that in consequence we should have mindlessness before complete organic decay and death take place. The organ that thus fails in its work contains within it representations of every other organ; its chief duty being to harmonise the action of them all for the welfare of the general organism. Every time any organ was active, some part of the brain cortex was active too. Every time an organ suffered it suffered also. These are the conditions which commonly end in senile dementia; but not always, as we shall see. This is not a simple and uniform type of insanity; far from this, there are few of the clinical forms of insanity that present such an immense variety of feature. Some of the cases are melancholic, some of them are maniacal, some are enfeebled, some are delusional, and some few are even immoral and impulsive in general mental character. The two most common types are, first, the occurrence of maniacal excitement with certain senile features, tending in a longer or shorter time to pass into dementia—usually indeed having in it from the beginning some of the elements of dementia; secondly, a type in which melancholic symptoms are the first mental disturbances, and in which there is more hope of recovery, partial or complete, and less chance of passage into dementia. This last results often enough from some disturbance from without or within, of a brain whose power of resistance is becoming impaired in early senility.

I shall shortly sketch the chief features of these two senile types of mental disturbance. A man over seventy, commonly over seventy-five, who has been looking old for some years and whose articulation has been losing its crispness and finer co-ordinations, begins to fail in his sleep. He soon gets restless both at night and by day. He becomes morbidly suspicious about small matters. He cannot carry out a fixed purpose.

He is very forgetful, and perversely obstinate. Sometimes his moral sense fails him and he speaks and acts in immoral ways, entirely contrary to the tenor of his past life. I have known such persons take to cheating, to lying, to deception of all sorts, and to sexual immorality of a scandalous kind. His regard for convention becomes less, and he often thus shocks his family and friends. In these respects, as pointed out by Dr. Savage, the brain in its process of senile dissolution loses first the qualities that in the process of evolution had come last into existence. The next stage of the case is one in which the most troublesome symptoms are sleeplessness and restlessness and sometimes noise at night, oftentimes with dirty and degraded habits, and even great violence and destructiveness. This nightly mania is, of all the symptoms, the most troublesome to manage; and often compels asylum treatment in spite of the greatest natural reluctance to adopt it in such aged persons. The brain exhibits, in fact, its normal nightly lowering of action in an extreme and pathological degree. It is interesting to compare this senile nocturnal mania of the brain, thus undergoing dissolution, with the common night delirium of the undeveloped brain in the fevered child of neurotic constitution. During the day, especially in the early stages of his attack, the restless, noisy, and entirely unreasonable man of the night before is often enough quiet, and, in a sort of automatic way, coherent and reasonable. He is sometimes provokingly so, for it is difficult to make relatives who have not seen his condition believe he has been so ill during the night. In a few cases we see grandiose delusions of wealth and power simulating typical general paralysis. Gradually the symptoms of general mental enfeeblement come to prevail over those of brain excitement, and if the patient do not become exhausted, or die, he lives on as a senile dotard, often restless and troublesome at night, till a paralytic shock, or heart failure, or an attack of bronchitis brings life to an end. And in connexion with paralytic shocks I have often seen a slight attack of this sort stop the maniacal symptoms, and produce a quiet, manageable, paralytic dotage. This form of senile mania is the most frequent one, but it is by no means so general as might be imagined. I was myself surprised to find that only about 60 per cent of my asylum cases of "senile insanity" were of this kind. The others largely consisted of cases of melancholia; some of them of great intensity, and many of these suicidal. Of this class 30 per cent ended in recovery. Certain persons in old age, with "senile hearts," atheromatous vessels, and poor nutrition of body, are apt to become unduly depressed when any febrile illness comes on, or anything goes wrong with them. The curable cases of senile melancholia usually occur at the earlier ages, from sixty-five to seventy-five. Some of them present a very curious series of symptoms of what might be called "automatic motor misery." The facial expression of persons of this class is one of deep depression; they weep, they groan, they wring their hands, and any ordinary onlooker would regard them as profoundly miserable; but all this will pass off in a moment from no outward cause and the patients will recollect nothing of it. They would seem not really to have felt the depression in any proper sense. The motor machinery of grief had been in operation, but the sensory and perceptive apparatus was not in action, or had disappeared by senile dissolution. Many of the "recoveries" are of course not such in an absolute sense, but only represent a recovery from the symptoms of excitement or depression, and transition into a quiet normal dotage. In a few cases vivid hallucinations of sight and hearing, and in others convulsions and epilepsy complicate senile insanity. No doubt the pathological explanation of this is that destructive lesions of an irritative character, whether gross or microscopic, are occurring in the sensory or motor centres of the cortex. Metchnikoff's bacterial conception of old age has yet to stand this test of proof, but many of the facts of senile insanity would fit in well with his hypothesis.

The *treatment* of senile insanity is often a difficult problem, because it implies not merely the giving of drugs, but careful dieting, unceasing watchfulness, and a most difficult combination of rest and exercise in the fresh air. A good nurse, skilled and patient, is the first requisite. Change of scene or travel is of course out of the question; the old do best with old scenes and ways. Home or an asylum is commonly our only choice; warm and airy apartments, sunshine if it can be got, warm clothing by day and night, easily masticated and easily digested non-stimulating foods, of which warm milk and eggs should form a considerable part, are to be provided. Cod-liver oil works wonders in some of the thin anaemic patients. Night feeding is commonly advisable. To combat the dirty habits of the senile insane is often most trying work to the nurses. As to sedatives and hypnotics, my own experience is strongly in favour of sulphonal and paraldehyde judiciously given and carefully watched. I have undoubtedly tided more mild senile cases of the restless type through their attacks by the use of sulphonal in doses of 10 grains and upwards combined sometimes with bromide of potassium in 20 grain doses than by any other drug. Tonics and the hypophosphites are often very useful. A general watchfulness over the bowels and other organic functions by the nurse is always very necessary. Sometimes cardiac tonics are useful. Let the senile patient rest and sleep when he is inclined to, no matter what time of the day it is.

We are gradually building up a rational pathology of senile insanity, and we may say the same of normal senility. In about one-half of the cases, gross lesions, such as "softenings" of the brain and arterial disease, are found. Endarteritis and periarteritis are both now found to be very common, though in many cases microscopic sections are necessary for their demonstration. By these means Dr. W. F. Robertson, pathologist to the Scottish Asylums' laboratory, has demonstrated such disease in the majority of senile cases he examined. In nearly all these cases, and in the others in which no such gross lesions are found, there are microscopic atrophies and pigmentary and granular degeneration of the large motor cells, the protoplasm of which has wasted and changed in character, having undergone chromatolysis, and the processes of which have largely disappeared. In some of the acutely maniacal cases the membranes are thickened, and the vessels altered in their coats, in somewhat the same way as we find in cases of typical general paralysis, or alcoholic insanity; and as in these diseases, spider-cells may abound in the outer layers of the cortex. Many patients in whom such lesions as these last are found have taken too much alcohol throughout their previous lives. In nearly all of them there is found more or less general atrophy of the convolutions and shrinkage in bulk of the whole brain. The whole subject of the mental and motor symptoms of senility is a most interesting one and well worth scientific study in the light of recent brain physiology and histology. Shakespeare in *King Lear* has given us an incomparable representation of mental senility.

REFERENCES

1. Alzheimer. "Die Seelenstörungen auf arteriosclerotischer Grundlage," *Ztschr. f. Psychiat.*, Berl., 1902, lix. 695.—2. *Idem.* "Neuere Arbeiten über die Dementia senilis und die auf atheromatöser Gefässerkrankung basierenden Gehirnkrankheiten," *Monatsschr. f. Psychiat. u. Neurol.*, Berl., 1898, iii. 101.—3. Buchholz. "Über die Geistesstörungen bei Arteriosclerose und ihre Beziehungen zu den psychischen Erkrankungen des Seniums" (contains full bibliography), *Arch. f. Psychiat.*, Berl., 1905, xxxix. 499.—4. Clouston. *Clin. Lectures on Mental Diseases*, 1904.—5. Hammond. *Influence of Age upon the Mind.*—6. Robertson, W. F. *Edin. Med. Journ.*, 1896, xli. 613.—7. Metchnikoff on *The Prolongation of Life*, 1907.—8. Savage. "Case of Senile Insanity," *Journ. Ment. Sc.*, 1883, xxix. 231.—9. Zingerle. "Über Geistesstörungen im Greisenalter," *Jahrb. f. Psychiat.*, 1899, xviii.

Insanity of Phthisis

The question of the attribution of such mental disturbances as can properly be included under the term insanity to diathetic and toxic causes is one of great interest. It may be admitted that to be definitely insane a patient must have had in most cases an original tendency in this direction. A family history of neurosis, in some degree or other, is so common, that for every case of mental disease there must be 10 in which the weak spot, if one there be, never gives way. If some defect of structure be inherited at all, it may resist destructive influences of low intensity; or such influences may be altogether avoided. To shew how the working of the higher cortex of the brain is affected reflexly and directly by disease we need go no further than the manifest facts, now more and more recognised, of the physiognomy and psychology of many ordinary diseases; such, for example, as cardiac and visceral complaints. The mental symptom of the *spes phthisica* in consumption is a very old and certainly a true psychological observation; and this is not the only mental symptom of phthisis. Many authors of repute had long ago noticed the frequency of deaths from tuberculosis among the insane, and it is found that two-thirds of the deaths among idiots result from tuberculosis. As a matter of fact, pulmonary consumption is found three times as frequently as a cause of death among the insane as among the sane of the same ages; and the graver, the more prolonged, and more incurable the form of insanity the higher is the death-rate from this cause. About one-third of the chronic cases of complete dementia end in this form of death. This may mean that, from some cause or other, the condition which makes the lungs a fit nidus for the propagation of the tubercle bacillus exists in thrice the strength in the insane as in the sane; though it is not to be forgotten that the propagation of tuberculosis is favoured by the congregation of the insane often in overcrowded rooms, and by their greater carelessness in expectoration. Now, the chief of the constitutional conditions is probably a general debasement of the nutrition of the tissues and organs of the body, especially perhaps of the lungs. This may mean that certain nervous centres, governing nutrition, have been weakened in action. But beyond these suppositions certain clinical observations, made by me in 1863, induced me in that year to go into a careful clinical and statistical investigation of the whole relationship of tuberculosis to insanity, when I came to the conclusion that a closer and more interesting affinity exists between these two diseases than the high rate of phthisical mortality among the insane. My chief conclusions were that in certain families there is a close hereditary kinship between phthisis and insanity; and especially that there is a form of insanity so distinct in mental symptoms, and, clinically, so characteristic of pulmonary tuberculosis, that it may be called "phthisical insanity." The chief facts that led me to this conclusion were—(i.) That in a certain number of the insane the two diseases appear simultaneously, or, at all events, within a year or two of each other; (ii.) that in a large number of these there is a family history both of insanity and consumption; and (iii.) that in these cases a certain distinctive group of mental and bodily symptoms is almost always present. The knowledge of these facts has often enabled me to diagnose phthisical insanity by the mental symptoms alone, before the appearance of any symptoms in the lungs. This form of insanity cannot be said to arise from long-continued mental disease, or from the hygienic defects of asylums. The deficient cortical working that is the immediate cause of insanity, and an enfeebled nutrition of the lungs, which may be one of the important causes of consumption, exist together; and these morbid conditions mutually influence each other.

The general mental *symptoms* of phthisical insanity are a peculiar suspiciousness of mind, mild delusions, an alteration of disposition and conduct in the direction of unsociableness, irritability and moroseness, a lassitude and incapacity for exertion—at any rate at first—a want of interest in the usual avocations of life, and a morbid waywardness and perversion of feeling. The patients can pull themselves together for a time, and may have occasional gleams of high spirits, approaching subacute mania. Food is often refused; it is frequently supposed to be poisoned. The chief bodily symptoms are want of appetite, loss of flesh and incapacity to digest fatty foods, weak circulation, a low temperature, especially of the extremities, and various perverted sensations.

Some decided signs of pulmonary tuberculosis are discovered early in the disease, though they seldom advance very rapidly. Such patients have not the well-marked initial attack of acute mania or melancholia, running on into dementia, as in ordinary insanity. The disease comes on in the earlier ages of life, and is always a subacute psychosis. Often the mental and brain disorder so masks the phthisis that no subjective symptoms are felt by the patient at all, not even when the lung disease is far advanced. The *prognosis* is bad, but not so bad as I used to believe it to be. Under suitable early treatment, which, I may say, is that of ordinary early phthisis, about one-third recover from the mental disease; and many also from the phthisis. This result can only be attained by an early recognition of the fact that we have here to treat a mental disease, as it were on a phthisical basis. Phthisical insanity forms about 3 per cent of the admissions to asylums.

T. S. CLOUSTON.

REFERENCES

1. BURKART. "Über psychische Störungen in terminel Stadium der Schwindsucht," *Med. Co. Bild. Worterb. ärztl. Ver.*, 1874, xl. iv.—2. CLARKE. *Journ. Ment. Sc.* xxix.—3. CLOUSTON. *Ibid.*, 1863-4, ix. 36.—4. *Idem.* *Lectures on Mental Diseases; The Neuroses of Development.*—5. MACKLE. *Journ. Ment. Sc.*, 1878-9, xxiv. 25.—6. MAUDSLEY. *Pathology of Mind*, 1895.—7. MOSSELLI, ARTURO, "La Tuberculosi."

T. S. C.

INSANITY AND EPILEPSY

By ROBERT JONES, M.D., F.R.C.P.

EPILEPSY is a paroxsymal neurosis affecting consciousness. It has been defined as "a paroxysmal apyretic neurosis in which the victims are easily-impressed neurotics" (15). It is characterised by intermittent attacks, generally accompanied by convulsions, the major fit, consisting of tonic and clonic spasms. In this association there is no definite constancy; convulsions, as in the minor fit, may be absent (*petit mal*), and although Dr. J. Turner states there are epileptic phenomena without loss of consciousness, the affection of consciousness may vary from a defect only—a more or less fleeting impairment, such as vertigo, loss of memory, a dreamy condition, or special delirium (*épilepsie larvée* of Morel, the *masked epilepsy* of Esquirol, *psychical epilepsy* or the *convulsive idea* of Maudsley)—to an almost complete suspension, eclipse, or abolition of consciousness, as in the coma of the status epilepticus, this condition being responsible for 10·7 per cent of the deaths of epileptic patients in institutions.

The essential feature of a fit is a sudden temporary impairment or loss of consciousness, and the three well-marked clinical stages of epilepsy—the aura, the loss of consciousness with motor disturbances, and the subsequent dreamy or torpid state—may each or all of them be interchangeable with some form of mental disorder (psychical epilepsy). The mental states of the pre- and post-paroxsymal periods when no convulsions occur have special features of their own, and are called "epileptic equivalents." Epilepsy in its functional range is therefore closely allied to insanity, which is itself a symptom of many morbid conditions; the term being applied to certain results of brain disease and brain defect which invalidate mental integrity. In this alliance there are points of similarity as well as of difference or contrast, and they may be considered under the following heads:—

Morbid Anatomy.—Idiopathic epilepsy and insanity have this in common, that both are due to an affection of the organ of mind, and particularly to a disorder of the cortex cerebri. In both the disorder is of the highest centres, and is of so fine a nature that it may elude the minutest research; so fine is it that it has been called a disorder of nutrition only. The general opinion concerning epilepsy has been that the nutritive disturbances need not necessarily express themselves in recognisable morbid change; for, as in some cases of insanity, it has been definitely asserted that no discoverable lesion can be traced, although in other cases gross lesions may be present in both. Thanks, however, to the improved methods of staining, authorities have described after paroxysmal discharge in uncomplicated epilepsy three specific lesions—(*a*) a partial disappearance of the tangential fibres of the cortex, the "neurons of association" so-called, similar to those lesions in general paralysis; (*b*) an infiltration of nerve-cells by leucocytes; and, what is most important, (*c*) a disappearance (chromatolysis) of the fine granules in the protoplasm of the cell substance of the cortical nerve-cells. This cell alteration is of much consequence, in so far as it suggests an instability of equilibrium in the cortical centres, and appears to bear a close relation to the paroxysmal discharges. It is difficult to describe these nutritive changes otherwise than as of a bio-chemical nature, not to be revealed even by the most perfect histological methods. They have their homologues in the cells of the salivary glands, which after activity lose their zymogen granules. Hodge has proved the disappearance of stainable granules in the ganglion-cells of bees after fatigue. In epilepsy associated with insanity, Dr. Bevan Lewis has described highly significant changes in the cortical cells of the second layer. These changes begin in the nuclei, which have a bright, refractive, usually spherical body of a fatty nature in their centre, spreading until it may occupy the whole cell; the cell protoplasm itself eventually betraying signs of degeneration, and ultimately breaking down. Physiologically it is known that the cell nucleus plays an important part in the functional activity of the cell; and that its absence or variation by disease is a constant accompaniment of cerebral disturbances, which are indicated by loss of control. The small pyramidal cells of the second layer have been assumed to be preponderatingly sensory in feature and function, and to be organically connected with the larger deeper cells, which are said to be less preponderatingly motor. Possibly the upper layer may exercise an inhibitory control over the deeper, and lacking this their discharge will be liable to the periodicity of a nutritive rhythm. Sir W. Gowers somewhat differs from this doctrine, as he considers epilepsy to be a disease of the inhibitory fibres—that there is a diminished resistance on the part of the fibrillar "spongy" grey matter, rather than an increased production of energy caused by an active discharging cell lesion. He looks upon the tangential fibres of the grey matter—the "neurons of association"—as more at fault than the cells themselves. Dr. Hughlings Jackson, on the other hand, looks upon epilepsy as an increased nutrition, causing an exaltation of function and issuing in strong discharges. He maintains that hitherto we have not known the pathological process productive of this functional abnormality; but states that to consider epilepsy—occurring in neurotic families interchangeably with insanity or some neurosis by inheritance—as a neurosis of purely nervous origin, beginning in nervous elements which are primarily at fault, is less valid than to assert it to be primarily arterial or venous, and only secondarily nervous and due to damage by vascular changes. It is implied that the true pathological cause is vaso-vagal, and to be sought in nutritive disturbances, caused probably by a change in the cerebral circulation due to the occlusion of a vessel in the "arterio-cortical area," which can give rise to a "discharging lesion." This opinion receives support from the hypothesis, advanced by Cajal, that certain glia-cells related to the arteries of the cortex may exercise a contractility over them, so altering their capacity by dilatation, or otherwise, and that the blood-supply can vary the nutrition of these cortical centres. The protoplasmic neuroglial cells referred to exist in the grey matter; they are continuous with the perivascular lymph-spaces, and they are characterised by prolongations of foot-like cell-processes, which expand upon the perivascular sheath of the arteries and other processes which join collaterals from nerve-cells (see Figs. 41 and 42, Vol. I. p. 581). Their function hitherto can only be a matter of surmise, but their contractility has its analogue in the contractile pigment-cells in the frog's skin.

The morbid anatomy of epileptic insanity has received considerable attention during the last few years. Sciuti has described a dilatation of the lymphatic channels around the small arteries in the nervous system, with lymph stasis particularly in the convolutions of the insula, and in the anterior part of the temporal lobe. G. Guidi connected the pathogenesis of epilepsy with "carbamic intoxication," due to hepatic insufficiency and failure to complete the synthesis of urea, resulting in the presence of toxins which excite the fits. Kransky and de Bucke both confirm these researches, the former tracing the carbamic acid to an intermediate stage between urea and carbonate of ammonia, and deriving the carbamic acid from uric acid, whilst de Bucke derives it from a cytotoxin. Féré wrote on the urotoxicity of the epileptic insane, and noted that the urine is "hypo-poisonous" before the fits and "hyper-poisonous" afterwards.

He considers that the pathology of epilepsy is to be looked for in the convulsive effects of "urine-poisons" upon the cortical centres. Agostini traces these poisons to intestinal auto-intoxication. E. E. Southard describes a congenital segregation of cell groups in cerebral areas corresponding to "epileptogenous zones," which are separated from their controlling area by neuroglial overgrowth pressing upon these immature areas, and so through a gliosis causing a discharge of nervous energy. H. Claude and A. Schmiergeld are inclined to see pathological changes in the internal glands, and to associate these with the epilepsy. Dr. J. Turner (14) believes that epileptic insanity is associated with a congenitally defective structure of the nervous system, together with an abnormal blood state. He states that the "fit" is caused by a sudden stasis of the cortical blood-stream, due to intravascular clots, the stasis being caused by the accumulation of some toxin in the blood. He has shewn that there is evidence that the Betz-cells are immature in form and structure, and that the rate of coagulation of the blood is quicker in epileptics than in any other class of the insane. His conclusions are as follows:—(*a*) a quickened coagulation of the blood, (*b*) an excess of nucleo-protein in the blood, (*c*) an increased leucocytosis during the fits, and (*d*) in consequence of these an elevation of temperature during the period of continuous fits. J. Tolone and also Dr. Robert Pugh have published researches upon the diminished alkalinity of the blood in epilepsy due to the alkaline salts being lessened, and to the presence of an excess of acids from retarded elimination. Biernacki has also described an accumulation of lactic acid in the blood of epileptics. Dr. A. E. Russell considers that the essential feature in epilepsy is a defective nutrition of the cortex due to anaemia caused by cardiac inhibition, and that syncopal attacks pass directly into the more grave attacks with convulsions. He suggests a further study of the circulation in epileptic patients before the actual onset of the fit. Dr. F. W. Mott has published a full account of the morbid appearances of the brain, cord, and bodily organs in a case of status epilepticus (*vide* Plate III. p. 222, Vol. VII.).

Relation of Mental and Nervous States.—We believe in the psychophysical parallel of mental and nervous states; and we find in the excitement of insanity, which constitutes mania, that the mental exaltation is accompanied by violent, purposeless, and inharmonious sensori-motor disturbances—and as sensori-motor we describe the nervous centres which represent impressions and movements; whereas in the opposite condition of melancholia—characterised by a depressed emotional tone—there is a sensori-motor suppression. In epilepsy the sudden disturbance of consciousness is accompanied by sudden and often violent sensori-motor discharges. These discharges of the nervous arrangements are followed by exhaustion, indicated by paralysis on the motor aspect, and by loss of consciousness, which may amount to coma, on the mental side. The loss of consciousness is probably due to a negative functional lesion of the highest centres, as the paralysis is due to a negative functional state of the sensori-motor area. Both these are conditions of exhaustion. This is well illustrated in cases of conjugate deviation due to unilateral convulsions; the nerve tracts after a time become exhausted, and deviation takes place in the opposite direction through the unbalanced action of the healthy muscles on the unparalysed side. That tracts, through which these excessive currents have travelled, become exhausted, is evidenced by the increased knee-jerk and foot-clonus observed in post-epileptiform paralysis (*epilepticism* or the "*epileptic hemiplegia of Todd*").

States of consciousness, we can say, are concomitants of nervous states; the actual relation between these two concomitants remains unknown, but cerebral activity is not thought—the processes are parallel, but not dependent.

Personal conditions relating to epilepsy and insanity may be considered under—

(α) *Sex.*—Sir W. Gowers states that women suffer from epilepsy rather more frequently than men, and that the ratio is 60 to 50, although this is disputed by other authorities who state that idiopathic epilepsy is more frequent in males. In insanity the reverse is the case, for insanity occurs more often in males, although women who are insane preponderate, on account of the higher mortality among men, the proportion among the insane at the present time being 50 women to 42 men living. Men suffer from more acute and rapidly fatal forms of insanity than women, who thus "accumulate" and vitiate statistics by shewing an *apparent* increase of insanity in this sex.

(β) *Age.*—Three-fourths of all epileptics occur under twenty years of age; of these one-eighth are in the first three years of life. One-half of all cases occur between ten and twenty. Inherited cases are more frequent under twenty years than at subsequent ages. In the insane the majority of cases occur between the ages of twenty-five and thirty-five, the age most vulnerable to the combined effects of inheritance and stress. At all ages insane women preponderate, except between thirty-five and forty-five, when men are most numerous. In epilepsy women exceed men up to the age of thirty; afterwards it is the reverse.

(γ) *Civil State.*—Since epilepsy manifests itself before marriageable age most epileptics are necessarily single. This, for other reasons, is also the case in the insane. At the marriageable ages, and in proportion to the population, insanity is more frequent among the single than among the married or widowed. Of married persons insane women preponderate, probably owing to puerperal and epochal disturbances.

(δ) *Numerical Ratio of Epileptics to Insane in Aslyums.*—Epilepsy is stated to occur about once in every 1500 persons of the population, 40,000 of them being sane and 20,000 insane. In some districts it occurs as often as twice in every thousand. The proportion of epileptic insane admissions to non-epileptic insane admissions is 6·0 per cent; but the proportion of epileptics to the ordinary insane in asylums, owing to "accumulation," is usually about 10 per cent; because death from epilepsy is by no means so common as from the various kinds of insanity: general paralysis, for instance, being almost invariably fatal within a period of two years. Indeed it is rare for the epileptic insane to die during a fit; death occurring more commonly from the exhaustion of the status epilepticus.

(ε) *Inheritance.*—It is a principle of universal acceptance and a truism that an inherited neuropathic constitution is the great cause of epilepsy. It is found to occur in 50 per cent of all histories taken in asylums. The attributes of the parent have a tendency to be repeated in the offspring, as there is a continuity in the germinal plasm. There is a tendency on the part of nature towards the normal; an apparent disposition of the molecules under favourable conditions of selection and marriage to sustain and reproduce the healthy, shewing a predominance of the conservative and progressive over the degenerative tendencies. Functional and formative plasm must progress, and the tendency of diseased structures of themselves is active, and therefore towards extinction—witness the tendency to die out in certain royal families which, under compulsion, intermarry. The Jews have been stated by Sichel to be less epileptic and less alcoholic than other races; possibly these two factors are interrelated. As one would expect from the influence of cross marriages, there is a tendency to dilution or concentration of peculiarities in the offspring depending upon contrast or similarity in the parents, so that in this way we may have a metamorphosis of neuroses, and an interchangeability of the different forms—epilepsy and insanity, insanity and hysteria, migraine, asthma, or neuralgia. We recognise in certain families a proclivity which we call "neurotic," a tendency towards nervous instability and degeneration, seen in loss of control affecting either the motor, sensory, or mental aspect. Insanity produced by coarse brain injury, alcohol, local degeneration or disease in the parent, may yield epilepsy in the offspring; whereas the children of the truly "neurotic" or unstable parent are liable to idiocy, moral insanity, or more often the slighter aberrations known as moral obliquity, also probably to the more chronic and permanent forms of insanity with hallucinations. Echeverria states that an epileptic parent entails his disease upon 49 per cent of his offspring. Sir W. Gowers asserts that in pure epilepsy an inherited tendency is stated to occur in about a third (35 per cent) of all cases; and less frequently in males (33 per cent) than in females (37 per cent). In epilepsy associated with insanity an inheritance is traceable in 52 per cent, and more frequently in males (58 per cent) than in females (47 per cent). In the ordinary non-epileptic insane, of 1024 males admitted into Claybury a history was obtained in 683, shewing a neurotic taint in 386 cases, either of insanity, epilepsy, or paralysis (37 per cent of the total males, and 58 per cent in which the history was ascertained); and of 1261 females, with an ascertained history in 771 cases, there was an inheritance in 515 cases—a proportion of 66 per cent, or a proportion of 40 per cent of the total females, giving a proportion of 62 per cent in both sexes combined, and indicating that heredity occurs almost twice as often in the insane as in the epileptic, namely, 62 per cent and 35 per cent. Sir W. Gowers states that there is a family history of epilepsy itself in 66 per cent of his cases of pure epilepsy, and of insanity in 33 per cent. In 211 of the epileptic insane, whose histories were ascertained, there was an inheritance of epilepsy in only 16 per cent, and of insanity in 33 per cent; shewing that in this class insanity is the more powerful factor, and is inherited twice as frequently as epilepsy. Epilepsy appears to be more often the cause of epilepsy in the offspring than of insanity. When in epilepsy there is such an inheritance, it is the women that are more likely to suffer; that is there are more women with such a family history than there are men. In insanity it is the same, and this leaves room for the more frequent occurrence of accidental causes in men, who, as is known, are more exposed to stress and direct hurt than the opposite sex. In epileptics the transmission is more often direct from father to son, and from mother to daughter. In the epileptic insane, men receive their inheritance somewhat more often through the mother, and women through the father. In the ordinary insane, transmission varies. In some forms, however, such as suicidal insanity, it is more often direct than the reverse; the same is the case in melancholia; whereas mania appears to be more often crossed in transmission. It may be worthy of record that Thomann-Koller and Diem found a history of insanity in the ancestors of 59 and 66·9 per cent respectively of healthy persons, and they suggest that in regard to the etiology of inheritance the exact nature of the disease should be studied in the antecedents before conclusions are finally formulated.

Form of Insanity.—Taking the interval between fits in the epileptic as more likely to represent the general mental tone, there is more depression than exaltation. The depression is not quite representative of melancholia, as there are many epileptic inmates of asylums who are weak-minded from progressive dementia. In the ordinary insane, mania or exaltation is twice as common as melancholia or depression; and half as common as all other forms combined. Speaking generally, the insanity of the epileptic is different from that of the ordinary insane. Whilst the lunatic is solitary and egotistical, the epileptic is sympathetic and associative. The epileptic is generally industrious, and benefits to a marked degree by occupation and congenial industrial pursuits. This is the basis of "colony" treatment now advocated for the epileptic and the epileptic insane. Roughly speaking, the epileptic insane are of two classes, namely, the simple or weak-minded, who are generally advanced in years and infirm; and the non-confirmed but mostly chronic, among whom are some of the most impulsive, dangerous, and homicidal inmates of asylums. The former number about 20 per cent, the latter about 80 per cent of the epileptic insane, and of these 20 per cent are of the "very" dangerous.

Basis of the Aura.—The cerebral cortex is the anatomical or physical basis of the aura, and the medium for the transmission of sensations. It is at present described as consisting of five layers of cells—(i.) the molecular or tangential layer, consisting chiefly of cells with caudate "foot-processes" on the surface, a few nerve-cells with dendrons and two or three axis-cylinders, together with a few medullated white fibres (Exner's). On section of the grey matter, there may appear to the naked eye here a white line—the outer line of Baillarger. (ii.) The layer of small pyramidal cells with dendrites upwards and an axis-cylinder downwards. (iii.) The granular layer in sensory projection areas and in the anterior and posterior association areas. (iv.) The large pyramidal layer whose axis-cylinder process gives seven or eight collaterals, and then continues to the anterior horn arborisations. The collaterals of these pyramidal layers form two white plexuses visible to the naked eye as white streaks (inner and outer line of Krause, inner and outer line of Baillarger — the outer being also the line of Vicq d'Azyr). (v.) Polymorphic layer of cells of various forms, with single axis-cylinders and numerous dendrites. Below that is a layer of arcuate fibres which connects one convolution with another. The axis-cylinders are now stated to be only one for each pyramidal cell, and they form projection fibres ending in a network, which may terminate round anterior horn cells, as is the case with the pyramidal cells of the third layer of the precentral convolutions. Their collaterals have one of two destinations: (*a*) they form commissural fibres to the opposite hemisphere ending in terminal cortical arborisations, thus co-ordinating functions in the two hemispheres; (*b*) associated fibres to other parts of the cortex of the same hemisphere. To a nerve-cell with its prolongations and ramifications Waldeyer applied the name neuron, now regarded as an "independent protoplasmic unit," and he divided the whole central and peripheral nerve structures into three systems of neurons, namely, first, centripetal or sensory neurons transmitting excitations from the periphery to the brain; secondly, neurons of association connecting the various sensations in the brain, and augmenting, retarding, or changing them according to the fineness, multiplicity, and almost infinite development of the collaterals, ultimately transmitting them to, thirdly, the centrifugal or motor neurons which convey nerve-waves to the muscles. As regards the aura it has been asked, Where is the actual seat of the sensation or warning? Forel replied that it is in the brain at the point of arrival of the peripherally-excited wave; and that it begins centrally, namely, at the terminal arborisation of sensory fibres (synapse). Dr. Bevan-Lewis believes the aura to be the subjective aspect of the nervous discharges in cortical centres. The scheme in the diagram, taken from a preparation after Ramón y Cajal, represents a reflex cerebral circuit—what has also been called the diastaltic arc. To the right, A, are axis-cylinders ascending to sensory terminal centres; they carry peripheral sensations through the optic thalamus to the neurons of reception in the grey cortex. At B are seen neurons disposed horizontally; their afferent or cellulipetal protoplasmic prolongations are connected with the neurons of reception, whilst their cellulifugal or efferent protoplasmic prolongations join the neurons of discharge C. An aura originates, owing perhaps to vascular changes, in the neurons of association—the "tangential" layer; and is peripherally projected by the neurons of reception to various sense-organs in distal relation to them: it then spreads through the neurons of discharge to pyramidal horn-cells causing motor convulsions. The wave of discharge may modify

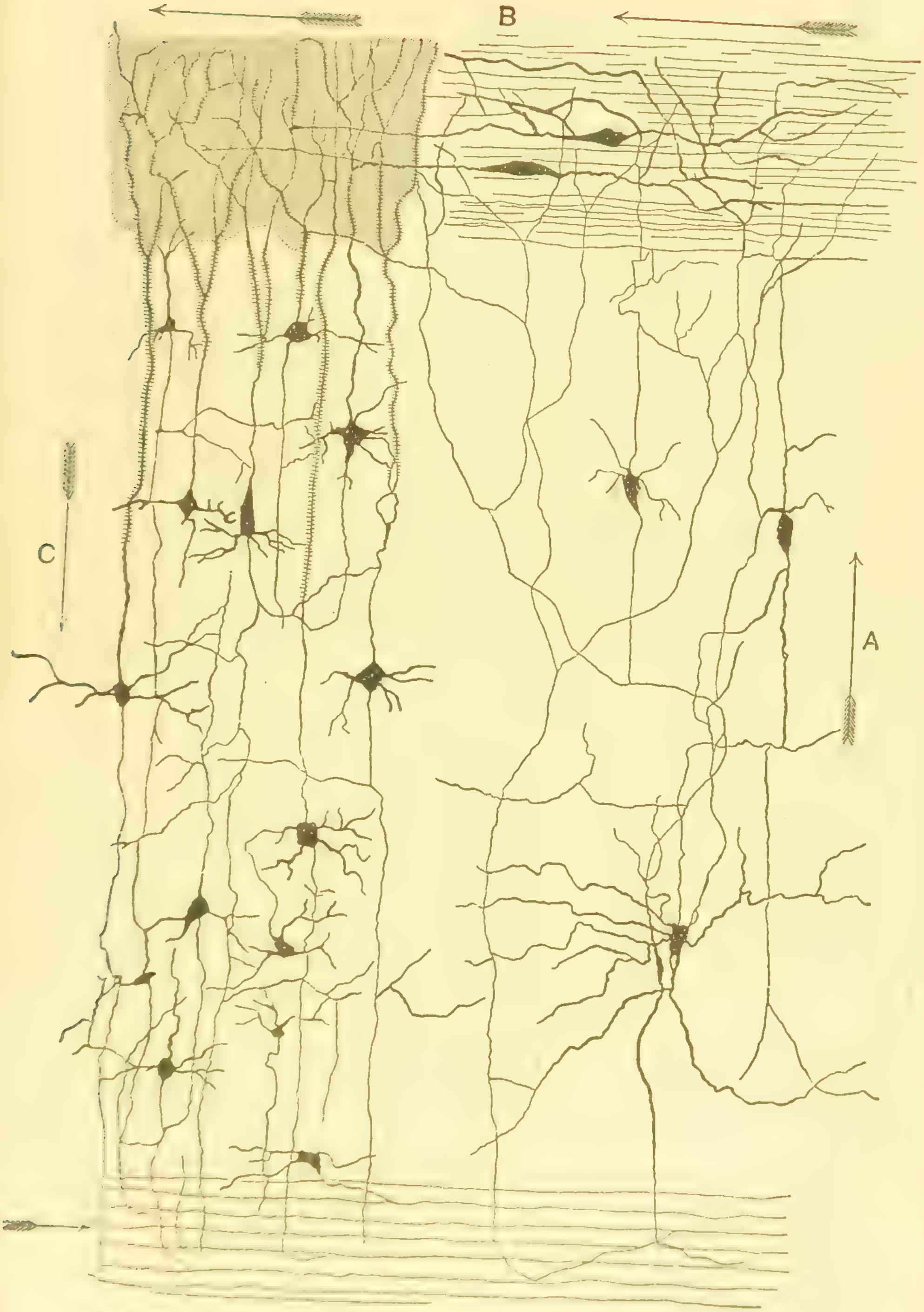

FIG. 27.—A reflex cerebral circuit. (From a preparation after Ramon y Cajal.)

associated sensations in force and character, owing to variations in the associated neurocymes; and upon these will depend the character of the mental or somatic reactions. According to the limitation of these disturbances to the same group of neurons, so will the impressions or sensations be also limited; the same order brings about the same delusions, the same acts and the same sequence of events, a repetition of the symptom-group being characteristic of epilepsy. The experiments of Dana and Ransom on the human subject are interesting in relation to this scheme of the aura, and the sequence of events. Excitation of the cortex was made over the arm area, which immediately caused a motor discharge of which there was no recollection, consciousness having vanished before the discharging lesion had been transformed into motor energy in the anterior horns. It is not improbable that sensory disturbances may precede the motor. Sir V. Horsley has repeated these experiments upon the human subject with weak currents. The central convolutions of the cortex have been looked upon by Dr. Ferrier as purely motor; but by Munk, Bastian, and others as primarily sensory; the motor area being relegated to the bulb and cord. Some experiments, however, tend to support the opinion that they are kinaesthetic. Dr. A. W. Campbell and Dr. J. S. Bolton divide the cortex into the precentral portion—psycho-motor in function—and the postcentral—psycho-sensory; the anterior and posterior parts of the brain being association areas.

Nature and Variations of the Aura.—The aura records the last act in consciousness, and immediately precedes unconsciousness. It may indeed precede the fit by hours or days, though usually only by moments, when it is sudden, acute, and immediate. If the aura be absent, there is generally no recollection of the fit, unless some pain be present from injury inflicted upon the tongue, or some soreness of the muscles after the struggle. At night, wetting the bed may be the only significant discomfort or reminder of a fit. In the absence of these signs, epileptics not infrequently deny fits altogether, and often have to be reminded of them by their nurses or fellow-patients. As to the frequency of the aura, Russell Reynolds gave statistics of 81 cases of uncomplicated epilepsy; in 33 there was no warning, and only a doubtful one in 13 others. He stated that in the majority of cases an aura does not exist. Sir W. Gowers and Sieveking state that an aura is present in 50 per cent of cases. In epilepsy associated with insanity Guinon and Raichline found the aura present in 64 per cent, Marinesco and Sérieux in 66 per cent, and, from an analysis of 116 cases in Claybury, we have found it in 66 per cent. The probability is that it would be present in more cases were it not for inability in some patients, in whom the mind is permanently weakened, to describe sensations accurately. Or indeed the aura may never rise into consciousness. The aura preceding the fit is generally constant for the same person. If a definite interval occur between the aura and the fit it is often of great advantage to the sufferer. As to the character of the aura, it may be—(i.) that of *ordinary sensation*, which may occur in the hand, arm, leg, or one side of the trunk. Sir W. Gowers states that arm sensations never occur above the wrist, but they may be felt as they pass upwards, giving rise to sensations in the arm. Skin sensations are not uncommon—numbness of the fingers, creeping sensations in the head and neck, tingling, heat or cold, and various pricking sensations. The name *epileptogenous zone* was applied by Brown-Séquard to the part of the skin in certain animals experimented upon, which on being irritated produced epileptic convulsions. It is said to be almost unknown in the human subject. In a case of Jacksonian epilepsy of the right arm and hand, by shaking the limb severe convulsions in it could be produced at will, followed by general convulsions of the whole body, which became involved in one violent epileptic seizure with loss of consciousness. Heat in the eyes, fire between the teeth, or a feeling as if the teeth were coming out (the person holding her right hand over her mouth to prevent them falling out), are all known as auras preceding the fit. (ii.) *Motorial*—a stiffness or limpness, a general trembling of the whole body, and certain peculiar circular movements. A running gait, *E. cursiva*, also may precede the fit. Megazzini has described the formation of circles (*E. circumcursiva*), and a rotation in the long axis of the body (*E. rotatoria*) as an aura, or as replacing the fit and followed by stupor. He attributed them to unilateral cerebellar ischaemia. Bilateral motor auras are not frequent, except in the trunk as general tremors. (iii.) *Vocal*—the aura may consist of certain phrases which are mechanically repeated. (iv.) *Visceral*—probably influenced through the vagus—are not uncommon, and may be in the throat, thorax (cardiac and respiratory), or abdomen. If abdominal, they are usually referred to the epigastrium, and are most frequently on the left side. The fits that follow epigastric auras are very often severe. Cardiac sensations are often very painful; some complain of strings being wound round the heart, others of a palpitation with a choking sensation; and, when recovering from a fit, they have a mechanical way of moving their hands as if winding imaginary skeins of thread or taking foreign bodies off the tongue. Those who have feelings referred to the throat and chest are not infrequently extremely restless and troublesome. (v.) *Vasomotor* auras are not common. (vi.) *Cephalic* auras, such as vertigo, a swimming in the head, somnolence, or these in combination with other auras, such as giddiness with disturbances of vision, are frequent. (vii.) *Special-sense* auras.—(*a*) Olfactory is rarest. Hallucinations of this sense are not more rare in the insane than in those who are only epileptic, although the opposite statement has been made upon eminent authority. (*b*) Gustatory aura is next in frequency; and then (*c*) auditory, mostly sudden explosive noises, whizzing and hissing, less often distinct voices; when these occur they may influence conduct after the fit. (*d*) Visual auras are the most common, and occur twice as often as all the others together. Sometimes they approach, but more often recede. Lights may be seen after the sight is lost, shewing that discharges may occur in a disused or an inhibited centre. Illusions of sight are frequent; perhaps these are more nearly related to psychical auras, in so far as they rule conduct when occurring. All the auras, being presumably central in origin, are for the same reason psychical; but they are also sensory in so far as they are projected peripherally, and more specially to the various sense-organs. It has been seen that the last impression in consciousness before a fit may be revived after the paroxysm, and sustained on regaining consciousness. This has been called the *epileptic echo*, and may take the form of a directing voice, an *imperative idea*, or a *fixed delusion*. Thoughts occurring as an aura, or directing voices heard before a fit, whether persecutory (which is most frequent) or the reverse, may thus be the incitement to impulsive actions performed subsequently without consciousness. (viii.) *Psychical* auras.—The basis of the intellectual and moral life is in the sensations and emotions, and as the aura is of central origin with peripheral projections and manifestations, it would appear that all auras are in their nature psychical, although it is convenient to describe them as they appear to occur in somatic-sense organs. Mental associations referring to abstract qualities, such as the good, the evil, or exaltation or depression, not referable to special-sense organs, may perhaps be more appropriately considered as psychical; and, using the term as synonymous with mental, there are undoubtedly auras of this description. The term, however, has been indifferently used and loosely applied; it has been considered as synonymous with the *masked epilepsy* of Esquirol, the *épilepsie larvée* of Falret and Morel, also with the state generally described as *automatic*, which is that of imperfect consciousness with actions apparently conscious. This state is neither a warning nor a premonitory sign of an impending seizure, neither constant in its character nor periodic in its occurrence, but a pre- or a post-paroxysmal condition which may even replace the fit altogether. Sometimes a patient can stop the fit by retarding or controlling the aura, as with a piece of string round the arm in Jacksonian epilepsy. A patient states that if by an effort of will he can fix his attention upon one particular train of reasoning, he can stop the fit. It is possible that a new sensation is imparted to nerve-fibres, which raises their inhibitive power, and thus prevents the spread of the discharging lesion. (ix.) *The epileptic cry* is not an aura, although it occurs immediately before the distressing features of the fit. It is due to spasm of the respiratory muscles forcing air through a closed glottis. There is usually one long cry; but there may be several interrupted ones, according as the spasm is tonic or clonic. Patients rarely recollect the cry, as it generally comes on after the loss of consciousness. The cry is of far greater import to those attending upon the epileptic insane than the aura, as nurses and attendants, who are always about, get to know the cry and recognise the person from whom it comes. Thus they can render assistance before the fall, and often prevent it. Nurses say they can recognise the fit from the cry, and at night when sleeping in bedrooms adjoining patients' dormitories, they can distinguish from a large number who is in a fit. The aura is a subjective phenomenon, the cry very distinctly objective.

Pre-paroxysmal Stage.—Certain conditions of mind occur both in the ordinary and the insane epileptic, which are best described under this heading. A change in the moral conduct of the individual may take place, to which the term *moral mania* has been applied. He may become sullen and moody, and fall into delusions of suspicion; or he may give way to exceptionally violent and sudden fury, which only those who have seen it can appreciate. Dr. Maudsley, calling it the *furor transitorius*, describes it as having no bounds. The patient is absolutely regardless of consequences, he observes no obligation to others, and is defiant of reason. His actions during this stage are impulsive, blind, wild, and furious; and he is quite unconscious of his destructive energy. He is subject to gusts of passion and calls out with maniacal and vociferous force, insisting on trivial demands as if his life depended on them. He shouts night and day, and is sleepless for many nights; he takes no food, and his eyes stare with a desperate glare. However much in ordinary mania the gentle answer may turn away wrath, upon the irritable, spiteful, and quarrelsome mood of the epileptic it is wasted; nay, it may even aggravate the ruthless progress of his uncontrollable, impulsive, paroxysmal violence, which in its fierceness and vehemence is terrific and fiendish. If a fit occurs at this stage, a calm succeeds, excitement ceases, and reason returns. But a fit may not occur, and the mania becomes a true *epileptic mania* taking the place of the fit. This condition may happen in both sexes, women sometimes manifesting hysteroid symptoms associated with violence. Another condition which may occur before the fit—which is in this case of a vertiginous and momentary character—is a dazed and stupid dulness, lasting for a few minutes only, rarely longer. The patient does not fall, and there are no convulsions. He continues in a dreamy state, and performs actions of which there is no subsequent recollection. For example, a student at college entered a class other than his own, and without his gown; he then insulted the professor, and immediately fell into a fit. On recovery he had no recollection of his delinquency. A youth at table suddenly gets up in the middle of conversation, quaffs the various drinks belonging to other guests, and then falls in a fit. Dr. Savage says this automatism occurs more often after than before the fit, and calls it the *automatism of the post-epileptic state.* The fits that usually accompany this condition are not of the convulsive variety, and, being often of momentary duration, may not be noticed.

Paroxysm.—This has been described elsewhere (p. 493). I will only state that the destructive effect upon the mind depends rather on the frequency than the gravity of the seizure; the convulsions in themselves being less harmful than the slighter attacks.

Post-paroxysmal Phase.—Epileptics are more dangerous in this stage than in any other. They frequently have hallucinations which are systematised into delusions. They fancy that their friends or their enemies are about them, they can see and hear them, and they do most unaccountable things. They are more continuously noisy, violent, and perverse in this stage than in the pre-paroxysmal: but they are less impulsive and explosive, being guided more by hallucinations than by blind fury. Actions in this state are also less conscious. This is the *period of automatism*, the *dreamy state*, the state of "*dual consciousness.*"

Dr. Maudsley asserts that this condition may precede or replace an ordinary convulsive seizure, but automatism is almost always a post-epileptic phenomenon. This state much resembles somnambulism, for there is a partial suspension of consciousness in both; and some writers regard them as having a common origin in the neurotic temperament. There is but little doubt that automatism depends upon the inheritance of certain habits noted in the individual species. There is no recollection of events that may have occurred during this period of imperfect consciousness, even when actions end in the direst results. Anger, violence, retaliation for imaginary wrong, purposeless acts, assaults, murder, and indecent exposure have all been recorded during this phase. When the mental symptoms replace the convulsive seizure, the condition has been described by Echeverria as *intermittent mental epilepsy*. It is the *masked epilepsy*, or the *épilepsie larvée* already referred to; but it is now generally understood that a slight fit precedes this automatism. The fact that it occurs most frequently in the slighter forms of epilepsy, in which the symptoms are in the main subjective, momentary, and fleeting, and that it is a known sequel of some attacks of the graver form, support the view that automatism is in its nature post-epileptic. Often the very cases in which the most serious consequences follow are those in which it is most difficult to prove medico-legally that disease sufficiently accounts for the acts. The slighter forms of automatism are not uncommon. Conduct is often the only clue to a fit in these cases. Generally speaking, the actions repeated during the automatism are those performed daily by the person, as the centres in most frequent use discharge most readily when the inhibition of the higher centres is withdrawn. To this repetition of habit the French apply the name *professional automatism*, as examples of which a tailor mimicked work for ten minutes after the attack, and a laundry-man repeated his usual technical movements for twenty minutes after a vertiginous attack. Dr. W. Orange quotes the case of a woman who, while cutting bread with an infant in her arms, was suddenly seized with a slight fit, after which she automatically cut up her child. Persons starting off to their usual daily callings will find themselves at the end of a day or two (and to their profound astonishment) far from home without knowing how they got there (*comitial ambulatory automatism*). Undressing is a frequent and often very inconvenient form of automatism. Dr. Hughlings Jackson attributes these acts to an affection of the highest cerebral centres, the exhaustion of which by the discharge removes the inhibition from the next highest centres of which those most frequently in ordinary use respond most readily. "*Double consciousness*" resembles the automatic state, but is usually more prolonged. All the organic functions are normally performed, but there is no consciousness of ordinary life, all its actions being unreasonably performed in a mechanical manner. A case is recorded of dual consciousness for one year following a fit; after another fit the patient emerged into her previous conscious life, but the past year was to her a total void. Violent hysteroid conditions may follow fits in young women, when they become deceitful, vain, mischievous, and troublesome, often falling into delusions about the sexes (*vide* also p. 503). *Verbigeration* has been noticed in some during the post-paroxysmal automatism, but the condition hardly corresponds to that of katatonia described by Kahlbaum. There are a few instances of all these varieties in every large asylum.

Effect upon the Mind.—(*a*) *When unassociated with insanity.* A high degree of intelligence, amounting even to genius, has in some cases been associated with epilepsy; and it is well known that men of mark and note, who have created epochs, have been leaders of men, or have originated schools of thought and systems of religion, have been subject to fits of epilepsy. Julius Caesar, Augustus, Caligula, Mahomed, Joan of Arc, Savonarola, Swedenborg, and Napoleon Bonaparte are all alleged to have suffered from epilepsy. This greatness of mind may occur also in persons disposed to insanity, of which alliance many painful examples are on record. A certain instability is essential to nervous action. In the normal stage, which allows of a ready re-arrangement of the nervous molecules with a free and easy liberation of energy, this instability necessarily exists in varying degrees; and mental development amounting to genius may coexist with morbid instability of some parts of the nervous mechanism. Epilepsy may coexist with a healthy growth and development of the intellectual faculties, and it is not until the fits become frequent that the malady exercises a destructive influence; the *petit mal* is believed to signify a more retrogressive condition. Even when no definite mental weakness is present, a certain peculiarity is common to the class, and dominates their character,—a peculiar *irritability*, with which may often be noticed a fervour and a zeal which makes these patients particularly susceptible to religious impressions. The epileptic is apt to be influenced greatly by the mystical or the awe-inspiring, and he is disposed to morbid piety. He has an outer religiousness without corresponding strictness of morals; indeed the sentiment of religious exaltation may be in great contrast to his habitual conduct—the key-note to which is hypocrisy—which is a mixture of irritability, vice, and perverted instincts. (*b*) *When associated with mental weakness in the young*, epilepsy is often the cause of imbecility, varying from the lighter forms to the most profound idiocy; 25 per cent of all the cases admitted in one year into Earlswood were there through epilepsy. Infantile epilepsy begins in the majority of cases during teething, and in the first year of life; hereditary epilepsy, as we have already seen, manifests itself earlier than the non-hereditary form. In respect of prognosis, the most improving cases are those in which fits begin after puberty, in which, that is, the neurosis is not inherited. In adults the mental changes are of two kinds—one patient is sombre, distrustful, irritable, and quick to take umbrage, to quarrel, or to wound and strike; the other is obliging, obsequious, cajoling, full of effusiveness, and of a shallow and untrustworthy goodness.

After a longer or shorter time, depending upon the frequency of the fits, the probability is that mental enfeeblement will set in; hallucinations may arise in some cases, setting up delusions which become systematised, and, as the fits continue, dementia will ensue. The chronic dementia of epileptics is characteristic. The aspect of these dements is dull, listless, and heedless, with a dreamy, heavy, and unintelligent expression. The face is not infrequently distorted and ill-shapen from scars or injuries due to falls. Saliva may dribble from the mouth, and when the patient walks his carriage is drooping, the head and neck being downcast and bowed. His mental reaction is slow, although there are still irritability and impulsiveness; his habits are faulty and he is solitary, he prefers to be left alone. He is neglectful of his personal appearance and oblivious of his surroundings. His muscular movements are sluggishly performed; he dresses or feeds himself (when able to do so at all) in a clumsy and ill-directed fashion; his common and other sensations are blunted; he feels and handles things as if his hands were numb from cold; his gait is dragging, and when he sits, or lies in bed, he prefers to do so huddled together in a heap, or crouching more like an animal than a human being. His impaired circulation gives him the appearance of an inanimate heap with venously congested extremities. A characteristic aspect is also known and recognised in some of the varieties of insanity, shewing the relation of the two diseases. In the intervals of fits, that is, between any two seizures, insanity in the ordinary epileptic is very rare; in asylums the epileptics are fairly sane between the attacks, the insanity in them being generally intermittent or remittent. The tone of mind is melancholic or depressed, although ordinarily there is no permanent mental derangement. The melancholia may either be accompanied by ideas of persecution, and a tendency to fits of passion—such as irresistible impulses to homicide and suicide, or by hallucinations of a depressing, fearful, or terrifying nature. The general sanity of the interval is noted by the sociability and altruism of epileptics—often the first sign of convalescence in the ordinary insane. They may often be seen in little coteries, or friendly groups, in the grounds or the wards of asylums. Rarely is the ordinary lunatic inclined to associate with his fellows, or to shew consideration to those around him. Egotism and independence are his doctrine and mode of life, and he is slow to recognise any obligation to others. In some cases of congenital weakness this perversion is an interesting peculiarity, for, like colour-blindness or tone-deafness, it is an inborn constitutional feature; whereas in the insane and the epileptic advanced in dementia it follows the rule that the latest-obtained and last-developed acquirement, being a faculty of the highest centres, is the first to suffer. The moral sense and the conduct being dependent upon the higher emotions are the first to be affected, and this change is often noticed by the friends of the patient long before it becomes necessary to place him under treatment.

Relation of Epilepsy to General Paralysis.—It is not easy to discriminate between these diseases when, as is frequently the case, the latter is modified. General paralysis is of necessity a protean malady, recognisable by the association of certain cerebral or cerebrospinal symptoms, varying as the brain or the brain and spinal cord are respectively affected. When tabetic or motor disturbances are present alone, there may be no exaltation or special mental symptoms. The character of the seizures in general paralysis also varies; for there may be all varieties, from *petit mal*, or syncopal, hemiplegic, monoplegic, and unilateral twitching, to epileptiform or apoplectiform seizures, not one of which alone is pathognomonic of the disease. The pupils are most often fixed in general paralysis, the light-reflex being absent, which is not the case in epilepsy, although when it is associated with hemiplegia the pupils are often unequal. A history of syphilis is usually obtained in general paralysis, but this is not specifically the case in epilepsy proper, although some authorities have recently associated the two more closely than has hitherto been the custom. The different effect of treatment upon the two diseases, and the prognosis involved, make it necessary that a correct diagnosis should be made; and, speaking generally, the symptoms upon which most reliance can be placed are the character of the seizures—which ordinarily are convulsive or congestive—and the pupillary reaction. The Wassermann serum-test and the degree of lymphocytosis in the cerebrospinal fluid also help to a diagnosis. The Noguchi test has recently been used for diagnostic purposes in doubtful general paralysis. Patients in the fits of general paralysis rarely bite the tongue, and there is no aura. The general paralytic seizure is of longer duration, and is usually continuous; whereas in the status epilepticus intermissions may be noticed. The cry has been considered peculiar to the epileptic fit, but this is not the case. The history of the patient may aid us; for epileptic fits, if occurring in middle life, and associated with mental symptoms, and with the rapid onset of a paralysis, must always suggest the diagnosis of general paralysis. The general relation of epilepsy to insanity is best summarised by Dr. Savage, than whom there is no higher authority. "Epilepsy may be a true neurosis by origin and relationship. It tends to a marked degeneration of mind, and affects the moral side of man specially. It tends to idiocy if present in infancy. It leads to rapid degeneration if occurring in youth. It may be associated with genius. It is often associated with hallucinations of a special type. It may be associated with delusions. It may be related in various ways with regular attacks of mental disorder; thus insanity may precede, replace, or follow the fits of epilepsy. There may be an attack of mental disorder of the ordinary type, or there may be a period of automatism. Chronic epilepsy with chronic insanity produces a very hopeless and dangerous class of insane patients, in whom some special ideas and specially dangerous impulsive attacks are present." The relation of epilepsy to other forms of insanity has been studied by Magnan, who suggests that one form may be inherited through the father, and another through the mother. Cases of paranoia combined with epilepsy have been reported. Ziehen explains the mental condition as a delusional state, through incorrect deductions made during the confusion associated with the fit. Buchholz, on the other hand, suggests that the epilepsy weakens the mind, which gives way to its inherited tendency to develop delusional ideas. The relation of epilepsy to alcoholic insanity may be divided into two parts: (*a*) the fits associated with acute alcoholism which are intense and violent in the latter, but unaccompanied with the aura and the cry, the patient having a cloudy recollection of the delirium, whereas in epilepsy there is no recollection and no response during the fit; and (*b*) the fits associated with chronic alcoholism in which also there is no aura, but the fit is limited to a group of muscles and not general. There is also a tendency to clonic or involuntary movements of the muscles of deglutition in the spasms of chronic alcoholism (3).

Treatment.—The medicinal and operative treatment of epilepsy are discussed in the article on this disease (p. 512), and references are also made in that article to the influence of diet.

The subject of a suitable dietary for the *epileptic insane* has received very careful attention from time to time. Experiments have been made with exclusively vegetable diets, but experience has proved a mixed one to be the most appropriate. The protein of vegetable substances is found in association with relatively large quantities of starch which, being enclosed in a network of cellulose, is excessively resistant to the action of the digestive juices; whereas protein obtained from an animal source is more readily assimilable and more sustaining. The reasons, however, for suggesting a vegetable dietary were that in the metabolism of a nitrogenous animal diet, lactic and phosphoric acids accumulated in the urine; and, as Araki, working in Hoppe-Seyler's laboratory, has proved, lactic acid appears also in the urine after severe epileptic convulsions. Therefore to diminish acidity from this cause by a simple dietary might act as a valuable therapeutic agent. What control a purely vegetable diet may exercise over lactic acid, or what relation lactic acid bears to fatigue-products, is a matter of which at present we know but little.

Flechsig's treatment, consisting in the daily administration of less than a grain of opium increased gradually to 4 or 5 grains, indeed to a maximum of 10 or even 15 grains, for a period or five or six weeks, and then followed by bromides in daily doses of 1 to 2 drams for two months, has proved disappointing. Opium is unsuited to cases with hallucinations; or with a tendency to renal disease, bronchial catarrh, or plethora. The routine treatment advocated at Claybury is the administration of bromide of potassium, sodium, or strontium together with borax every night for a year, to provide a free evacuation of the bowels, and the administration of sedatives (chloral and bromide) by the rectum during the status epilepticus. Nourishment by the bowel must be administered when the patient is in this state. The treatment by hypo-chloridisation is also in use, the salt-cellars being filled with sodium bromide at dinner for the more "epileptic." Hyoscine is used for excitement, and as much occupation, employment, and amusement as possible are provided.

Bechterew's fluid, in so far as it consists of the bromide salts, has been found beneficial. The same may be said of the Dragées antinerveuses de Gélineau, which probably derive their medicinal value from the same salts, although arsenic and picrotoxin are said to enter into their composition. The treatment advocated by Ceni of administering to epileptics their own serum or that of other epileptics has not so far been carried out in this country. Operative measures have been recommended for the cure of epilepsy, and both Mr. Rushton Parker and Mr. Damer Harrisson have published results. On the other hand, it has been stated that apart from focal or Jacksonian epilepsy, idiopathic or true epilepsy has been relieved by operation even where no apparent *raison d'être* existed for operative measures.

As to the recent growth of opinion in favour of the segregation of this class in what has been called the "Epileptic Colony," it must be pointed out that this method was initiated nearly fifty years ago by a Lutheran clergyman in Westphalia, Pastor von Bodelschwingh, who was actuated in the first instance by philanthropic motives. He believed it possible to found a hospital where those suffering from epilepsy might perhaps be cured, or, if recovery were impossible, might have a home in which their mental faculties could be developed, or at any rate preserved from deterioration.

The treatment aimed at securing freedom from anxiety, while encouraging contentment and industry by occupations suitably selected according to individual tastes; thus, by assisting patients to lead even and well-regulated lives, they might be led to forget their affliction, and helped to think that they were taking some part in the work of the world. A small farm with four patients has now developed into a colony of over 2000. The same system has been successfully carried out by Bourneville at the Bicêtre and in the Fondation Vallée for girls who are epileptic and defective. Industries and trades, gardening and farming, domestic work, and minor scholastic attainments are taught, and with great benefit, both patients and scholars feeling that they are doing something to help themselves and others. This interest and contentment diminish the fits and prevent further mental deterioration.

Considering that the natural termination of epilepsy is a hopeless dementia, the results have been extremely favourable; 6·5 per cent are stated to be discharged recovered, and 20 per cent returned to their homes improved after varying periods of residence. These encouraging results have brought into existence many similar establishments, both on the Continent of Europe and in America—the States of New York and Pennsylvania, California, New Jersey, Massachusetts, and Ohio (the oldest) having large institutions for the care of their epileptics. Craig Colony, in New York State, has had much thought bestowed upon it and is well equipped for research. In this country the need was strongly urged for (i.) epileptic children; (ii.) epileptics not insane; and (iii.) the epileptic insane. A National Institution was founded in 1893, at Chalfont St. Peter in Buckinghamshire, for the adult epileptic *not* insane, and is now being extended. The routine medical treatment at Chalfont places every patient upon half a dram of bromide of potassium at bedtime; occupations, mostly in the open air (agriculture, horticulture, and dairy work), are provided, and a carefully arranged dietary is prescribed—avoiding "strings and stones, skins and bones." The epileptic seizures are said to be less frequent, and the general mental and physical condition to be ameliorated. Some cases, however, prove unsuitable and have to be transferred to asylums. The first home of this kind in England was at Maghull, near Liverpool, established in 1888. Since then institutions have been established at Alderley Edge in Cheshire by the David Lewis Trustees. There is a special colony also at Lingfield, in Surrey, one at Starnthwaite, and a "home" at Bodmin and Much Hadham, Herts, and one at Sandlebridge. The Defective and Epileptic Children Act (1899) permits local authorities to make special provision for epileptic children, but hitherto no County Council has adopted the Act, although London contemplates Residential Schools under the Act, also the borough of Blackburn proposes to erect one as well for epileptics as for mentally defective children, and to accommodate 40 boys and 40 girls. The Worcestershire County Council also proposes to erect a Joint-Home, under an order made by the Local Government Board, for 210 epileptic and mentally defective persons not certified as insane.

To the friends of the "colonists" the relief must be immeasurable, and it is impossible to over-estimate the advantages of these "Homes" to the community, which is thus no longer hampered by an unproductive burden.

The London County Council has considered the subject in respect of their epileptic insane, and a colony for 300 patients (247 males and 53 females) has been established at Ewell in Surrey. There are over 19,000 insane persons in the asylums of the Council, and of these 1944—a proportion of about 10 per cent—are epileptics. It is computed that 1 in every 1500 of the population is an epileptic, and there are therefore over 4000 epileptic persons (male and female) within the metropolitan area. Indeed there are over 600 sane adult epileptics in the London workhouses. Altogether in the various asylums and licensed houses there are over 30,000 insane persons in London; and it may be computed that among these are possibly 3500 insane epileptics, the greater number being males. The proportion of epileptic admissions, as compared with all other admissions into asylums in England and Wales during the five years ending 1909, was 7·6 per cent, being in the proportion for the two sexes of 8·8 males and 6·4 females, roughly nine males to six females. Thus, there are probably about 3500 insane male epileptics in London under the supervision of the Lunacy Commissioners; but not more than about 30 per cent of these would benefit by special occupation; in the intervals of their paroxysms these patients are quiet, able-bodied inmates who already do much of the work in asylums, and with more suitable occupation their lives would probably be much happier.

The scheme of the London County Council in regard to the epileptic insane has been to erect and support (*a*) a hospital for the cure of epilepsy; (*b*) an industrial institute with a number of callings for the adults; and (*c*) a home for the demented.

Robert Jones.

REFERENCES

1. Bourneville. *Brit. Med. Journ.*, 1907, ii. 459.—2. Claude and Schmiergeld. *Rev. Neurol. and Psychiat.*, Edin., 1909, vii. 412.—3. Elié, L. *Journ. de méd. de Par.*, 1908, 2. s., xx. 36.—4. Guidi. *Riv. sper. di freniat.*, Reggio-Emilia, 1908, xxxiv. 110-158.—5. Harrisson, Damer. *Brit. Med. Journ.*, 1907, i. 318.—6. Mott, F. W. *Arch. Neurol.*, Lond., 1899, i. 493.—7. Parker, Rushton. *Brit. Med. Journ.*, 1907, i. 1477.—8. Russell, A. E. *Lancet*, Lond., 1909, i. 963, 1031, 1093.—9. Sciuti. *Ann. di nevrol.*, 1907.—10. Sichel, M. *Rev. Neurol. and Psychiat.*, Edin., 1908, vi. 443.—11. Southard, E. E. *Amer. Journ. Insan.*, Balt., 1907-8, lxiv. 607.—12. Tolone, G. *Manicomio*, Nocera, 1907, xxiii. 33-46.—13. Turner, J. *Journ. Ment. Sc.*, Lond., 1907, liii. 1-84.—14. *Idem. Ibid.*, 1908, liv. 638.—15. Woods, M. *Journ. Am. Med. Assoc.*, Chicago, 1908, l. 663.

See also textbooks by Bianchi, 1906; Clouston, T. S., 1904; Kellogg, J. H., 1897; Bevan-Lewis, 1899; Macpherson, J., 1899; Maudsley, H., 1895; Régis, E., 1895; Savage, G. H., and Goodall, E., 1907; Stoddart, W. H. B., 1908; Tuke, Hack, 1892. The following journals should also be consulted: *Brain*; *Journal of Mental Science*; *Journal of Nervous and Mental Diseases*; *Review of Neurology and Psychiatry*, Edin.; *Archives de neurologie*; *Deutsche Zeitschrift für Nervenheilkunde*; *Monatsschrift für Psychiatrie und Neurologie*; *Neurologisches Centralblatt*; *Revue neurologique*.

R. J.

RECURRENT INSANITY

By A. R. Urquhart, M.D., F.R.C.P.Ed.

Over and above the tendency to relapse in the ordinary forms of mental derangement, there is a well-marked class of cases in which this tendency is a prominent feature. Too often the tendency passes into a habit, and the habit becomes incorrigible. A brief reference to recurrent insanity is of importance to the student of mental diseases because it elucidates the laws of periodicity, it draws attention to facts of varying psychical phenomena in the same person, it emphasises a very real danger, and it indicates a line of treatment which must not be overlooked in practice.

We have to take into account the laws of periodicity in dealing with all classes of diseases. The annual round of spring, summer, autumn, and winter brings in its train such maladies as bronchitis, insanity, diarrhoea; each in its season. Suicides are most numerous in summer, just when nervous diseases attain their maximum. Similarly, in the individual there are periodic changes of condition, mental and bodily: changes consistent with health, and therefore indispensable; changes more or less morbid, and therefore antagonistic to personal well-being. When we pass from the consideration of mere moods and feelings, or from the ordinary effects of the menstrual molimen, to the study of actual disease, we are at once face to face with a great array of facts illustrative of periodicity. There are the phases of relapsing fever, of recurrent appendicitis, and so forth; and this character is certainly not less marked in regard to nervous diseases. The nerve-storms of megrim and epilepsy are familiar to us in professional life; and further, the metastases of nervous maladies, the see-saw between one and another, are not less evident to the careful observer. Dr. Blandford has placed these observations in their proper relation to insanity, and it is unnecessary here to follow them out in detail.

The forecast of the "recovery" of an insane patient is complicated by this tendency to relapse; and, as already pointed out, the net number of patients who remain sane—that is to say, who have recovered and do not relapse—is a very different figure from the gross recoveries. We hold no man recovered until his life-history is complete. The outcome of these inquiries, then, goes to prove that insanity illustrates the biological law of periodic changes, and that our aim must be to restrain these within the bounds of health. In their recurrent manifestations there is nothing to be added to the symptoms of mania or melancholia except to note the frequency with which the same ideas and acts are repeated in periodic cases. This is so marked that it might disclose the real nature of the malady to a watchful observer. Empirically, in a case twice maniacal or melancholic we may speak of "a second attack"; and on the third attack rename the case "recurrent mania," or "recurrent melancholia," as may be. Although periodic insanity may begin at any age, it may be stated that if a patient, young in years, of hereditary tendencies to insanity, has a sharp attack of mental disease, and recovers speedily, the prognosis is unfavourable in this respect. Should there be signs of degeneracy on the first onset, dementia, or, more rarely, periodic insanity is to be feared. The omens are inauspicious, and treatment must be at once and constantly employed to avert such issues.

Circular insanity was first specially described by Baillarger under the name of *Folie à double forme*, and by Falret under the name of *Folie circulaire.* It is a well-marked form of mental derangement occurring in cycles, the attacks being characterised by a sequence more or less regular—one period of depression being succeeded by another of excitement, which again is followed in some cases by an interval of sanity or quiescence. Much has been written about this malady, and stress has been laid upon the difference in the initiatory and succeeding stages in different cases. There is no need to recapitulate the symptoms in detail. The melancholic and the maniacal stages present the ordinary features of similar isolated attacks in other patients, with perhaps a predominance of the sexual proclivities. As with other periodic insanities, the influence of heredity, the more frequent occurrence in women, the unfavourable prognosis are all strikingly exemplified. It is not a common form of mental derangement in this country. The patients labouring under circular insanity, in Dr. Clouston's experience, were nearly all persons of education; and far more than a due proportion of them were members of old families. Two cases now under my care present these features, but conform to no law of seasonal or sexual periodicity. During the lucid intervals they appear quite sane to any ordinary observer, but on closer examination mental weakness is discernible. It is remarkable that these nerve-storms should be so severe, so long continued, and yet leave the patient, in many cases, with intervals of mental and physical well-being, it may be up to the close of a long life; although in advanced cases there is an undoubted tendency to dementia.

In the *treatment* of periodic insanity perhaps the most important consideration is in respect of the possibility of improvement, or control, by the effects of change. Should the vicious circle be established but little can be done towards cure. Therefore, when an adolescent patient, of hereditary predisposition to insanity, quickly recovers from an attack of acute mental disease, some risk must be accepted in urging a complete change of circumstances. It may be too late to resist the beginnings, but it may be possible to repair damages and to prevent iteration. To this end there is nothing more potent than change of scene and circumstances, repeated as discretion may dictate, together with a wise guidance on the lines of health and sanity. It is unnecessary to discuss the value of hypnotics, rest in bed, exercise, occupation and amusement, baths and massage, tonics and stimulants. The physician must consider the individual case, and the particular stage of the malady; in no disease is the mere treatment of symptoms less appropriate, or the mere administration of drugs less effectual.

In former days much legal fine writing was spent upon the occurrence of "lucid intervals." These now merit no such exceptional consideration. The questions affecting responsibility and competency are to be decided in the individual case, and in the light of the whole circumstances of the patient.

A. R. URQUHART.

REFERENCES

1. BAILLARGER. "Folie à double forme," *Bull. Acad. de méd.*, Paris, 1853-4, xix. 340.—1*a*. BLANDFORD. *Insanity and its Treatment*, 4 ed., 1892, Lond.—2. BONNET. "Folie a double forme," *Arch. clin. des mal. ment.*, 1862.—3. FALRET, "Folie circulaire," *Bull. Acad. de méd.*, Paris, 1853-4, xix.—4. FOVILLE. "Folie à double forme," *Brain*, London, 1882.—5. GREIDENBERG. "Ueber recurrirende Psychosen," *Centralbl. f. Neurol. u. Psychiat.*, 1891.—6. KERR, H. *Glasgow Med. Journ.*, 1899, liii. 414.—7. KIRN. *Die periodischen Psychosen*, Stuttgart, 1876.—8. KRAEPELIN. *Clinical Psychiatry* (Johnstone's Translation), London, 1904.—9. PILCZ. (Etiology and Morbid Anatomy), *Monatschr. f. Psychiat. u. Neurol.*, Berlin, 1900.—10. RITTI. "Folie a double forme," *Dict. encyclop. des sc. méd.*, 1879; in Tuke's *Dict. of Psychological Medicine*, 1892.

A. R. U.

MELANCHOLIA AND HYPOCHONDRIASIS

SYN.—In the older English writers *Lypemania* and *Phrenalgia* : they also used Melancholia as a collective term for insanity, without regard to emotional exaltation or depression. Fr. : *Monomanie triste ; Aliénation partielle ou dépressive.* Germ. : *Trübsinn*, *Tiefsinn*, *Schwermuth.*

By HENRY RAYNER, M.D.

THE mental disorders in which emotional depression is predominant may be grouped for convenience under this heading. Hypochondriasis is but a pathological variety of melancholia, and, although often dealt with separately, it will be treated here in its *psychological* relation.

Introductory.—The interpretation of melancholia as a morbid mental state is based on the following considerations :—States of consciousness are divided into feeling, ideation, and volition ; but no one state of consciousness can be regarded as pure feeling, ideation, or volition, some element of each of the other two aspects being always present also in greater or less degrees. The greater the predominance of one aspect of consciousness, the less is the share of the others, the more are they inhibited : thus, in intense ideational activity volition is often automatic, and small sensations are unnoticed ; so in excessive volitional activity, as in violent strife, pain is frequently unfelt, and thought is a mere reflex ; in intense emotion, ideation and volition are greatly affected, so that in great joy thought is often incoherent and action purposeless ; in great fear thought is arrested and volition paralysed. Intense morbid emotion has similar results.

Emotional states invariably affect the processes of bodily nutrition ; thus, by fear the circulation may be depressed even to fainting ; respiration is diminished, as shewn in the sighing of grief ; secretion may be lessened, as seen in the dryness of the fauces, and arrest of the secretion of milk ; excretion is affected, as evidenced by the limpid urine ; probably metabolism also is affected, mental shock often being followed by forms of auto-intoxication (by jaundice, for example), and assimilation is interfered with probably by the arrest of the secretions of the internal glands.

In depressed emotional states the power of resistance to external influences is diminished, as manifested in the diseases arising in beaten armies ; and the power of nutritional repair is lowered, as is commonly seen in surgical cases.

The general peripheral sensations, again, especially the visceral, are greatly influenced, as Sollier and others have shewn, by emotional impressions ; and lowered coenaesthesia is asserted by Dumas to be the invariable antecedent of depressed emotional states, whether arising from somatic causes or of mental origin. A local peripheral irritation also may produce marked emotional changes, and these may disappear immediately on the removal of the irritation.

In diseased conditions, especially in the depressed emotional states, this interaction of the mind on the body and of the bodily state on the brain establishes a vicious cycle of nutritional disorder, which tends to increase and prolong the disease.

Definition.—Melancholia, from the foregoing considerations, may be defined as a painful state of consciousness, unrelated or disproportioned to any painful mental impression or bodily condition, of abnormal persistence, and accompanied by excessive restriction or disorder of ideation and volition, and by diminution of the nutritional activities.

Prevalence.—Melancholia is a form of disorder found in a very large proportion of the admissions to asylums in England and Wales; thus, in 1907 the Commissioners in Lunacy reported that 2322 men and 3658 women were admitted with this form of disorder; being 35·7 per cent of all admissions, namely, 24·8 for men and 35·4 for women. In the private asylums these proportions are 26·4 and 39·2 per cent. These figures do not represent the full extent of its occurrence, since a very large number of cases are treated without admission to asylums, and a large proportion of suicides probably occur in early stages or temporary conditions of this form of disorder.

Incidence.—The figures in the preceding paragraph shew that the disease occurs more frequently in women than in men; and more often among the well-to-do than amongst the poorer classes. The reports of colonial asylums lead to the inference that it is more frequent amongst civilised peoples.

Etiology.—*Age.*—Melancholia occurs with increasing frequency as age advances.

Race probably has little influence, as compared with the complex conditions of civilisation.

Religion might be expected to exercise considerable influence, and, although obscured by other conditions, probably does do so. Mohammedanism, for example, with its fatalistic doctrines, ought to have little effect; more emotional creeds should produce melancholia more abundantly; but no statistics are available on this subject.

Occupations have an undeniable influence; those which involve the use of the mind rather than of the muscles, which are sedentary rather than active, monotonous rather than varied, or are concerned with poisons, such as alcohol or lead, furnish more than their quota of melancholia.

Predisposition.—Individual predisposition to this form of disease is found in the children of parents who have suffered from melancholy, or in whom other forms of insanity, or of defects of control, such as intemperance (in alcohol, morphine, opium, etc.), or of neuroses (epilepsy, etc.) are recorded. Phthisis, too, is often found in the family history: a combination of this disease with psychoses or neuroses is especially unfavourable. The physiological conditions of the puerperal state, of lactation, of the climacteric, and of senility favour the occurrence of melancholia; any condition, indeed, which results in prolonged imperfect nutrition or toxic states tends in this direction.

The proximate causes of melancholia are usually of slight intensity but protracted duration. In the predisposed, however, intenser causes of briefer duration may engender it. The history of the case commonly reveals protracted malnutrition, due to mal-assimilation, toxic conditions, or defective rest; these factors being commonly associated, or deriving from one another.

The defective assimilation may result from insufficient or inadequate food, chronic indigestion—especially from bad teeth or defective mastication, organic conditions of the stomach (dilatation, displacement, etc.), protracted ill-health (from exhausting discharges, loss of blood, semen, pus), or other conditions of disease.

The toxic conditions which produce it are usually of slight intensity, such as tippling, insidious poisoning by lead, influenza, or malaria, gout, constipation, defective action of the kidneys or liver, and so forth.

Imperfect repair may result from a long-continued output of energy disproportioned to the strength or assimilative power of the individual; or from protracted mental causes, such as worry, anxiety, disappointment, grief, shame, or general unhappiness; from continued pain, or peripheral nerve irritation, as by a disease of the skin. These last causes are specially operative when they interfere with sleep.

In nearly every case, however, the causation is complex, and hence the disorder is often curable in the earliest stages.

Predisposed persons are characterised by defect of assimilative power, either absolutely or in relation to their nervous activity. Periods of great energy often alternate in them with periods of exhaustion and depression, as is strikingly illustrated in the lives of some eminent men. Functionally, while often possessed of considerable mental power and ability, such persons are irritable and easily exhausted, are often variable and uncertain in their social relations, and are often celibate. Physically, they are commonly slight in build and bone, and with chylopoietic viscera under par. They commonly have dark complexion, eyes, and hair. The teeth tend to decay early, thus exaggerating their weakness of digestion. The stigmata of degeneration are not usually well marked in them, but they are thin even at periods of life when, as Dr. Clouston remarks, "it is physiological to be fat." We often hear in them of night-terrors or sleep-walking in childhood, and of emotional disturbances at puberty.

Pathology.—Melancholia has been regarded by some observers as merely the central result of a vicious coenaesthesia, from somatic abnormalities. This, however, is disproved, at least in those cases in which a single peripheral irritation is the cause. The essential central condition, moreover, is not, at the outset, one implying pathological change of structure, for the symptoms may be marked one day or hour and be absent the next. Moreover, complete recovery may occur even after years of intense disorder. The conclusion may be drawn, therefore, that the essential condition may be one of nutrition, possibly in the nerve-cells, as seen in animals after prolonged stimulation; or be one of lymphatic stasis or altered blood-pressure, possibly from toxic or auto-toxic conditions. In a series of cases examined at the Bethlem Hospital, Dr. Maurice Craig noted a prevalence of increased blood-pressure in melancholia, of decreased pressure in mania. The variations of the disorder probably vary with the area of the brain affected. Thus, to adopt Flechsig's views, we should expect in the hypochondriacal cases to find the sphere of bodily sensibility specially implicated; in those shewing marked volitional disorder, the muscular sphere; and so on.

The chromatolysis of the cortical nerve-cells, as demonstrated by the newer staining methods, the pallor, distortion or displacement of the nucleus, and the varicose atrophy of the protoplasmic processes, found not only in melancholia but also in mania, have pathological significance; but the matter is not yet ripe for discussion.

In view of our great ignorance and of the daily progressing knowledge of this ignorance, it is well at present to abstain from a discussion of the many structural changes that have been described in connexion with these states.

Prodromes.—Melancholia may gradually arise in predisposed persons, in whom it may seem to be but an exaggeration of the natural character. Burton, in his *Anatomy of Melancholy*, quaintly describes such a case. Commonly, however, after exposure to one or more of the conditions already mentioned, for more or less protracted periods, depressed spirits and sleeplessness gradually culminate in veritable melancholia.

Rapid or sudden evolution of the disorder usually occurs only in strongly predisposed persons; and from such intense causes as mental shock, debauch, severe toxic conditions, or acute insomnia.

Melancholia may also be a secondary condition after mania or stupor. It may follow obsessions, hypochondriasis, hysteria, or neurasthenia; and may be associated with chorea, epilepsy, general paralysis, or other organic diseases of the brain.

Degrees.—The *emotional* disorder may range in degree from slight failure of pleasure and interest in life to the most ecstatic dread or horror.

The *intellectual* abeyance may vary from a mere difficulty and sense of effort in thought to the involuntary concentration of the attention on a single idea. The disorder, commonly associated with incomplete attention to all but the predominant idea, leads to defective conception; the judgment is perverted, and errors thus accepted become delusions.

The *volitional* arrest varies from slight apathy to the absence of all volitional effort, and its disorder from mere restlessness to incessant gestures of misery; or phases of quietude may alternate with impulsive activity.

Disorder in conduct necessarily follows, and is especially manifested in neglect of self and circumstances, culminating in impulse to self-destruction.

Variations.—These variations in degree give rise to characters which may be more or less persistent, and may even form distinguishing features of the disorder throughout its whole course. To these and other variations particular names have been applied, and a very profuse and confusing nomenclature is the result.

Melancholia has been divided into the simply affective, without delusion, and the intellectual or delusional. It is more convenient and systematic to consider these varieties, together with the symptoms of the special area of consciousness predominantly affected, in the orders of emotion, volition, and ideation.

Emotional Characters and Varieties.—Every emotion may be affected in every degree of intensity, giving an infinite variety of suffering to what has been aptly described as a "psychologic Inferno."

Thus the attitude to past events may range from regret to bitter remorse; the present state may be one of discomfort or of ecstatic horror, and the future may be regarded with apprehension, dread, terror, or despair.

The dissolution of disease may often be recognised in the disorders of emotion; the highest, most complex, most recently developed, and consequently least-organised feeling being the first to be diminished, perverted, or lost. In mental action, moreover, the higher functions control the lower; and the loss of this higher control may lead to the unrestrained activity of the lower. Thus the representative abstract feelings are usually the first affected in melancholia; and, by the loss of their control, fuller play is allowed to the lower, more presentative feelings. From this it results that the individual lives more and more in the subjective feelings of the present.

The abstract sentiments are often the first to be affected. If the higher aesthetic tastes have been developed, these no longer yield their wonted pleasure; intellectual effort of any kind is painful, and there is no longer satisfaction in acquiring knowledge. Delight in the new and strange is lost early, and even the feelings of surprise and wonder on which these are founded are diminished, so that startling impressions, unless they happen to be in harmony with the predominant morbid feeling, are ignored or excite little attention. In this general defect it is remarkable that the feeling of the ludicrous often persists, and in its lower forms may even be exaggerated. The case of Carlini is often quoted in this connexion.

The moral sentiments, too, are often affected early. There is no pleasure in duty, in honour, or in the contemplation of abstract moral sentiments or of personal ethics. Complaint is commonly made of a sense of moral deficiency and unworthiness, which in extreme cases culminates in a causeless conviction of sin or of crime; the memories of former moral lapses are revived and exaggerated, or evil temptations take possession of the mind. The name *Melancholia moralis* has been given to such cases of delusion; or in which, as sometimes happens, there is moral perversion. The religious sentiments indeed usually cease to yield pleasure, comfort, or exaltation; they bring only misery and depression, regret or remorse, culminating in hopeless despair of this world and of the next (*M. religiosa*). Or again, the sufferer is tormented by blasphemous ideas, or voluntarily gives vent to outrageous language. Sometimes such perversions are intended to shock other persons, or to inflict self-pain. Sympathy, in its higher form, is usually reduced; the suffering or complaints of those around them leave them cold. The contagion of sympathy, the quasi-reflex form, however, is sometimes increased in this sense that the patient may adopt and imitate the melancholic expressions or acts of others if these harmonise with his own feelings. Thus are epidemics of suicide originated. This form of sympathy occurs in children and undeveloped persons; indeed, this reduction is most commonly found in young, uneducated, or ill-developed persons.

The specialised instinctive feelings are usually affected early, especially in persons in whom they were prominent and active. Thus, melancholics continually complain of their loss of regard for their family and friends, and tax themselves with this evidence of their own wickedness. The perversion of these feelings is indeed often remarkable; so that an especial aversion from those nearest and dearest, amounting even to hatred, may be manifested, a perversion which may find vent in bitter accusations, or even in homicide.

The *social feeling* is almost invariably lessened, as evidenced by the avoidance of the company of others, and loss of pleasure in it. The solitariness of the melancholic is well known, and may be noted wherever a number of the insane are congregated. This is sometimes perverted into active dislike, or even hatred, of their fellow-men (*M. misanthropica*), and may even lead to malevolent acts (*M. malevolens*).

The *self-feeling*, expressed in that love of approbation which is perhaps the lowest grade of social feeling, is commonly diminished; in extreme cases all care for the opinion of others is lost, and at times such patients act in a manner which would even suggest that they desire to lower themselves in the opinion of others. The loss of self-feeling is shewn even more forcibly in the neglect of lifelong habits, and in the abandonment of cherished ideals, aims, and ambitions. In the slighter forms of mental depression the physician has often to guard the patient from giving up occupations, pursuits, or positions which on recovery would be bitterly regretted. The *egoistic self-feeling* is invariably depressed early. The melancholic usually complains of his sense of unfitness for his work or duties, even while still performing them satisfactorily to others; of his loss of all feeling of satisfaction in his actions or belongings, and of care for personal appearance or health. Perversion of self-feeling may culminate in self-loathing or hatred; and very commonly in loss of the instinctive desire to preserve life (the fundamental self-feeling), resulting in neglect of health, or even in self-mutilation and self-destruction (*Suicidal melancholia*).

The feelings which have *anger* as their fundamental emotion are markedly affected. The melancholic is no longer indignant at moral wrong; combativeness is lost, and submission is often made with meekness and humility to events that in health would have been strenuously resented. In the earlier stages this is often recognised and complained of by the sufferer. Perversion commonly manifests itself in grumbling, discontent, and dissatisfaction; but occasionally it grows into vindictiveness, usually in persons who believe themselves acted upon by external influences, especially if associated with illusions and hallucinations.

Fear, which is perhaps the most fundamental of animal emotions, is always present; and, as a rule, the greater the defect in the higher areas of emotional feeling, the more prominent does fear become. Fear may vary from a mere loss of moral courage or timidity, from vague feelings of apprehension of impending calamity, even to panic terror. Fears in ordinary states of melancholia are not usually persistent; but in some cases (commonly of slowly advancing depression of no great intensity) they become fixed, may persist for months and years, and may dominate the conduct of the patient more or less; although he may recognise that they are unfounded. The variety of these fears is very great, and the records of the subject have been greatly encumbered by the invention of innumerable names to describe them; the tendency being to make each name into a disease. Nothing could be more absurd, for in the early stages these fears often coexist, and they vary from time to time. Moreover, if the basis of the fear be inquired into, very various explanations are given. Thus, in claustrophobia, whilst one patient fears that the room will close in upon him, another fears that other dangers may befall, and others again fear that they themselves may become mischievous. It is sufficient to say that fears may be associated with every sensation, thought, or action; and that such names as claustrophobia and agoraphobia are superfluous (*vide* also p. 749).

Volitional activity is inhibited in degrees varying from a mere distaste for doing or deciding to an absolute inertia. When the motor volitional arrest is extreme the sufferer may sit or stand in one position for hours, shunning all efforts of will; the attitude and facial expression usually being that of hopeless dejection: even speech may be in abeyance. When such patients do move, by their own volition or by the impulse of others, their motions are slow, heavy, listless, and spiritless; the speech is often monosyllabic, a whispered monotone. These quiescent cases usually yield without resistance to the impulse of others, and for such cases the name *M. passiva* has been used.

The name *M. cataleptica* has been applied to cases in which this passivity is accompanied by a certain amount of "flexibilitas cerea"; but where this is present there is usually some degree of stupor, a condition which may shade off into *Mélancholie avec stupeur*.

M. attonita.—In another group of cases the volitional arrest is equally complete, but the attitude is fixed and rigid, expressive of acute, painful emotion (often of remorse or dread); this is the form to which the name

M. attonita is properly applied. A rare, extreme state of this form is that in which the patient stands upright with upturned eyes in the attitude of ecstatic terror or horror, and cannot be made to lie down (*M. ecstatica*).

The passive patients, but especially those suffering from the attonita form, instead of yielding to the impulse of others may resist every attempt to impel them to move, and to such cases the term *M. resistiva* has been applied; in some instances this resistance may become violent and even aggressive. This resistive form, when complicated with convulsive attacks, has been called *M. convulsiva*; in such cases there is usually also some degree of stupor.

The arrest of volition (aboulia) is often seen in an inability to decide or to choose, and in others in an inability to carry out an act when decided upon. The volitional disorder may vary from a vague, purposeless restlessness, leading to a tendency to wander (*M. errabunda*), to a fixed unreasoned desire to get away from the place in which the patient happens to be. When the emotional pain is more acute the disorder is expressed in involuntary gestures, cries, or exclamations, these being characterised by their repetitive monotony. To such cases the name *M. agitans* or *agitata* has been applied. The motor restlessness may vary from a mere biting of the nails or wringing of the hands to the most frantic gestures; the exclamations varying from a mere moaning, or a repetition with monotonous iteration of some phrase, as "Save me, save me!" "Don't shoot me," "Don't burn me," "What have I done?" to frantic outcries. Often such sufferers pace to and fro like caged animals, with a facial expression of the most intense misery. The active expression of terror or horror in these cases markedly contrasts with the dejection of the passive cases. Commonly they pluck out their hair, or rub their skin into sores. In its most extreme and acute form it may even rise to the height of maniacal excitement, and merits the name *M. maniaca*; some patients remain chronically excited (*M. excitata*), and may be aggressive and homicidal; usually they are actively suicidal, preferring violent means.

In other cases controlled quiescence is associated with occasional uncontrolled, impulsive activity (*M. impulsiva*). The acts may be entirely unpremeditated, being apparently suggested by some impression upon the senses, as in the breaking of fragile objects; or by vague resentments or antipathies, as in kicking or striking their associates. In others the impulses may be persistently of one kind, such as the expression of foul, obscene, or blasphemous ideas (coprolalia); or the impulse may be to suicide or homicide. The impulse may be complained of and resisted, or it may be wholly uncontrolled.

The volitional manifestations vary and alternate in most cases, although they sometimes become fixed, and form the predominating character. Most patients are usually more quiescent after meals, and many are less controllable by night than by day.

Dr. Stoddart has asserted that motor rigidity is present in all cases, especially affecting the proximal groups of muscles, the peripheral being least affected. This statement, however, requires verification; it is very difficult to estimate and compare the degree of resistance in groups of muscles of such very different power.

Ideational Varieties.—Intense emotional and motorial disorder may exist for long periods without producing such disorder of thought as to result in delusions; and the protracted resistance offered by well-developed minds is strongly contrasted with the feeble resistance of the youthful, ill-developed, or inherently weak minds.

Attention is the earliest process to be affected. This, in its higher voluntary form, has a large motor element, the sense of effort; in depressed states this sense of effort becomes painful, and is early complained of, as it destroys mental pleasure and the sense of capacity. Thus the higher intellectual efforts are shunned, and there is a tendency for the mere reflex attention to the perceptions and feelings of the moment to predominate. Hence complaint is often made of inability to divert the attention from bodily sensations, passing events or impressions, business or family matters, in endless reiteration.

Hypochondriasis.—The arrest of attention on the bodily sensations, which constitutes hypochondriasis, may arise from the mere habit of self-attention, from actual bodily disease, or from central causes. It may vary from a mere unreasonable fussiness about health to the most extraordinary delusions, with intermediate stages of doubt (*Folie raisonnante mélancholique*).

The attention may be fixed on some local sensations, or may exaggerate every possible sensation, even recognising those bodily functions which ordinarily are unconsciously performed (for example, intestinal peristalsis or vascular changes). Several varieties arise in this way. The most common is the predominance of attention to the sensations of the digestive tract: the attention to deglutition often makes this process less easy, hence a notion of obstruction or even destruction of the stomach; or the sensations accompanying gastric and intestinal digestion are felt excessively, and are variously interpreted. The bladder, too, is usually associated in this hypochondriacal attention. Commonly a painful sense of depression in these cases is referred to the epigastric or hypochondriac region. Self-homicide may result in these cases from attempts by operation to relieve some imaginary condition of the bowels or bladder.

In *head hypochondriasis* (which, as Dr. Savage says, might rather be called *brain hypochondriasis*) the attention is fixed on sensations in the head. Sensations accompanying vascular changes would appear to be the basis of most of these: sensations of weight, pressure, want of pressure, hollowness, emptiness, lightness; with delusions of destruction, loss, or decay of the brain, in endless variety. In some the attention is concentrated on the mental operations which, by the very act of attention, are rendered difficult or impossible.

In *sexual hypochondriasis* attention is concentrated on the genital organs, and the mind is occupied by fears respecting their state—fear of impotence being predominant. These patients commonly also have illusions in regard to the bearing of others towards themselves, believing that aversion to be manifested in words or gestures. If such an one form a matrimonial engagement, his fears are often exaggerated, even to suicide or acute mental disorder. This form is much more common in men.

Nostalgia, as it especially affects the inhabitants of marshy and mountainous regions, is characterised by melancholic attention to sensations in the pneumogastric nerve area; it has been suggested that this is due to the loss of the habitual lung sensations, arising from the extremes of atmospheric pressure to which they have been accustomed. Home-sickness, which rarely passes beyond depression, is probably due to the sense of loss of habitual social stimulations. The Japanese are said especially to suffer from it.

The expectant attention induced by melancholic ideas generates illusions, and probably also hallucinations, which are always simply the expression of these central ideas.

Delusion.—The limitation of attention to the feelings and perceptions of the present tends also by repetition to strengthen them, and thus more and more to exclude the higher representative elements of thought; the subjective elements thus becoming more and more predominant. The patient, conscious of his changed feeling, at first may recognise it as due to his state of health; but if the change is more marked he may endeavour to account for it by some external cause, some bodily disease, or moral defect. He is constantly endeavouring to explain the changed self-feeling: for example, self-dissatisfaction passes on to a conviction of wickedness or criminality, then to the idea of a special form of criminality, or, in an extreme case, to the conclusion that the "ego" is changed, and that the self-feeling is that of the "devil" or other embodiment of evil; the reversal of this process may also be commonly observed in the devolution of delusion. The false conclusions, however, do not usually command belief at once; in many conditions they are doubted, although they cannot be dismissed; or disbelief or doubt alternates with belief. It is to be borne in mind, too, that, in melancholia, belief is often strenuously impressed upon others, and a portion of erroneous belief sometimes lingers from habit, or at times even from obstinacy.

The longer the false conceptions exist, and the more often they are repeated, the more firmly do they become organised; so that a mere apprehension of some noxious influence in the environment becomes a definite conviction of persecution, and later assumes the systematised form of persecution by particular persons or associations for very definite ends.

The delusions vary with their origin; if attention has been directed from the outset to the bodily sensations, as in hypochondriasis, they are usually expressive of some change of sensory impression: the delusion may be that the body is brittle, or made of iron; that the sufferer is transformed into an inanimate object; that he is increased or diminished in size; is dead; that his internal organs are destroyed or putrid, and so on. Delusions of this kind frequently occur in ordinary melancholia, especially in epileptics, in the senile cases, and in general paralytics. In the two latter classes we see especially delusions concerning the digestive tract; for instance, that the throat, stomach, or bowel is destroyed. In such cases, too, the peristalsis of the bowels, being morbidly perceptible, is ascribed to the movements of animals; the rumbling of flatus to noises made by animals. Sounds of the heart or of the veins may likewise form the basis of illusions.

Morbid impressions of the special senses may, it is said, result in the delusion that the self is changed (for example, that the natural self-feeling cannot be recovered), or in the belief that the whole world is changed, often as a result of their evil state. It has been suggested that some morbid condition of the muscular adjustment of the eye or ear, by some change in the perception of the environment, may account for this, but there is no proof of the suggestion.

When the external attention is governed by intellectual delusion, the expectant attention leads to confirmatory illusions to the effect that the acts, gestures, or words of those about him refer to the patient; or illusions of smell and taste may arise, especially that food is putrid or poisoned. Delusions of purely emotional origin usually correspond to the emotional habits of the patient, the description varying with his intellectual character. Thus, in advanced age the delusions are especially likely to be concerned with property, such as pecuniary ruin; in later middle life, that character and reputation are destroyed; in early manhood, or in women, that the family is ruined, and so on; or if religion, politics, or society formed the leading interest of the normal life, these subjects form the basis of the delusions. Yet stranger delusions arise from the realisation of dreams; others again from adopting the delusions of companions (*folie à deux*).

Symptoms in Conduct.—The melancholic, although shunning society and accustomed pleasures, may for a time continue to perform the habitual occupation or routine of duty; but ultimately he neglects even work and duty, and remains unemployed, brooding, and solitary; or he may wander aimlessly from home. Personal appearance and cleanliness are neglected; but it is only in very extreme cases that the calls of nature are not habitually attended to. Lastly, even self-preservation is forgotten; heat and cold pass unnoticed, food is neglected or refused, and very commonly attempts at self-destruction or self-injury are made.

Suicide.—The suicidal impulse of melancholia must be suspected in every case. In many the idea of self-destruction arises, but is firmly controlled; in others the desire to commit the act is openly avowed and attempted; in others again the desire is cunningly hidden, and the utmost ingenuity exerted to carry it out: this proclivity is usually the result of delusion, but not infrequently of a blind unreasoning impulse; the means of committing suicide may suggest the act; or again it is a persistent volitional obsession.

The passive forms of melancholia favour the less violent means of suicide, as by starvation, poison, drowning, and strangulation; the more restless patients incline to violent means, such as precipitation, burning, or throat-cutting.

The monotony of ideation in melancholia is sometimes apparent in the very refusal of other ways of self-destruction—in the pursuit of a chosen method; for example, one melancholic swam across a canal to throw himself under a train.

Self-homicide occasionally occurs in melancholia, as in patients who have died from the effects of self-mutilation (plucking out eyes, castration, etc.) without suicidal intent—perhaps, indeed, in the endeavour to escape from imaginary dangers.

The suicidal impulse is reported to exist in a more or less pronounced form in about 56 per cent of the cases admitted to English asylums; it is more frequent in men than in women, and it increases with advancing age.

Causal Characters.—Special characters occur in connexion with special causes. Thus, melancholia in the young, if associated with hysteria or masturbation, often leads to exaltation, which may take the form of expansive ideas of philanthropy and social effort, or of personal prowess. The melancholia of the puerperal state is said to be frequently accompanied with perversion of the affections; love of husband and offspring may be replaced by aversion, or even by hatred and homicidal violence. Climacteric patients are said, more frequently than others, to have lustful delusions, dreams, or impulses.

Gout, rheumatism, and arthritis give to melancholia a peculiarly irritable, querulous character, manifested by complaints and dissatisfaction, sometimes culminating in delusions of injurious treatment. Phthisis, again, may be accompanied by great querulousness, complaining, discontent, sullenness, and irritability—a reversal of the "spes phthisica." Disease of the aortic valves and mitral valvular incompetence are said to be accompanied by a predominance of anxiety and apprehension, together with irritability and querulousness (*vide* Vol. VI. p. 475).

The melancholy associated with general paralysis is commonly marked by great exaggeration; that accompanying epilepsy is usually impulsive, and sometimes aggressive.

Aberrant Cases.—The course of the disorder gives rise to many varieties; it may be *remittent*, if intervals of sanity alternate with derangement. These remissions may be more or less complete, and the alternations may be of a day's or a week's duration. *Recurrent* is the term applied to cases in which the remissions are far longer and the periods less regular, as in cases which recur at special periods of the year, with the recurrence of pregnancy, and so forth. The daily, monthly, and yearly cycles of nutritional habit may be the foundation of some of these cases. Change of diet and habits, induced by season or occupation, may act in this way.

The name *mania à double forme* has been applied to cases in which mania alternates with melancholia; the alternations being daily or of longer duration. Stupor occasionally alternates with melancholia; and in circular insanity the patient again and again passes gradually from mania to melancholia: the circuits in a given case usually occupy a wonderfully uniform period.

Alternation is the term applied when the disorder alternates with the activity of other diseases. Alternation has been recorded in glycosuria, albuminuria, exophthalmic goitre, phthisis, gout, and asthma.

Bodily Symptoms.—*Nutrition* is usually very defective. There is often loss of weight in the prodromal stage, especially if there be much sleeplessness. The decrease of weight is sometimes very rapid, and gives a serious aspect to the case. The muscles are diminished in bulk; but the loss is especially in fat, even in the hard fat of the temporal region. Wounds or injuries are repaired slowly, and are readily influenced for the worse by exposure to cold.

The *skin* becomes dry and harsh, and is often sallow from pigmentation, which occurs in patches, and of various distribution. The hair is commonly harsh, dry, brittle, and consequently untidy; and greyness increases rapidly in melancholia. The nails are thin and friable, often shewing transverse furrows; in restless cases they are commonly broken or bitten down to the quick.

The *blood* in many cases (50 per cent) is deficient in red corpuscles and haemoglobin. In some, the red corpuscles are normal in number, the haemoglobin being in defect. The circulation is enfeebled. There is a tendency to venous fulness, dependent parts easily become congested, and the extremities are often cold. The pulse in acute conditions is quick, and is easily quickened by stimulation; the sphygmographic tracing usually indicates a feeble systole.

The *digestion* is usually disordered. The teeth are commonly defective; the breath offensive and even fetid; the tongue large, flabby, and indented by the teeth. In restless patients the tongue is smaller. The stomach generally gives evidence of delayed digestion or even of dilatation, and may contain much alkaline or acid mucus. The bowels are almost invariably sluggish or constipated. The motions are dry or scybalous, and contain little bile. The appetite is usually bad or altogether in abeyance; or there may be the most strenuous refusal of food. The refusal may be based on sensory delusions in regard to the character of the food (of its being human flesh, poisoned, etc.); or on a supposed state of the throat, stomach, or bowels; on emotional delusions or hallucinations; on mere want of volition, or on the intention of committing suicide by starvation. In a few cases, however, the appetite is even excessive; or occasionally a patient will eat enormously with the intention of self-injury.

Menstruation is usually diminished or arrested; but metrorrhagia may occur, and commonly the mental condition is worse at these periods. The sexual appetite in both sexes is usually reduced or absent, but eroticism and self-abuse may be associated with the condition.

The *reaction time* in the majority of cases of melancholia is distinctly prolonged.

Subjective Symptoms.—The commonest bodily feeling is that of "sinking" in the precordial or epigastric region; it is very variously described. Abnormal sensations about the head — of weight, pressure, fulness — occur in great variety, but rarely amount to actual pain.

Sleeplessness is usually a well-marked and troublesome symptom, especially in the early stages of the disorder, when also it is very closely related to the intensity of the suicidal impulse, and to the refusal of food. Dreams are often horrible and terrifying, patients sometimes saying that they fear to fall asleep. The dreams of melancholics are probably often the sources of delusion, especially in cases of *hysterical origin.*

Progress and Terminations.—In very acute recent cases recovery may occur almost suddenly; but such cases are very apt to relapse. When suicidal impulse has been well declared, a sudden apparent recovery suggests simulation.

Recovery is ordinarily steady and gradual, but occasionally the improvement is intermittent. Improvement usually follows better sleep, resulting in better nutrition, increase of weight, more active circulation, and brightening complexion. In women the menstrual function becomes more normal. Mental improvement is usually first manifested by increased power of attention to the environment, so that occupation is gradually resumed. Attention to cleanliness and personal appearance returns, and is followed by return of affection and social feelings, and a gradual restoration of the habitual interests and pursuits. Delusions gradually diminish; the belief, for example, of being a devil falls to one of ordinary criminality or wickedness, and doubt begins to alternate with belief.

Convalescence is sometimes attended with a certain degree of mental torpor and sluggishness, and at times there is even some exaltation, the patient becoming for a time brighter and more active-minded than in his ordinary condition.

In less favourable cases the condition may persist for many years, and yet end in recovery; even after twenty years. In others the disorder passes on into stupor; in others into mania, either by rapid or gradual transition; in others again it loses its emotional character, and becomes delusional. Uncomplicated melancholia, however, tends but very slowly to dementia, that bourne of all chronic insanity.

Death in melancholia, apart from suicide or self-homicide, may occur from exhaustion, extreme sleeplessness, restlessness, or refusal of food. It is often ushered in by a low form of pneumonia.

Prognosis.—Melancholia justifies a more favourable prognosis than other forms of insanity. More than 50 per cent of those admitted to asylums recover; of these Dr. Clouston estimates that one-half recover within three months, and four-fifths within the first year.

Recovery occurs after very long periods (even twenty years), and after intense forms of the disorder (even after three years of stomach-tube feeding). Recovery is often very complete, so that the highest and most active brain-work may be performed; and persistent, so that patients may live a long subsequent life without a relapse.

Chronic uncured melancholics, too, are often long-lived, and in complicated cases tend very slightly to dementia.

Death in the acute stage is rare, unless from bodily disease. Dr. Clouston estimates its frequency at one per cent, but this is probably below the average.

No intensity of melancholia should deprive us of hope, so long as the delusions are not systematised, and there is no incurable bodily disease.

The conditions which make for chronicity are a marked heredity, developmental stigmata, associated neurotic disorder, prolonged action of the proximate causes, slow evolution of the disorder, extreme youth or senility, and habits of self-abuse.

The unfavourable mental symptoms are rapid systematisation of delusions, especially with hallucinations; and the association therewith of loss of memory, incoherence, or mental confusion. The restoration of nutrition without corresponding mental improvement is of bad omen.

The prognosis in cases of mental depression not amounting to certifiable melancholia is chiefly based on the association of hereditary psychoses or neuroses, and on the degree of disturbance of sleep and general nutrition. In the absence of these, the neurasthenic, the hysteric, the hypochondriac, climacteric, and depressed states may persist for a long time without leading to suicidal impulse or delusion.

A bad sign in climacterics is the excessive growth of the secondary sexual hair.

Treatment.—The causes of melancholia are usually complex: rarely is a case found in which two causal conditions, at least, are not present, more commonly several in combination.

In the early stages the removal of a cause will often arrest the progress of the disorder; although when the disorder is thoroughly established the removal of a cause may have little apparent effect. The fully developed disorder, by the interaction of the cerebral and the bodily conditions, tends to its own prolongation. Hence those who have to deal principally with well-established disorder are apt to be sceptical of the effect of removing proximate causes.

The first point in treatment, therefore, is to relieve all unhealthy bodily conditions, such as disorders of mastication, indigestion (stomach dilatation), constipation, troubles of the sexual organs, of the organs of sense; thus an exhaustive examination of every organ and function is necessary. The habits of life require no less rigid a scrutiny and attention; especially on the mental side, in regard to sexual, domestic, business, social, and religious worries.

Mental Treatment.—The arrest of attention in melancholia is a good measure of the intensity of the disorder, and a good test of progress. By the diversion of attention—where this is possible—to simpler modes of mental action the functional expenditure can be definitely and systematically lessened; such a diversion probably implies comparative rest in those cerebral structures whose nutrition is specially affected.

The reduction of the emotions in melancholia to their simplest modes of expression must be remembered, and the painfulness of premature attempts to revive the higher forms of mental feeling should be avoided. The stronger the fixation of the attention in a given case, the lower in the scale of mental effort must we descend in our attempts to divert it. Where no voluntary attention is possible our efforts must be limited to mere sensory diversion. The appeals to sensory attention may be effected, in common sensation, by the various methods of massage; by cutaneous faradisation, or franklinisation; and by hydrotherapy, in which, by variations of warmth and force of jet, pressure and temperature sensations are added to those of touch. The hot-air and vapour bath, the wet pack, and similar means act also in the same direction. Monotonous sounds were recommended by Burton, such as the sound of falling water. The diversion of attention by these means, though incomplete, is sometimes demonstrated by the production of sleep. A more powerful means of diversion is found in passive sensory impressions involving the lower forms of emotional feeling; for example, of familiarity or novelty, as in viewing new or familiar scenes, or in listening to music.

Volitional diversions of attention (occupation or games) must also be employed whenever practicable; and in using these we should ascend from those that are merely motor and almost automatic to those demanding sensory attention also: we shall then advance to those involving combined intellectual, sensory, and motor effort; and, lastly, to purely intellectual work. By thus arranging occupations in the order of the functional expenditure involved, the estimation of their value is more easily made; as well as of the extent to which they should be employed.

In cultivating the higher forms of volitional occupation patients should be taught and encouraged to break off at the first feeling of imperfect attention, resuming it after a few moments of muscular exercise brisk enough to stimulate the circulation. In this way the duration of attention may be gradually extended from a few minutes to hours. The greatest efforts, too, should be made in the earlier part of the day. After the last meal the attention should be diverted by the most automatic of occupations, or by mere sensory diversions.

The attention in melancholia is often directed to some special area of consciousness—to the entoperiphery, the epiperiphery, to the mental operations, or even to the volitional side; and benefit may be obtained by diverting attention from the affected to the comparatively unaffected areas. Thus, where the attention is entoperipheral, as in the hypochondriacal, its diversion should be made by motor and intellectual appeals. When, on the other hand, the attention is concentrated on the environment, appeals to the motor side are especially indicated. The hallucinated insane are amongst the most industrious of our patients, and often seem to obtain relief in incessant occupation. Where introspection predominates, appeals to sensory attention and motor activity are specially indicated; such patients being much benefited by change of scene and variety of surroundings. The whole of the patient's time, as far as practicable, should be occupied by such attempts to divert his attention by occupation, amusement, and therapeutical methods. Melancholics left to their own thoughts are assuredly beating the mill-round of their disorder deeper and deeper.

Changes of circumstance, with the passive influence they exert in directing attention and otherwise affecting health, demand a separate consideration. The stereotyped course that all melancholics should be removed from home must not be too readily accepted. Like every other means of treatment, it should be adopted only for good and sufficient reasons. Unless these are present, patients in the early stages can often be treated to advantage at home, until they are sufficiently improved to leave without suffering from the disadvantages which such a change commonly entails. Where the presence of certain relatives constitutes the drawback to home treatment they may perhaps be removed.

Change from home, travelling, asylum treatment, and the like turn much on the pecuniary means, the habits and experiences of the patient, the condition of bodily health, the mental state, and the prognosis—questions so complex that each decision must be arrived at on its own merits.

Those patients whose attention can still be controlled may sometimes, therefore, be treated without removal from home (when other means of treatment can be added), by the revival of accustomed or even novel occupations and amusements.

When the attention is preoccupied by domestic and family troubles or worries, change under careful supervision from home, family, and friends becomes absolutely necessary. The effects of a simple change of place should, as a rule, be tried, before venturing on the complex changes of travelling, or committing the patient to prolonged unalterable conditions, as on board ship. The danger of sending patients to travel without preliminary trial of the effect of change is very great. We must be assured that noisy hotels, irregularity of sleep, unaccustomed or unsuitable food, and the worries of travelling will not affect the patient unfavourably. However able the travelling companion, it is difficult to save the patient from some of these injurious influences, and from questions and decisions which are too trying to him. Incipience and convalescence are the proper stages for this remedial measure.

The social change may be limited to the companionship of a carefully selected person, medical or lay; to the society of a single household; or, in slighter cases, to the larger society of a boarding-house, hotel, hydropathic institution, or spa. Usually the social surroundings may be advantageously increased in this order, the frequency of change of place being regulated by results. Among the poor little can be done in this direction, and consequently a resort to more stringent means of supervision is often necessary in them, even in an early stage.

When the attention is volitionally directed to the self-feeling, when there is uncontrolled expression of the predominant feeling, delusive idea, or impulse, constant supervision and control become necessary, either in an asylum or suitably arranged home, with a much greater monotony of environment. The form of supervision is of course mainly dependent on pecuniary means; but that method is always to be preferred which brings to bear on the patient the greatest amount of skilful and individual attention. The influence of the discipline and routine of a large asylum is undoubtedly beneficial in later conditions, but in the early stages individual attention and consideration are all-important. On the care and skill brought to bear on the early stages of the disease much of the result of the treatment depends. Attendants and companions should be chosen, therefore, with very great judgment, and, if found unsuitable, changed without delay. As a rule, the more intelligent the companion the better; but the physician has no nicer duty than the selection of the person best fitted to deal with a given case.

In the treatment of melancholia the first step is to gain the confidence of the patient. The most careful and exhaustive inquiry into the feelings and ideas of the sufferer is absolutely necessary. Until the fundamental melancholic idea is discovered complete confidence is not gained, and the physician's personal influence for good is greatly weakened. In the earlier stages of the disorder this influence is of the utmost importance. With the complete confidence of the patient reason and volition may often be actively enlisted, and the suggestions of the physician under these conditions have a great influence on the progress of the case. Hypnotic suggestion can rarely be employed in melancholia, and should be avoided, since by it volition is enfeebled rather than strengthened. The confidence of the patient must be retained by absolute truthfulness, by sympathy and firmness. An atmosphere of persistent hopefulness is not without its effect on these patients, strongly as they may deny its influence.

Arrest or recovery in melancholia sometimes follows physical or mental shock, or the excitation of strong emotions. Thus sympathy for friends in sickness and trouble, or the pressure of a real anxiety, will sometimes result in cure. Emotional influences, however, can rarely be brought into play therapeutically. Religious emotion has been tried, but in my experience it is a dangerous means of treatment. Fear also, although at times successful, is a double-edged instrument.

Against established delusions direct argument is hopeless, harmful, and to be avoided; those founded on illusion and hallucination may, however, be excepted. In the stage of doubt, whether in the inception or retrocession of delusion, reasoning and kindly ridicule, especially when indirect, are not without their effect. Fears and obsessions in the early stages may be combated by systematic volitional efforts made at the suggestion of the physician.

The degree of *rest* to be employed is of the utmost importance. Rest in bed at the outset has been recommended in all cases; but some patients are not thus benefited, and the point must be decided on the merits of each case. Rest in bed is indicated in acute cases with great nutritional disorder, when great insomnia, sitiaphobia, or suicidal impulse is present, but not isolation; on the contrary, companionship and amusement are to be provided, and massage if the rest be protracted. Yet in cases of extreme sensory irritability rest in bed with isolation may be indicated. Erotic persons, or those having sensory hallucinations or illusions, are not to be kept in bed if it can be avoided.

The amount of *exercise* taken should always be carefully regulated, for actual fatigue will often produce relapse. In the early stages, and wherever it is possible, muscular drill is of the greatest service at the outset; later, a carefully regulated exercise must be taken,—walking, golfing, cycling, boating, or riding, as the circumstances permit.

The melancholic should spend as much time in the open *air* as possible, consistent with the avoidance of fatigue; the best hypnotic is a day in the open air.

Physical Treatment.—Attention to digestion is the first point. The teeth should be put in order, as bad mastication and the swallowing of contaminated saliva are very detrimental. The stomach should be examined for retention of food; and in many cases of loathing or refusing of food lavage may be used with advantage, hyperacidity, or the reverse, being neutralised by the wash.

Food should usually be in excess, in the young even largely so. If there be obviously much gastric irritability and disorder, a milk diet may be indicated; but the diet may vary from this to the choicest productions of the kitchen.

Alcoholic beverages may be needful for young adults; but in other persons, whose excretory organs are enfeebled, they must be used with caution or not at all.

Refusal of food may often be overcome simply by putting the food in the mouth with a spoon; in other cases a feeder (the nose being held) is sufficient; but at times forcible feeding by the nasal or stomach tube, with chalice or pump, is necessary. The nasal tube has the advantage in cases where the resistance is very strenuous; but there is more danger of passing fluid into the air-passages, and only very liquid food can be given by it. The stomach-tube, passed into the stomach (there is no special advantage in one shorter than this), has in its favour the greater rapidity and the greater variety of nutriment. The resort to forcible feeding should never be delayed when refusal is once definite; it may be practised from two to five times a day, the intervals not being less than three and a half hours. Pounded meat, freshly made beef tea, milk, eggs, pulped cooked vegetables and fruit, in their due proportions, should form the basis of the feedings, which may be peptonised, or pancreatised, if indicated; and lime juice, alcohol, or medicines may be added if necessary. Rest in the horizontal position after each feeding is sometimes required to prevent vomiting, whether involuntary or self-induced. Rectal alimentation becomes necessary in extreme cases, and in this resort defibrinated blood has been found useful. Constipation must be treated by diet and the usual remedies; if allowed to persist, it increases the malady.

Sleeplessness.—Sleep is usually in defect in melancholic states, especially in the earlier stages; and a most important question arises in regard to the use of narcotics and sedatives. The habitual use of narcotics is well known to destroy the normal habit of sleep, and to result in various nervous and mental disorders. Moreover, the state of unconsciousness produced by narcotics is not physiologically identical with normal sleep; and it is difficult to believe that the addition of a toxic condition to an already disordered state of nutrition can be curative. Many competent observers, however, recommend and use them. My own observation and experience tell me that nothing is more prejudicial than their use in melancholia, and that only in exceptional conditions is even the most temporary resort to them justifiable.

Local Treatment.—In many cases there seems to be an over-fulness of the cerebral venous system and probably a lymphatic stasis; and I believe that patients in early stages have relieved this condition and have obtained benefit by frequent deep inspiration, the hands being brought down over the large veins of the neck at the same time so as to empty them.

The application of cold to the head by douches and by alternations of heat and cold is distinctly beneficial. A jet of cold water over the cervical sympathetic is often of use. Shower baths in the young, when they can be borne, are most efficacious.

Galvanism of the head offers great promise of good, but as yet no satisfactory account of its systematic use in a large number of cases is on record.

The general treatment must be directed to keeping the skin, kidneys, and bowels in free action. The action of the skin should be promoted by hot-air or vapour baths, and warm baths; also by daily friction with rough towels or brushes. Active muscular exercise, however, is the best means when this is possible. Both skin and kidney action should be promoted by copious watery fluids, taken, if possible, apart from meals.

Tonics and stomachics may be given to promote appetite and digestion; iron is very commonly indicated, and at times phosphates and phosphorus appear to do good.

Intercurrent febrile conditions have often been followed by recovery in cases which had become stationary; and, in imitation of this, treatment by thyroid extract has been tried with some success.

Associated diseases, such as gout, must of course receive their appropriate treatment, which often leads to amelioration or removal of the mental disorder.

Henry Rayner.

REFERENCES

1. Allison, H. E. "Simple Melancholia and its Treatment," *Med. Rec.*, N.Y., 1897, ii. 39.—2. Browne, Sir J. C. "On Dreamy Mental States," *Lancet*, 1895, ii. 1, 73.—3. Brush, E. N. "An Analysis of One Hundred Cases of Melancholia," *Brit. Med. Journ.*, 1897, ii. 777.—4. Dheur, P. "De l'état de sensibilité chez les mél.," *Gaz. hebd. de méd.*, Paris, 1897, n.s. ii. 78.—5. Dumas, G. *Les États intellectuels*

dans mélancholie, Paris, 1895.—6. Ferraud. "Sur la mélancholie," *Bull. acad. de méd.*, Paris, 1896, 3e s., xxxvi. 518.—7. Gasquet, J. R., and J. A. Cones. "Age in Relation to the Treatment of Melancholia," *Journ. Ment. Sc.*, 1897, xliii. 483.—8. Greenwood, H. H. "Lavage in Refusal of Food of the Insane," *ibid.*, 1898, xliv. 62.—9. Halloverden. "Über Heilungsvorgänge besondere bei Melancholie, klinisches und theoretisches," *Allg. Ztschr. f. Psychiat.*, Berlin, 1896, liii. 203.—10. Kiernan, J. G. "Quebracho in Melancholia," *Al. and Neurol.*, St. Louis, 1896, xvii. 155.—11. Mesnard. "Leçons cliniques sur mélancholie," *Ann. de la policlin. de Bordeaux*, 1895-96, iv. 357.—12. Mitchell, S. W. "An Analysis of Three Thousand Cases of Melancholia," *Journ. Nerv. and Ment. Dis.*, N.Y., 1897, xxiv. 738.—13. Paraut. "La Tremblement gélatineux de la langue chez les aliénés mélancholiques," *Ann. méd.-psychol.*, Paris, 1896, 8. s., iv. 372.—14. Pritchard, W. B. "The Diagnosis and Treatment of Melancholia," *N. York Policlinic*, 1896, vii. 70.—15. Schloss, H. "Über die Beziehungen zwischen Melancholie und Verrücktheit," *Jahrb. f. Psychiat.*, 1895, xiv. 114.—16. Serieux, P. "Le Traitement des mélancholiques par le repos au lit," *Rev. de psychiatr.*, Paris, 1897, n.s. 194.—17. Stewart, N. "Convulsive Melancholia," *Med. Rec.*, N.Y., 1897-8, i. 265.—18. Tuke, Batty. *Insanity of Over-exertion of the Brain.* Edinburgh, 1896.—19. von Krafft-Ebing. "Über Melancholie," *Allg. Wien. med. Ztg.*, 1895, xl. 247.

H. R.

MENTAL STUPOR AND CATALEPSY

By G. H. Savage, M.D., F.R.C.P.

Stupor is used to indicate those mental disorders which serve as links between dementia and melancholia.

In cases of true stupor there is suspension of mental action, but not its destruction. The supply of nervous force seems to be cut off, but can sometimes in the earlier stages be turned on again under certain conditions. It is often impossible to distinguish between cases of simple stupor and those of melancholia or dementia.

Stupor varies in degree, and consequently differs in appearance. Generally it must be looked upon as a defective power of reaction to ordinary surroundings. This defect may be due to suspension and not to want of nerve power; it also differs from suspension of mental functions due to some dominating idea. It is a state of mental confusion with want of will. In many cases little else than want of will power can be observed; and it is in such cases that the separateness and special work of the will are perceived. Stupor may be primary or it may be secondary.

There is still difference of opinion whether there be such a disease as acute primary dementia, apart from stupor; and certainly the same state has been described by authors under the two names.

There are three groups of symptoms which produce almost the same appearances. First, there is melancholia attonita, or melancholia with stupor, in which shock—physical or moral—has arrested all mental expression; in such cases there is a form of prolonged panic, with a dominating idea or feeling; secondly, there is a form of dementia in which there is no dominating idea or feeling, but in which there is complete suppression of all reaction to the surroundings; lastly, there is an intermediate state in which there is no dominating idea or feeling, but in which there is automatic reaction to the surroundings exactly parallel to hypnotic automatism.

The *causes* of these different states are similar. Neurotic heredity plays an important part in many instances. Severe sudden shock, whether pleasurable or painful, is often the immediate cause; the nervous exhaustions following epileptic fits or acute mental disorder are equally efficient causes; blood poisoning, whether due to poisons such as alcohol or fevers, may also leave states of stupor; prolonged privation and exposure or wasting illnesses are no less dangerous. There seems to be some special relationship between mental stupor and sexual faults. Masturbation is credited with the production of a very large proportion of these forms of disorder.

Malnutrition of the brain tissue, mechanical interference with the circulation, and causes of rapid nervous discharge are the principal physiological causes of stupor.

General Symptoms.—The onset may be sudden, following at once a sudden shock; on the other hand, the causation may be gradual and yet the onset sudden. It is often preceded by a period of malaise and unrest of variable depth and duration. It may last for long periods, and may pass off gradually or suddenly. It may be periodical or recurrent. It may take part in regularly systematised mental disorder (*vide* "Katatonia," p. 987). It may be the termination of other forms of acute or recurrent mental disorder. It may end in profound dementia or in death. It may be part of general paralysis of the insane. The symptoms vary to some extent according to whether there is a dominating idea or not. As a rule, the patients are not emaciated; they are often flabby with sallow complexions and greasy skins, their hair is scurfy, lips soft with saliva either dribbling or retained in the mouth, pupils often large and mobile, tongue —if it can be seen—flabby and indented; there is often causeless smiling or laughing; the general weight of body may be maintained. The hands get a swollen, semi-transparent aspect; and are very blue, with tendency to whiten when pressed on. Circulation is generally feeble, but not always slow; the heart's action also is feeble. The circulation is not greatly increased by such drugs as amyl nitrite, and the sensory reactions are slow, thus, snuff often will not produce sneezing. Appetite is not active, but food is taken when given; though the patient, if he had to help himself, would probably starve in the midst of plenty. The muscles may be rigid, but the tendency is rather to catalepsy; the sphincters act normally, but there is a disposition to retain excretions—thus saliva is often held in the mouth, urine in the bladder, and faeces in the rectum. There may be a recurring proclivity to masturbation, but this is not common in the more profound cases. Sleep may be good, but the patient, if left to himself, would neither undress nor wash. He may be emotional, and occasionally he is impulsive; and by his sudden outbreaks may become dangerous. There may be some sensory hallucinations to which on recovery the patient may refer. He may lead a kind of dream-life, thinking he is no longer among human beings in this world, but that he has passed to another. The reflexes are often exaggerated.

In nearly all these states there is anaemia with reduction of the red blood-corpuscles.

Melancholia with stupor is considered on page 970. In these cases there is some dominating delusion; and memory and consciousness remain, though the delusions and sensory disorders may colour the recollection. The general aspect is that of mindlessness with some expression of dread and resistance, rigidity of muscles, and general opposition to everything that is being done to or for the patient; the sleep is bad, food refused, general bodily health failing. This state may gradually pass off, it may remain permanent, it may give place to maniacal excitement, or it may lead to simple dementia with limited mental power. The course may be of short duration; but as a rule such patients take many months in recovery; they need healthy country surroundings, regular attendance, good food, alcoholic stimulants, and, as a rule, frequent feeding. Massage may be of use in a few cases, but generally speaking it fails. Hot baths with mustard in them are useful stimulants. Galvanism through the head has done good in some cases. Where the circulation is fairly good, shower baths are of use, but they must be watched carefully; sometimes the reaction is deficient.

Careful and constant watching may be necessary to control masturbation, and to see that the bladder and rectum are duly emptied.

After recovery there is a more or less clear recollection of what has happened during the illness.

The danger of suicide is not great, as a rule; but impulsive acts may occur. Death may come by accident or some secondary disease, such as bronchitis or pneumonia.

Katatonic stupor (acute primary dementia) is the name of the second group of cases; such are met with after fevers, after epileptic fits, and in some cases after a shock. The aspect is much like that of melancholia attonita; but there is a silly look without any appearance of dread, and the attitude is less that of resistance than that of indifference. There may be catalepsy. The sleep is often fair, the appetite good, and food taken with assistance; there is no active opposition to anything which is being done for the patient; he may retain his water or faeces till taken to the closet, but when there he may pass the excretions normally, or he may pass urine and faeces inadvertently.

He is apparently without any memory, and on recovery will not have any knowledge of what has been going on during his illness. In some cases there are periodical recurrences of states of this kind. They may be permanent, or may pass off suddenly or gradually; they are rarely replaced by mania or any other form of mental disorder. There is little tendency to impulsive acts. There is little danger to life. The patients often get gradually fatter. Healthy surroundings and general simple stimulation are the best remedies. The line of treatment depends, of course, to some extent on the cause of the trouble.

In epilepsy I have seen more harm follow from the indiscriminate use of bromides than from the fits themselves. In alcoholic cases I think the complete withdrawal of alcohol is indicated. Free purgation and the use of Turkish or shower baths, with electricity, are of service.

In the third group I have placed the **cases of true stupor which resemble hypnotic states.** In these heredity, shock, acute illness, and the like causes of nervous exhaustion are generally the active excitants. There is a silly or abstracted look; a tendency to laugh or smile without external cause; evidence of hallucinations generally auditory; cataleptic muscular states, with some rigidity and inattention to needs of the body as far as food and excretion. There is often a masked memory, so that the patient on recovery has a dream-like recollection of the past.

These hypnotic states may pass off; they may become recurrent or may lead to more permanent mental weakness.

Allied to this latter condition I would mention cases of temporary loss of consciousness with fully-organised automatic actions. In these cases patients lose themselves, and are found by friends perhaps after some days' absence, they being quite unable to give any account of what they have done in the interval. Some of these cases doubtless are masked epileptics, but there are others in whom no epileptic history can be obtained (*vide* also p. 503).

KATATONIA.—Under this head Kahlbaum, in 1869, described certain cases in which stupor plays a part. There is considerable difference of opinion whether this state can be looked upon as a specific form of mental disorder; the general feeling among English alienists is against accepting it as such. In this disease there are generally hysterical or hypochondriacal symptoms of a very varying nature; these may range from simple hysteria to hysterical paralysis; the hysterical stage may be followed by a period of melancholia or stupor, or the melancholia in turn may give rise to stupor—there being the three stages of hysteria, melancholia, and stupor, the order of the occurrence of these symptoms varying. The symptoms not only vary in their order but also in their intensity. In severer cases, besides motor inhibition and dulness, there is a tendency to cyanosis of the extremities, and to salivation.

There is generally a stage in which there is catalepsy, and also a stage —which has been considered the most characteristic—of gesture and of "verbigeration," this latter being a term used for the repetition of a syllable; thus the word "incidence" would be thus pronounced in-in-in-in-in-ci-ci-ci-ci-de-en-en-ence, in many cases the voice rising in pitch as the word is pronounced.

There is no doubt that certain chronic cases of mental weakness pass into or through a stage which may be called katatonic, and certain epileptic cases also exhibit the same symptoms; but it is not clear that the phase ought to be ranked as a separate disease.

I have met with one or two cases in which similar symptoms present in highly neurotic subjects have recurred, and have been repeatedly recovered from. There is no special treatment for these cases, and in many instances they may be treated out of an asylum.

Catalepsy.—This symptom may occur in various forms of mental disorder, but cannot claim to be a special disease. Dr. Stoddart describes it as a state in which there is a blind unconditioned obedience to suggestion from without, and he includes such symptoms as *Flexibilitas cerea* and automatic obedience and imitativeness. It was described by Hack Tuke as a neurosis in which sensation and mobility are suspended and consciousness more or less modified, the chief characteristic being a tendency for any muscular attitude into which a patient is placed to be maintained for an indefinite period. This is best seen in hypnotism, in which most constrained positions may be maintained for periods which are quite beyond the power of the person in a natural state. It may occur in post-epileptic states, and is frequent in hysterical subjects. It occurs in dementia precox and in some cases of katatonia and mental stupor. It has no special value as a symptom, but in my opinion it is rarely present in any but unfavourable cases.

G. H. Savage.

REFERENCES

Stupor: 1. Capitan et Kortz. "Contribution à l'étude clinique de la stupeur," *Méd. mod.*, Paris, 1895, vi. 632.—2. Whitwell, J. R. *Brain*, London, 1895, xviii. 66. **Confusional Insanity**: 3. Moiselle, E. "La Pazzia confusionale o disnoia (confusione mentale)," *Gazz. d. osp.*, Milano, 1895, xvi. 500.—4. Worcester, W. *Am. Journ. of Insan.*, Chicago, 1894-95, li. 71. **Psychic Paralysis**: 5. Clelland, S. M. *Bull. Psych. Sect. Med.-Leg. Soc.*, 1894-95, xii. 300. **Katatonia**: 6. "Case of L. W. Dodson," *Med. Rec.*, N.Y., 1895, xlviii. 28.—7. Blaman, G. A. "A Half-Century of American Medico-Psychological Literature," *Am. Journ. Insan.*, Chicago, 1894-95.—8. Goodall, E. "Observations upon Katatonia," *Journ. Ment. Sc.*, London, 1892, xxxviii. 227.—9. Kahlbaum, K. "Zur Kasuistik der Katatonie," *Monatsschr. f. Psychiat. u. Neurol.*, Berlin, 1902, xii. 22.—10. *Idem.* "Klinische Abhandlungen über psychische Krankheiten," Heft I., *Die Katatonie*, Berlin, 1874.—11. Neisser, C. "*Über die Katatonie, ein Beitrag zur klinischen Psychiatrie*," Stuttgart, 1887.—12. Verity, W. P. "Katatonia," *Journ. Nerv. and Ment. Dis.*, New York, 1886, N.S., xi. 695. **Catalepsy**: 13. Jones, T. *Brit. Med. Journ.*, 1863, i. 585.—14. Lasègue. *Arch. gén. de méd.*, 1864.—15. Linas. Art. "Catalepsie," *Dict. encyclop. des sc. méd.*, Paris, 1873, xiii. 59.—16. Puel. *Mém. de l'acad. gén. méd.*, 1856.—17. Tuke, Hack. *Dict. Psycholog. Med.*, 1892, i. 184.—18. Warnock, C. T. *Journ. Ment. Sc.*, Lond., 1895, xli. 82.

G. H. S.

STATES OF MENTAL WEAKNESS

(DEMENTIA)

By A. R. URQUHART, M.D., F.R.C.P.Ed.

MENTAL weakness is the general characteristic of all cases of insanity. It is, as has been shewn in the foregoing pages, associated with excessive self-consciousness and painful feelings in melancholia, as well as with loss of control and excitement in mania. It is evident in the hysterical, in the neurasthenic, and in the degenerate—in all the protean forms of mental disorder. In the less formidable it may only be apparent as a slight and temporary diminution of inhibitory powers; or it may prove to be, even if slight, the insidious premonition of a mental involution of the gravest import. It may be absolute, inherent, congenital—the fatuity of idiocy; or it may be absolute, degenerative, acquired—the senescence of a worn-out brain. Between these extremes the grades are infinite, and, for the purposes of description, are classed in clinical groups under esoteric titles which are yearly increasing in number and complexity; such as paranoia, confusional insanity, insanity of negations, and so on. The mental weakness which is congenital or acquired in infancy is recognised as *idiocy* or *imbecility* (p. 874). It constitutes a definite clinical group—definite in etiology, symptomatology, pathology, and treatment. The weakness which is partial, and involves, as the prominent feature, mental degradation in the department of ethics, has been named and described as *moral insanity*.

The immediate purpose of this section excludes consideration of these specialised studies, and limits attention to cases of dementia—cases which are broadly characterised by evident defect in mental function, manifested after the age of puberty. To this class belongs the great majority of the chronic insane. Dementia is, in fact, the common goal.

Except in old age *partial dementia* is comparatively rare. In the blunting of the faculties, the loss of energy, or perhaps the slight stupidity consequent on advancing years, a partial dementia is frequently noticeable. We must also recognise in premature senility a similar train of symptoms, such as the underlying pathological conditions are capable of producing. It can be readily apprehended that physiological conditions of brain exhaustion must eventuate in dementia of a more or less pronounced kind; and this is a subject which merits more careful inquiry than has yet been entered upon. Kraepelin has shewn that bodily fatigue is demonstrably injurious to thought, and that mental exhaustion weakens the brain power in definite curves and ratios. This may appear to be the proving of a truism; but it is among those transitory and slight aberrations that we must first seek for elucidation of problems as yet unsolved. The mental symptoms of the period of convalescence from exhausting disease have also been subjected to careful analysis; and much more may be hoped from such investigations than has yet been discovered by the observers who have entered on this field of work.

Dementia may be acute or chronic, primary or secondary. I shall deal with these forms of mental weakness seriatim, and afterwards refer to the general questions affecting care and treatment, and other points of interest.

Acute dementia (*Stupor*) is characterised by suspension of the intellectual, moral, and instinctive faculties—a loss of sensibility. It is, in fact, a mental stupor, and as such has been described in a preceding section (p. 984). The name *anergic stupor* was first applied to this disease by Dr. Hayes Newington, in order to mark the suspension of mental function which is the essential feature of the malady, and to separate from it *delusional stupor*, which is an entirely different condition. Inasmuch, however, as an anergic stupor is evidently a deprivation of mental function in direct consequence of disease of the brain, we must regard it as a true dementia. Reference is made to it here in order to emphasise the diagnosis between this form of stupor and melancholic stupor, with which it may easily be confounded. There is, in this country undoubtedly, a tendency to reserve the term *dementia* for such cases as are incurable, and therefore to substitute *stupor*, when, for instance, the apparent weak-mindedness so often following mania proves to be curable. The introduction of the term *dementia precox*, which is generally regarded as the German equivalent for the insanity of adolescence, renders it more necessary to make this differentiation and to prefer the term *stupor* to *acute dementia*. It is not proved that dementia precox is a clinical entity, in fact it may be regarded as a terminal condition of like nature to other forms of secondary dementia; but those who use the name admit its occasional curability (*vide* also p. 929).

Dementia, taking the name in a broad sense, really marks a weakening of brain power, resulting from conditions in which the cortical cells are either disordered in a manner which it is convenient to call "functional," or affected by degenerative lesions of a grave and unalterable kind. We are not yet in a position even to state definitely that such a profound change as vacuolation of the nuclei of these cells is incurable. It would appear, indeed, that the recuperative powers of the cortical cells may be greater than has been supposed.

Primary dementia is undoubtedly a rare form of weak-mindedness, although cases do occur in which blunting of the mental powers begins gradually and insidiously, without any apparent acute stage to usher in the symptoms. Most commonly these cases are senile, and of course chronic. The physical basis is the cortical degeneration, which is progressive and incurable.

Secondary dementia is the condition of mental weakness into which so many acute cases subside. The great majority of insane patients detained in asylums, or otherwise cared for, is classed under this division.

When we speak of a patient as being sunk in dementia we immediately picture a person who has passed through an attack of acute insanity, and has fallen into a permanently crippled or a permanently unstable state. Indeed this too common result has been graphically insisted on by Dr. Clouston in his definition of mental disease as "a tendency to dementia."

Some degree of dementia may be looked for in the course of ordinary mental disease. An initial stage of depression or irritability, followed by mania or melancholia, is very frequently succeeded by weak-mindedness, temporary or permanent. This, indeed, closely corresponds with our general experience of other maladies. The patient is conscious of malaise, the attack supervenes, and thereafter, during convalescence or decadence, there is a period of languor and exhaustion. We cannot make exception of brain disease in studying degenerative change. The cortical elements are exhausted, their fine processes are obliterated, the cells are subjected to destructive influences. The indications of treatment are towards repair and recuperation. If successful, the dementia is temporary; if unsuccessful, it is permanent. We have an analogous, though less prolonged, degeneration in the history of Bright's disease. The acute and curable form may be of short duration, dangerous to life, threatening the conditions of existence; whilst the chronic and incurable form pursues its course to the bitter end, alleviated, and in some degree controlled, by care and treatment, but in the essential nature of the malady unalterable.

The striking feature of all forms of dementia is the diminished mental power evident in thought and action. The controlling power is weakened; inhibitory power is damaged. Thereby the individual may manifest exaggerated normal impulses or perverted impulses; inasmuch as attention is enfeebled, memory is impaired, or even destroyed, and in consequence comparison (judgment) is incompetent. We are now considering secondary dementia; but these characteristic traits are also part and parcel of chronic mania, and of recurrent insanity, which must also be classed as states of mental weakness. This was clearly apprehended by Griesinger in his classical treatise on insanity, previous to which the true nature of chronic mania was not recognised.

We have seen, then, that the clinical groups into which states of weak-mindedness may be divided fall into the same natural divisions as other maladies, and must now refer to the general array of symptoms by which dementia is to be measured. As the result of old age, degenerative brain disease, nervous exhaustion, mental shock, attacks of mania, or more rarely melancholia, dementia is gauged by the depth of mental degradation; and its nature is determined by the antecedent causes. The dementia consequent on fevers is very different, as regards prognosis, from the dementia following upon epilepsy. Alcoholic dementia may be curable, and syphilitic cases also are sometimes susceptible of cure. We may even be hopeful of the best results from operative interference when obvious weak-mindedness is caused by a tumour of the brain in an accessible position. It is true that the depth of mental degradation does not always correspond to the chances of curability. As in other forms of insanity, the defect or disorder may be less intense than indelible. Cases occur in which apathy is not apparent, in which the ordinary duties of life are performed with seeming propriety, as in the case of Professor Titel (cited by Griesinger), who believed himself to be Emperor of Rome, yet continued to read his course of lectures in college. But in the course of medical practice the advice of the physician will generally be sought when the malady is more pronounced, when the mind and conduct are manifestly disordered.

The first step is to ascertain the cause. The history of the patient is of prime importance. Is the dementia the result of inherited mental disease? Dr. Clouston presses this point so far as to say that all pure cases of secondary dementia originate in hereditary and adolescent cases of insanity. Without entirely accepting this statement, for the prognosis may be good if the patient be young and the case free from degenerative lesions, it must be admitted that the hereditary element is of the gravest import. Still, the exceptional cases are numerous enough to encourage active treatment, and to sustain hope. Other considerations which must weigh with the physician are the influence of physical diseases of an exhausting character; the influence of poisons, such as alcohol or syphilis; the influence of such special nervous disorders as epilepsy or general paralysis. These diseases are dealt with in other sections, and do not require to be specially reviewed in connexion with this subject. Suffice it to say that each must be weighed in consideration of the individual case.

While loss of memory is to be tested in relation to recent as well as to former events, and the judgment by reference to what may be competent to the individual, it must be observed that judgment is less deeply affected if assistance be given by the examiner. The difficulty of dealing with more abstract questions, especially by persons originally of limited intelligence and poor education, must have due consideration. For instance, Dr. Savage in investigating the case of Gamble, tried for murder, found him incompetent to resolve the simplest problem in school subtraction; but on stating the question in a concrete form, "You have four apples and I take away one," the correct answer was readily given.

It is of importance to recognise that the higher mental faculties, the results of refinement and education, being the latest added in the scale of civilisation, are often the first to deteriorate; but the exceptions are so many that it is impossible to lay down a definite rule. The diminished control over emotions, the fleeting impression made by the events of the day, the loss of natural affections and social qualities, the incapacity for or disinclination to the accustomed duties of life, constitute the evidences of that diminished mental power which is the prominent feature of dementia. Such are the negative symptoms; but dementia is not an affair of negation only, there are positive symptoms which in ordinary cases are not less noticeable. The attitude and expression of demented patients are characteristic. They frequently crouch or stoop, they seek to cover themselves with clothing over head and ears by day and night. These postures—as indeed are all rhythmic, automatic, fixed eccentricities—are of evil omen. The expression is equally noticeable: it is vacant, with fleeting traces of emotions which are irrelevant or too easily roused. The bodily habit is often spare, sometimes obese, never healthy. The ruddy hue of health is seldom seen, not even when these patients are constantly out of doors. The mental ruin, the moral degradation are written large upon those doomed to dementia. Cortical degeneration is evident to their finger-tips.

That would not be a complete description of dementia which failed to take into account those cases which present characteristics of excitement or destructiveness. Many patients require special attention, because of their faulty habits, throughout a long course of years; some bolt their food, and are often in urgent danger of suffocation; some neglect the calls of nature, and so demand constant vigilance; some are subject to sudden suicidal or homicidal impulses, for the storm which wrecked the mind does not entirely die down, but flashes up from time to time in brief and violent gusts.

Symptoms of untoward import are negative in character. Persistent apathy, inattention and loss of memory, degradation of the higher mental qualities down to the lower, a perverted nutrition, a lean body with a voracious appetite, a persistent attitude or habit of posture are all of evil omen. Should a patient increase in body weight, and at the same time present no real improvement in mental condition, his future must be regarded with grave anxiety.

No study of dementia can be complete without reference to Dr. Maudsley's vivid portraiture of this condition. At first sight it would appear that the degenerative changes conditioning it should be as disastrous to life as they are to well-being; but demented patients live long even under the most unfavourable conditions. In the end they are prone to fatal diarrhoea or insidious pneumonia; but neglectful of ordinary sanitary precautions, forgetful of common decency, they survive in spite of many dangers. It should be remarked, however, that the expectation of life for all classes of the insane is less favourable than that of mentally normal persons.

The *treatment* of dementia must follow on the consideration of the individual case. If the patient is young, and it is apparent that the stage of weak-mindedness after a maniacal attack is unduly prolonged, every week of delay in convalescence tells against the chances of recovery. In such a case the treatment must be stimulating, the environment as healthy as possible. The patient should be discharged from asylum care at the earliest opportunity. Travel and interesting companionship are valuable aids to restoration. The indiscriminate prescription of travel in the treatment of mental disorders is much to be deprecated; in melancholia the risks run, and the injury thus caused, are often indefensible. In dementia, on the other hand, it is hardly possible to overrate the value of all reasonable means calculated to arouse the patient to occupation and diversion. On the energetic and systematic employment of such remedies as will awaken the system to a healthy metabolism, and strengthen the patient generally, depends in great measure the successful issue of the case. If, unhappily, the dementia should prove incurable, it will be difficult to sustain active interest in the slow progress of the malady. Still, intercurrent diseases have occasionally roused unpromising patients to sanity; and, proceeding on the experience thus gained, Dr. Lewis C. Bruce has recorded beneficial results from the effect of thyroid feeding. It does not appear that blistering of the cranium has any favourable influence on these cases; modern treatment is rather directed to the improvement of the general health by tonics and less violent measures.

If we are called upon to deal with a case of "organic" dementia, or a case in which the mischief is wrought by brain tumours or other "coarse" disease, it would be manifestly absurd to pursue such methods as above indicated. To the classical symptoms of tumour is often added a profound melancholia, which demands early operative interference. Again, prognosis in the dementia of myxoedema is now most favourable. A few years ago such patients seemed doomed to progressive mental decay; now under thyroid feeding they improve and recover. These are indeed miracles of healing. In cases of alcoholic dementia there is no better remedy than iodide of potassium in full doses; whilst those of syphilitic origin are frequently susceptible of improvement under a course of the green iodide of mercury combined with small doses of opium. The educated physician will accept no rigid rule for the treatment of weak-mindedness, he will limit his endeavours by no stereotyped formula; he will deal with each individual case after consideration of the causes, on searching examination of the actual bodily state, and in conformity with the general indications. Evidence of the toxic nature of insanity, long suspected but unproved, is rapidly accumulating, and a more hopeful expectation is encouraged by the clinical and laboratory work of later years. Naturally this expectation is applicable to the earlier stages of faulty metabolism, before the disordered cerebral cortex passes into ruin.

The question of proper care must be solved by the same individual reasonings. If the dementia is profound and means are narrow, the case will probably be best dealt with in an asylum where adequate nursing and special appliances are to be found. If the weak-mindedness be less pronounced, precautions may be required for the prevention of injury to the person, or disaster to his estate. It must not be forgotten that there is a possibility of recrudescence of the acute mental disease. As I have said, patients apparently in a state of harmless dementia have suddenly manifested symptoms similar to those of their first acute attack, and in that condition have committed suicide or murder. A powerful mental influence may reawaken old ideas, which have been to all appearance long dead after years have passed in apparent torpor.

The proper provision for chronic dements is a question of the greatest importance, in view of the constantly increasing size and number of asylums for the insane. No method can be regarded as satisfactory unless it provides for frequent medical supervision and competent care and treatment, as well as for suitable accommodation of a more material kind.

A. R. URQUHART.

REFERENCES

General Works: 1. BIANCHI, L. *Clinical Psychiatry* (Transl.), 1906.—2. BRUCE, L. C. *Clinical Psychiatry*, 1906.—3. BUCKNILL and TUKE. *Manual of Psychol. Med.*, 1879.—4. CLOUSTON, T. S. *Clin. Lect. on Ment. Diseases*, 1896.—5. GRIESINGER. *Ment. Pathol. and Therapeutics* (Transl.), 1877.—6. KRAFFT-EBING. *Lehrbuch der Psychiatrie*, 1889.—7. KELLOGG. *Mental Diseases*, 1897.—8. KRAEPELIN. *Clinical Psychiatry* (Transl.), 1906.—9. LEWIS, BEVAN. *Mental Diseases*, 1889.—10. MACPHERSON, J. *Mental Affections*, 1899.—11. MAUDSLEY, H. *Pathology of Mind*, 1895.—12. RÉGIS, E. *Man. of Ment. Med.* (Transl.), 1894.—13. SAVAGE, G. H. *Insanity and Allied Neuroses*, 1891.

Special Articles: 1. ALDRIDGE, C. "Ophthalmoscopic Observations," *West Riding Med. Rep.*, 1874, iv. 291.—2. BLANDFORD, G. F. Art. "Dementia," Quain's *Dict. of Med.*, 1889, i.—3. BOLTON, J. S. "Amentia and Dementia," *Journ. Ment. Sc.*, London, 1905, li. 270 *et seq.*—4. CAMPBELL, A. W. "Pathology of Aged Insane," *ibid.*, 1894, xl. 638.—5. CLAYE-SHAW, T. Art. "Dementia," Tuke's *Dict. of Psychol. Med.*, 1892.—6. CLOUSTON, T. S. "Secondary Dementia," *Journ. Ment. Sc.*, London, 1888-89. xxxiv. 325.—7. CRICHTON-BROWNE, J. "Acute Dementia," *West Riding Lun. Asyl. Rep.*, 1874, iv. 265.—8. GREENLEES, T. D. "Blood-vessels in Dementia," *Journ. Ment. Sc.*, 1885-86, xxxi. 327.—9. GUCCI in *Neurol. Centralbl.*, 1888, vii. 583. —10. MESCHEDE. "Acute Dementia," *Journ. Ment. Sc.*, 1887, p. 50.—11. NEWINGTON, H. N. "Stupor," *ibid.*, 1874, p. 372.—12. PICK, A. "Dementia praecox," *Prag. med. Wchnschr.*, 1891, xiv. 312.—13. TUKE, D. H. "Mental Stupor," *Trans. Internat. Med. Congress*, London, 1881.—14. VIGOUROUX, "Démence précoce," *Ann. méd.-psychol.*, Paris, 1894, 7^{e} s., xix. 69.—15. WHITWELL, J. R. "Mental Stupor as a Pathological Entity," *Brain*, London, 1895, xviii. 66.—16. WIGLESWORTH, J. "Pathology of Acute Dementia," *Journ. Ment. Sc.*, London, 1883-4, n.s. xxix. 355.

A. R. U.

SYSTEMATISED DELUSIONAL INSANITY

(PARANOIA)

By the late CONOLLY NORMAN. With an Addendum by R. PERCY SMITH, M.D., F.R.C.P.

Introduction.—That which chiefly distinguishes man from animals consists perhaps in this, that in mankind the adaptations of the individual to his environment do not take place along those lines of activity, practically invariable in the life-history of a species, the trend of which we name "instinct." In man the place of this invariable instinct is taken by the highly variable judgment.

In the mind, therefore, the judgment is the most important, as it is the most human of the faculties. The judgment is impaired in all forms of mental disease. Where general consciousness is clouded, or where the primary functions of perception, reproduction, and so forth are profoundly affected, impairment of judgment, though present, is not a prominent or distinctive condition.

In three forms of mental disturbance especially is impairment of judgment a fundamental symptom—in idiocy, in general paralysis, and in paranoia (delusional insanity). In the first of these, judgment, the most adult of the faculties, has never been developed; in the second, the chief type of degenerative mental change in the individual, it is lost, in accordance with the general rule that faculties degenerate in the reverse order to that of their development; finally, in paranoia, an affection which may be considered as the most definite form of hereditary degenerative mental disease, the judgment either develops from the first along abnormal lines, or slowly acquires a permanently morbid mode of growth.

Delusional insanity (paranoia) may be defined as that mode of mental unsoundness which is specially characterised by delusion; that is, by beliefs not common to the race, which arise from the uncorrected action of the imagination, are fixed and systematised, and are not immediately connected with a predominant emotional state.

Delusions may occur in every form of mental disease; but in the acute forms—mania, melancholia, dysnoia—they are associated intimately with the predominant emotional state wherein they appear to take their rise, and which at the same time they reinforce. Delusions in the acute forms and in general paralysis are varying, confused, unstable, and even contradictory. When they are very fixed, as they often are in melancholia, they are still not schematised, not reasoned out in a systematic manner. The peculiarity which has been called the "systematisation or organisation of delusion," together with fixity of the morbid idea and usually slow development, forms the characteristic note of paranoia.

It has been supposed that, because there is in ordinary cases of delusional insanity a relative integrity of the faculties of perception, reproduction, and memory, the affection is merely a partial one; and, indeed, the term "partial insanity" is still sometimes applied, chiefly by lawyers, to forms of paranoia. On the same grounds Esquirol adopted the now rejected title of monomania, under which name he seems to have included only the sufferers from fixed delusions of exaltation. Taking into consideration the profound impairment of the highest faculty of judgment which is connoted in the acceptance of delusion, we perceive the deep significance of the symptom and its value as a test of mental unsoundness, quite apart from its convenience as a concrete and commonly clearly demonstrable fact.

Delusional insanity occurs in two forms, the original and the acquired. The former condition (first described by Sander) is distinctly rare; it is characterised by a tendency to delusion, occurring in deeply-tainted persons at an early age, who exhibit also general eccentricity and a slight degree at least of moral defect (waywardness, perversity, and the like). In later life a chronic fixed delusional state is engrafted upon this, which appears to be, so to speak, the congenital mental state. The condition is interesting on account of its rarity, and on account of its analogies and alliances with moral insanity and chronic affective insanity (*folie raisonnante*).

Usually paranoia is an affection of adult life. It is most frequent in persons having a strong bent towards insanity (9). Since the time of Snell it has been generally accepted as a primary form of insanity. Probably Dr. Bevan Lewis is alone among modern authors in holding that it is always secondary to mania or melancholia. It has been held, however, that this affection, though not secondary in the individual, is secondary in the family, occurring in the children of those who have suffered from one of the acute forms of insanity. But the taint may be acquired during the life of the individual, and it is not rare to see paranoia in persons whose family history is clear, but who have themselves been exposed for long periods to some injurious influence such as alcoholism or sexual excess. It is generally a disease of slow manifestation, being profoundly constitutional in its nature. It often attacks persons who have been eccentric, irritable, or of neurotic habits for years, or always. This fact, together with the acceptance of untrustworthy or often delusional reminiscences of patients, has led to the classification of many more cases as original than should, properly speaking, come under that category. Though really insidious in its onset, the outbreak often appears to be very sudden. This may occur as a genuine exacerbation, or be due to a dissimulation of symptoms which is frequently put on in the earlier stage. The character of the person who is about to drift into paranoia is self-centred. He may or may not be solitary in habits, but he is self-opinionated and self-absorbed. He may be dull and dreamy, or bustling and fussy. In the latter case little result usually comes of his activity, for he is inconstant and whimsical. In the merely intellectual sphere for the same reason he is rarely potent. He is at best a brilliant failure, sparkling but unstable. He is vain, greedy of praise, and deaf to reproof. He is suspicious, touchy, and, like nearly all the insane, "ego-centric." That delusions of the kind which occur in paranoia should arise in persons of this character is probably as natural as that maniacal or melancholic ideas should arise in the sufferers from mania and melancholia; nevertheless the delusions in the disease we are considering are rightly called "primordial" (Griesinger), for they do not appear to belong immediately to any emotional state, and they strike in upon the mind of the patient as a new train of events. With the appearance of delusion the prodromal stage is over. The forms which delusions take in paranoia are infinitely varied in detail, and yet present in their general aspects a singular degree of uniformity. They may be conveniently considered under two great heads.

Delusions of Persecution (Lasègue).—These are the most frequent. Among cases of paranoia which come under treatment for the first time, not less than 85 per cent exhibit delusions of persecution. In many cases these first appear in the form of insane suspicion. The patient is constantly on the look-out lest he should be watched; he is constantly thinking that trifling and indifferent matters have some special reference to him, and so forth. In this stage people look strangely at him as he passes, look at each other significantly, talk in a low voice about him; of this he is sure: the clergyman's sermons are directed at him; he sees references to his private affairs in the "agony column" of the newspapers, and so on. Illusion, or the phenomenon which has been called delusional interpretation, now reinforces the delusive beliefs; thus the innocent conversation of passers-by is made to deal with or to refer to him. It is seldom long before more definite delusions appear. The patient is maligned, is threatened; efforts are made to poison him, or to torture him. At first his suspicions are connected with a narrow range of objects, and his persecution comes from some vague quarter usually indicated as "they." "They annoy me." Soon, however, everything acquires a bearing on the patient, and "they" become some definite body of people (the Jesuits, Freemasons, some particular family, or the like); or some one or more persons are found guilty of the patient's suffering. The patient, who usually retains considerable powers of explanation, explains all this by recounting a conspiracy, which, originating in one or several people, has spread to enormous dimensions, and assails him from every point. The growth of this system of delusions, which gradually fills the patient's whole mind, constitutes that "organisation," or "systematisation," which gives its special feature to paranoia. Eventually the sufferer's mind is so filled with his troubles that he often refuses to believe that his case is not known to every one. The whole world is engaged in his affairs, and his interlocutors' questions are often met with the saying, "Why ask questions? You know all about the matter." In some instances the patient, if he is clever and persuasive, succeeds in convincing some member of his family, or his dependents, of the truth of some at least of his allegations, thereby adding a difficulty to diagnosis.

There may be a perfect system of insane delusion or suspicion without hallucination, but this is rare. Hallucinations are the rule; either hallucinations of hearing alone, or combined with hallucinations of the other senses. Hallucinations of hearing may at first be vague, such as indefinite noises, or hammering, ringing, and the like; but they usually take the form of human speech. Patients generally refer to them as "the voices." Sometimes a patient says that at first they were but whispers (perhaps delusional interpretation), but that later they have become distinct voices. In early stages the patient commonly hears a monologue; later, a second or many voices are heard. The matter heard is almost always unpleasant. Sometimes it consists of abuse, vile charges, threats, blasphemy, or obscenity; sometimes of silly gibing. Often the voices describe to the patient or to each other the mode of their action, their motive, or the nature of their conspiracy. Often they torment him by idly remarking on what he is doing or about to do: "Now he is getting into bed, now he is putting out his candle," and so forth. Sometimes they forestall his words just as he is about to speak. This is particularly apt to happen with regard to his prayers. On the other hand, the voices sometimes reply to the patient's thought, and occasionally in a strain which is so unexpected by him that he does not at once comprehend the meaning. Sometimes, of two voices, one threatens or abuses the patient, and the other warns or defends him, or expostulates. Clinically, both the last-mentioned symptoms are of bad import. It has been held that they point to a destruction of the sense of personality, but it is hard to see why on this supposition they should have a specially grave significance, considering that they both occur commonly enough in dreams. Some patients are tormented by hearing all their thoughts spoken. This has been called the echo of thought. The patient usually accounts for it in the belief that by some underhand means his thoughts are made public. Some patients recognise the voices they hear as the voices of persons whom they know (either used by such persons or stolen from them); patients who hear several voices distinguish them from each other by actual or fanciful names. Not infrequently patients say that they are conscious of voices without hearing them. Occasionally they feel them in various parts of their bodies, but most frequently they are rendered conscious of them by the movement of their own throat or tongue, which seems to frame the words. This phenomenon has been described under the name of psycho-motor verbal hallucination (17). The physiological explanations offered for this, and for some other problems connected with auditory hallucination, are far from satisfactory.

Hallucinations of vision alone are very rare in paranoia. Indeed, they are seldom a prominent symptom. In some patients the image of printed or written words, either habitually or occasionally, takes the place usually occupied by "voices" as a means of persecution or of cryptic communication (19). Many patients see hateful or revolting objects as it were at second hand, and say, "I am made to see so and so"; "Such and such things are projected before me"; seeming to recognise that there is less of reality in the visual than in the auditory impressions.

Hallucinations of taste are probably always unpleasant. They are sometimes indistinguishable from delusive interpretations. They are most important in connexion with delusions of poisoning, in which cases they are rarely absent. Sometimes filth of all kinds is placed in the patient's food, or introduced into his mouth when he is asleep.

Hallucinations of smell are also offensive. Patients complain of mephitic fumes, noxious gases, chloroform and other volatile poisons, odours of filth and decomposition. Certain patients perceive sexual odours, either produced in themselves or proceeding from others. Hallucinations of smell are very frequently associated with sexual delusions, especially in women at the climacteric period.

Hallucinations of the genital sense are common. They are more complained of by women than by men. On certain female sufferers from delusions of persecution all kinds of sexual outrages are perpetrated. They complain not only of being violated (by men, and in some cases by beasts, as a mode of torture and disgrace—outrages which they say occur mostly at night, or when they are asleep), but also of sensations produced

KING'S COLLEGE HOSPITAL
MEDICAL SCHOOL.

by occult means in their genitalia when they are awake, such as dilatation, and so forth.

Hallucinations of tactile sensibility and of general sensibility are usually combined, though they may exist apart. The commonest complaints are of shooting, darting, or stinging pains, attributed to the operation of various powers, most commonly electric batteries. Various sensations, which are sometimes described, of the limbs being moved, or of weight or heat, seem to shew that the muscular, the pressure, and the thermal senses, in so far as they are distinct, are also concerned.

In a well-established case of paranoia of some standing it is not rare to find hallucinations of all the senses. It is to be noted that hallucinations occasionally appear to be unilateral. This is particularly the case with auditory hallucinations, patients stating that they hear the voices with one ear only.

Besides the senses which bring the individual into relation with the external world, the inner sense, if we may so call it, of the operation of the intellect is profoundly modified; and what may be called hallucinations of this sense are variously combined with hallucinations of hearing and of the other senses.

Thus, it is a very frequent complaint that the patient's thoughts are read; his thoughts also are influenced; he is compelled to think of horrible or degrading subjects; by mesmerism or some such means he is rendered stupid and unable to think; he is made to sleep or rendered somnolent; he is kept awake — this may be by voices, by tortures, or by direct action upon the mind preventing sleep; he is compelled to be silent, or to say things which he knows to be wrong, or which he did not intend to say, or sometimes which he does not understand. Acts which have the appearance of impulse are sometimes accounted for in this way; that is, the patient states that he is made to do them by some external influence dominating his will. Some patients complain that their moral control is weakened, and that their moral feelings are tampered with; some assert that their mind is entirely in the hands of their enemies. A patient said to me, "They have my mind; they hold it in hypnotism." Quite apart from the patient's suspicion that people want to send him to an asylum, which may be a truth or a verisimilitude, he very often entertains the conviction that "they" are endeavouring to put him out of his mind, to make him really insane. This belief is clearly dependent in some cases on a sense of altered mental action, though not always. Sometimes it is oddly associated with the belief that the patient's relatives, or neighbours, or persecutors, are insane, by which he accounts for their injurious acts.

The emotional state of the persecuted paranoiac is usually indifferent. He bears the most astounding torments without becoming depressed. He looks upon his sufferings from quite a different point of view from the melancholiac. The latter feels himself a wretch who deserves his doom; he is humble, self-accusing, and abased. The paranoiac is the victim of foul wrong; he is proud, defiant, and self-centred. So with their complaints: Séglas has contrasted in a phrase of equal wit and propriety the litany of the melancholiac with the romance of the paranoiac. Nevertheless, there are no absolutely hard-and-fast lines in mental disease; and from time to time we meet with cases in which the symptoms of the two conditions are mixed. It is also to be borne in mind that paranoiacs occasionally commit suicide; sometimes to escape from their persecutors, sometimes in obedience to the command of a voice (divine or other); sometimes with the object of exposing and damaging their enemies.

On the other hand, the resentment of the persecuted in his own defence is a common thing, and often makes patients of this class very dangerous. There is an element of danger in all cases of persons who believe themselves persecuted, especially of those who have auditory hallucinations. Such people are apt to attack those around them suddenly, under the belief that they have somehow joined in the conspiracy against them, or have spoken injuriously of them. Many of the persecuted, however, are passive and retiring; others from the first resent the treatment they conceive themselves to be subjected to; others again endure their sufferings quietly for a length of time, and then arrange a counterplot, and try to avenge themselves. To these is especially applicable the expressive French phrase *persécutés persécuteurs.* The actual danger depends to a large degree upon whether the patient has personified his persecutors or not. A not infrequent danger originates in a notion, sometimes arising in the mind of the persecuted, which well exemplifies the peculiar crooked pseudo-logic which characterises him; namely, that if he murders some one he will have an opportunity of stating his case in court and exposing his enemies. It is often with this design that, in asylums, officials or fellow-patients are attacked.

Among the passive persecuted, especially among women, a common symptom is incessant change of residence, either in the form of travel or of merely moving from one house to another. This is done with the object of escaping persecutors, and usually at first the attempt is, for the time at least, successful (Foville, Ball). Change of scene produces a temporary amelioration. This migratory tendency often gives a useful clue in obscure cases.

When a patient is profoundly reticent, the suspicion which forms a fundamental element in his character will often shew itself in his acts. Thus he may cook his own food for fear of poison; live on nothing but eggs for the same reason; plug his ears to keep out voices; barricade his doors; blind his windows; stuff his chimneys, or, as in a case that came under my notice, sit at (literary) work with a small tent over him, to prevent his persecutors from stealing his ideas as he writes. In cases of this class the physician will do well to note indications of delusion obtained at the first interview. Very frequently, on the second, he will find the patient in an access of suspicion, believing that his visitor has joined the enemy.

Delusions of exaltation may occur as primary phenomena, or may be secondary to delusions of persecution. They range over an immense variety of subjects. A patient may believe himself to be God, a prophet, a great inventor, possessed of great wealth, talents, or beauty. Exalted delusions are less prominently connected with hallucinations than the persecutory. On the whole, the exalted paranoiac shews less general intellectual acumen and mental activity than the persecuted. In many cases persecutory and exalted delusions are inextricably mixed. Thus, a patient will say that he is a child of noble parents, the heir to great wealth, and that he is cheated, tormented, and imprisoned on that account; or that he is a prophet or a saviour, tortured by the wicked or tempted by fiends. Not rarely a patient has visual hallucinations of an exalted and cheering kind—as of God or angels; yet his auditory hallucinations are full of blasphemy, mockery, and insult. Occasionally a persecutory or a melancholic idea succeeds an exalted one, as when the saint becomes the victim of the ungodly; or the saviour feels that he must expiate the sins of his people. But more commonly the progress is in the reverse direction, and the persecuted becomes exalted. It does not always, indeed it does not generally happen that the advent of exalted ideas entirely drives out the persecutory notions. Usually, however, the latter become less pronounced. The change in the type of prominent delusion generally marks a downward step in the march of intellectual decay.

The followers of Magnan of Paris have laid down that there is a remarkable and very distinct form of insanity, which Magnan proposed to call *délire chronique*, which is characterised by the occurrence of four definite stages:—(i.) Disquiet and suspicion. (ii.) Persecutory delusion, with hallucination of hearing. (iii.) Delusion of exaltation. (iv.) Dementia. A good many cases run a course resembling this more or less closely; but there are no sufficient reasons for making of them a separate form of insanity. Many cases of paranoia are so slow in progress that a diagnosis depending on the succession of phases might need half a lifetime to confirm. In practice we find that the persecutory and exalted stages are not sharply delimited from each other. It has been held by some that the onset of exaltation after depression is due to the action of hallucination, but this is a superficial view. Hallucination and delusion do not stand to each other in the relation of cause and effect, but are the common result of that enfeeblement of judgment which allows a sensory impression or a figment of the imagination to be accepted without correction.

Further sub-varieties of paranoia are furnished by the sufferers from erotic delusions, delusions of jealousy, hypochondriacal delusions; and by the victims of what may be called litigious insanity (the *Querulantirresein* of v. Krafft-Ebing). A sexual tinge may colour all classes of delusion, but that which we specially note is the condition common in young men and elderly women, less common, but not rare, in young women, in which the patient conceives a passion for a person to whom perhaps he or she has never spoken, and builds up on little or no foundation an organised romance which occupies the whole attention. The pangs of misprised love often turn this patient into a veritable *persécuté persécuteur*. In this form, and in delusion of jealousy (beliefs of conjugal infidelity), actual hallucinations are occasionally very hard to demonstrate. Insane jealousy is very frequent in paranoia following habitual alcoholic excess; though it occurs in cases in which such excess has not existed (11). Litigious insanity is usually unaccompanied by hallucination. Full of persecutory ideas and insane suspicion, its unhappy victims live in an atmosphere of legal actions, which last until the lawyers have sucked them completely dry, when they are prone to adopt means which the law does not approve to avenge their persecutions. In either phase, they are a source of great misery to their relations (whom they commonly ruin), and to their acquaintances, the most compassionate of whom are the most likely to be their victims.

The delusions of the paranoiac are very often intimately associated with religious ideas; and his hallucinations take the form of visions of saints or divine personages, or of the voice of the Deity. The common notion, however, that there is a special type of religious insanity is not correct. The nature of religion, its mystery, its capacity for covering the whole mental field, its strong hold upon the immediate personality, sufficiently account for the frequency with which religious conceptions predominate in the diseased mind. That the unknown, or little known, should afford abundant food for insane conjecture is natural enough. Similarly, the sense of mystery which oppresses the man who is conscious that he is different from other people, naturally makes him turn for explanation to the most mysterious of subjects. The same conditions caused the persecuted of old to attribute their sufferings to witchcraft. Nowadays, particularly among half-educated people, hypnotism is a favourite form of mystery, and medium of persecution. So it has been successively with various natural forces and their applications — steam, electricity, the telegraph, the telephone, and more recently the Röntgen rays.

Connected intimately with this sense of mystery is the tendency which has been called *mystic interpretation*. In many cases the sense of something occult is always present, and every event of life, every detail of the environment, takes its place in the scheme of mystery, or has some hidden significance; some indirect bearing upon the patient which is not evident on the surface. It is singular to what a degree memory is warped in some cases by this tendency. Thus, a patient will recall incidents of many years back, often of the most trivial nature, incidents to which he himself admits he attributed no import at the time, incidents which he even says he had meanwhile forgotten; and he will now point to them as important links in the chain of evidence which confirm his beliefs.

The memory, in paranoia, often seems to remain acute for a long time. There may, nevertheless, be profound impairment of this faculty. Besides the errors arising from insane and mystic interpretation, and from actual delusions of reminiscence, we often find that though the patient's memory is keen on the subjects of his delusion, or on matters personal to himself, it is weak on other topics. In most cases delusions, though conforming to the general type, gradually change their precise form. One enemy or one form of persecution gives place to another; and the patient, who argues as vigorously for the reality of the second as he did for that of the first, has evidently forgotten the latter; sometimes he goes even so far as to deny point-blank that he ever entertained it.

The mode of origin of individual delusive beliefs is obscure, but can occasionally be traced. The apparent origin of a delusion in an hallucination or in a dream is, of course, not fundamental. The patient's own account of the beginning of his trouble is always untrustworthy. A symptom of his disease is a belief that he acts from the most logical motives, and he defends such belief with an appearance of close reasoning. Meanwhile, of course, he is quite inaccessible to reason on the subject of his delusions. A good example of this is given by the obedience to the commands of a "voice," even though the thing commanded may be wicked or absurd, or the voice recognised as demoniacal or hostile. Nevertheless, we can occasionally trace the genesis of delusion. Undoubtedly, patients are often accused by their voices of crimes or errors that they have really committed. Undoubtedly, also, real feelings of physical pain and malaise are interpreted as due to external agencies, and genuine feelings of mental confusion and failure are attributed to unseen influences. Positive antithesis and repulsion sometimes, too, suggest, or seem to suggest delusion; as, for example, delusions of violation and obscene hallucinations, which are most common in women of the class accustomed to banish sexual thoughts most completely from their minds. A curious further instance of the indirect origin of delusion on a sort of antithetical basis is to be found in the great prevalence of hallucination of hearing among those who have become deaf. This is sufficiently frequent to suggest that the vexation of losing a sense, and the temptation, common enough in the deaf, to suspect that they are the object of conversation going on around them, may be active factors in determining delusion. Hallucinations of sight among those who have grown blind also occur, but are not so common.

Although it has often been supposed that the form of insanity which we are discussing is associated with high mental powers, either in the individual or in his family, we find in practice that while supervening dementia is neither so early nor so profound as in other forms of insanity, genuine intellectual power is very rare. The patient may be saturnine and reticent (in which case his dull self-absorption is usually evident enough), or he may be voluble and loquacious on subjects connected with his delusions; but in either case his mental activity is limited, and even in its narrow field it is barren. Two or three facts are to be noted: among patients of the communicative type there are many who, while they proclaim the strongest opinions, restrict their actions always to declamation or writing; who are perfectly happy in the exercise of the chief function that seems to be left to them, pondering over their wonderful story, and reasoning it out in the mode they deem convincingly logical.

A good many find ordinary words not sufficient for them, and invent new ones, or apply existing phrases in new senses; this forming of new terms has been called "neologism." Thus, one patient complained that he was persecuted through a "system of vibration"; another suffered through an instrument called a "typhone"; the thoughts of another were "read by a hypophone, and translated into logarithms"; while another, a woman almost wholly illiterate, wrote that she was persecuted by "two wizards, called Harry Stottle and Legerdemain." Often the names or new words convey no suggestion of a meaning.

It frequently happens that the patient who is reticent in conversation, even to the degree of concealing his delusions absolutely, will yet reveal himself in writing. Some patients write all day. Some decorate their manuscripts with extraordinary drawings and mystic signs. Most of them repeat themselves without end. Self-revelation is common in the writings of the insane. Even among the sane, except in those who are trained to literary work, the difficulty of expression in writing is so much greater than in speaking that, once overcome, it leads the writer to forget to maintain his guard. Besides, there are many patients whose lives are largely a dream, and whom the contact with concrete existences will steady for a moment. These folks will often take a hint quickly enough from the facial expression, from the gestures, or from the tone of one who talks with them; and will therefore be discreet, whereas when alone with their writing materials, they soliloquise on paper. But whatever be the explanation, it is certain that valuable indications are very often to be found in the patient's writings, and therefore he should always be induced to write, and on subjects which seem to require elucidation, especially if the case be one in which there is any doubt.

CONOLLY NORMAN.

ADDENDUM.—The article on systematised delusional insanity or paranoia, written by the late Conolly Norman, gives such an admirable and comprehensive picture of the morbid mental states included under that heading that it would seem to be an act of vandalism to tamper with his text; but, nevertheless, it is desirable to call attention to some points upon which emphasis has been laid of late.

Many observers have pointed to the fact that although in its fully developed form systematised delusional insanity is characterised chiefly by the presence of fixed and systematised beliefs "not immediately connected with a predominant emotional state," yet if the history of the early development of the condition in any particular case is carefully ascertained it will be found that emotional disturbance has played a much more important part than is commonly believed. Moreover, although it seems to be untrue clinically to hold that paranoia is always secondary to mania or melancholia (which have been classified as affective mental disorders in contradistinction to intellectual disorders such as paranoia), yet this sequence does occur in some cases. The character of the person who is about to drift into paranoia is spoken of as self-centred, vain, greedy of praise, suspicious and touchy, and it is during this prodromal stage before definite delusions appear that emotional disturbance plays a most important part in the development of delusion. Even those who believe in intellectual disorder as a primary feature in paranoia classify patients suffering from the established disease upon an affective or emotional basis, namely, into the two broad groups of the depressive and the exalted.

In my opinion, the view that emotional disturbance in paranoia is entirely secondary to the intellectual disorder is erroneous and untrue clinically. This view has been held by Krafft-Ebing, Cramer, Ziehen and others of the German Schools; but Kraepelin points out that the opposition looked upon as fundamental between disorders of the understanding and those of feeling is only a psychological one and not at all clinical, and that in disease both are bound up together in an incalculable way. Further, he points out that the attempt to separate the paranoic group from other forms of insanity always ends with the statement that mixed forms and transitional cases occur between it and the "affective" mental disorders. Kraepelin's views are shared by Jolly, Moeli, Specht, Linke and others. It is indeed hard to believe that the patient who is developing persecutory ideas, whether of the general, hypochondriacal, sexual, or physical types, does not suffer mental pain as a fundamental part of his mental disorder; and that the patient developing exalted delusions, whether of the religious, erotic, or fantastic type, does not similarly enjoy pleasurable feeling, although it may be, as it frequently is, interrupted by annoyance at the non-fulfilment of his expectations, or combined with persecutory ideas. In my opinion the separation of primary affective from primary intellectual disorders is purely artificial, and in paranoia the affective state cannot be ignored.

The other point to which attention must be called is that Kraepelin classifies many of the cases hitherto considered as coming under the head of paranoia as cases of dementia precox of the "paranoid" form. Although there are cases of paranoia which do not present, even at the end of a long life, marked indications of failure of mental power other than systematised delusion, yet, as Kraepelin points out, many of the cases shew from the first an imperfect systematisation of delusions or else a rapid weakening of systematisation, or even in time lose them altogether and pass into a chronic weak-minded condition which remains permanent. This is perhaps most likely to occur in the cases originating in adolescence or early adult life, and is also met with in the cases described by Magnan as *délire chronique* in which dementia is the terminal stage. The dementia supervening upon the paranoid form of dementia precox is described as being associated with mannerisms, stereotyped movements, or forms of expression as in other forms of dementia precox, although cognition and memory are slightly affected. Dr. Stoddart speaks of dementia paranoides as differing from all other forms of dementia precox in that the terminal stage is long delayed, even for years. English observers are not fully agreed as to the justification for the transfer of cases from the class of paranoia to that of dementia precox.

R. PERCY SMITH.

REFERENCES

1. ARNAUD, F. L. *Ballet's Traité de pathol. mentale*, Paris, 1903.—2. BALL. *Leçons sur les maladies mentales*, 1880-83.—3. CRAMER. *Allg. Ztschr. f. Psychiat.*, Berlin, 1895, li. 286.—4. ESQUIROL. *Des maladies mentales*, 1858.—5. FOVILLE. "Les Aliénés voyageurs et migrateurs," *Ann. méd.-psychol.*, 1875, xiv. 5.—6. GRIESINGER. *Arch. f. Psychiat.*, 1868, i.—7. GRIMALDI. "L'Origine affettiva dei delirii paranoici," *Ann. di nevrol.*, Napoli, 1903, xxi. 314.—8. KRAEPELIN, E. *Psychiatrie*, 7th ed.—9. v. KRAFFT-EBING. *Lehrbuch der Psychiatrie*, 1875.—10. *Idem.* "Über der sogenannten Querulantenwahnsinn," *Allg. Ztschr. f. Psychiat.*, Berlin, 1879, xxxv. 395.—11. *Idem.* "Eifersuchtswahn der Männer," *Jahrb. der Psychiat.* xi.—12. LASÈGUE. "Délire de persécution," *Arch. gén. de méd.*, 1852, xxviii. 129.—13. LEWIS, BEVAN. *Manual of Mental Diseases*, 1899.—14. LINKE. "Nocheinmal der Affekt der Paranoia," *Allg. Ztschr. f. Psychiat.*, Berlin, 1902, lix. 256.—15. MAGNAN. "Les Délirants chroniques et les dégénérés," *Gaz. des hôp.*, avril 1884; also *Leçons cliniques sur les maladies mentales*, 1893; also monograph, *Le Délire chronique*, etc., par Magnan et Sérieux, 1892.—16. MENDEL. "Paranoia" in Eulenburg's *Real-Encyclopädie der gesamten Heilkunde.* Mendel revived the term Paranoia, which he first applied in this sense. He states that it was used in a more general sense by Vogel in 1764.—17. PRICHARD. *Treatise on Insanity and other Disorders affecting the Mind*, 1835.—18. SANDER. "Über eine specielle Form der primäre Verrücktheit," *Arch. f. Psychiat.*, 1868, i. 587.—19. SÉGLAS. "Les Hallucinations psychomotrices," *Progrès méd.*, 1888, viii. 124, 137; and memoir, *Les Troubles du langage chez les aliénés*, 1892.—20. *Idem. Leçons cliniques sur les maladies mentales*, 1895.—21. SMITH, R. PERCY. "Paranoia," *Journ. Ment. Sc.*, London, 1904, l. 607.—22. SNELL. "Über Monomanie als primäre Form der Seelenstörung," *Allg. Ztschr. f. Psychiat.*, Berlin, 1865, xxii. 368.—23. SPECHT. *Über dem pathologischen Affekt in der chronischen Paranoia.*—24. STODDART, W. H. *Mind and its Disorders*, 1908.—25. ZIEHEN, H. *Psychiatrie*, 3rd ed., 1908, Leipzig.

C. N.
R. P. S.

HYPNOTISM: AND THE TREATMENT OF INSANITY AND ALLIED DISORDERS BY SUGGESTION

By J. MILNE BRAMWELL, M.B.

Insanity.—According to Auguste Voisin of the Salpêtrière, no one believed it was possible to hypnotise the insane until 1880, when he first succeeded in a case of acute mania. In 1852, however, Esdaile reported that he had hypnotised 37 insane patients in the Calcutta Asylum. Few details are given, and he admitted that the therapeutic results fell far below his anticipations; but one patient, who had cut his own throat, was easily influenced at the first attempt, and rendered insensible to pain during the subsequent operation.

From 1880, Voisin devoted himself largely to the treatment of mental disorders by suggestion, and stated that he cured many cases of undoubted insanity. The majority suffered from mania, or from melancholia with hallucinations, suicidal impulses, and delusions of persecution. Many of the cases are of extreme interest, and are fully and carefully reported.

Répoud, of the Cantonal Asylum of Marsens, Fribourg, claims to have obtained results similar to those of Voisin. Burckhardt, of the Préfargier Asylum, records successful cases of delusional insanity and acute puerperal mania; Liébeault, hereditary suicidal monomania; Brémaud, religious melancholia following confinement; Velander, melancholia with mutism.

Similar experiments were conducted at Morningside Asylum, in 1893, by Dr. Robertson. He does not state that he cured any case of undoubted insanity, but he was able to control the worst case of suicidal and homicidal mania that had been in Morningside for ten years. He also reports a case of hypochondriacal melancholia which he cured in six weeks. The following is his summary of the uses of suggestion among the insane :—(i.) As a direct therapeutic agent: (*a*) in insomnia it may succeed in intractable cases in which narcotics have failed, and may be of great service where the brain nutrition is already bad, and the effect of depressing drugs undesirable; (*b*) when the brain is in a highly unstable condition, it may be of direct therapeutic value as a sedative in preventing an outburst of insanity from passing into mania; (*c*) it may be of service in dispelling fleeting delusional states and minor psychoses. (ii.) For the purposes of management: (*a*) in overcoming the morbid resistance of patients for their own benefit, *i.e.* in the administration of food and medicine; (*b*) suggestion may be used in cases of excitement and violence instead of mechanical, chemical, or physical restraint. In reference to the latter point, it is interesting to note that, about fifty-five years ago, Keen employed hypnotism extensively in Berhampur Asylum, India, and found it of much service in procuring quiet and maintaining discipline.

The following are successful cases from my own practice :—

(1) Mrs. ——, aged 35. Mania, with a determined attempt at suicide, followed by mutism and refusal of food. (2) Mrs. —— aged 30. Melancholia with suicidal impulses. (3) Mrs. ——, aged 46. Melancholia with strong suicidal impulses, and the fixed idea that she would commit suicide in the same way her father and uncle had done. (4) Miss ——, aged 38, November 1900. Mother died in an asylum; one sister had delusions, another obsessions. A cousin committed suicide, several were insane, and one had obsessions. In 1892 the patient had an attack of acute melancholia, lasting several months; since then she had always been depressed and peculiar. In 1895 she had a second attack of acute melancholia, and a third one in 1900. When I first saw her (November 1900) she was profoundly melancholic and almost mute. Result: recovered. Last report, November 1902, satisfactory. (5) Mr. ——, aged 37, May 1899. Melancholia with suicidal impulses. Father and sister insane. Result: recovered. Last report, 1903, satisfactory. (6) Mrs. ——, aged 56, February 1902. Mother, sister, and two brothers insane.

The patient had her first attack of melancholia fifteen years previously, followed by others, both prolonged and severe. Result: recovered. No relapse up to the last report. (7) Miss ——, aged 26, November 1907. Delusional insanity. Result: recovered. Last report, February 1909, satisfactory. (8) Miss ——, aged 40, March 1907. *Folie du doute* of twelve years' duration, which had become an insane delusion. Recovered. Later reports (1909) satisfactory.

Dipsomania.—Forel and many others claim to have obtained excellent and durable results in this disease. Ladame draws special attention to three of Forel's cases. All had had attacks of delirium tremens, and were inmates of his asylum. They were extremely difficult to manage, and expressed their determination to resume drinking directly they were liberated; but, despite this, complete recovery followed treatment by suggestion.

A considerable proportion of my patients suffered from dipsomania. In the successful cases, including both sexes, the duration of the disease had varied from five to seventeen years; some had had several attacks of delirium tremens and epilepsy, and in most there was a family history of alcoholism. In several instances repeated and prolonged treatment in a retreat had failed. Some of the earlier cases have now remained over eighteen years without relapse.

Morphinomania and other Drug Habits.—The following table, published in 1896, gives the results obtained by Wetterstrand in 51 cases:—

	Men.	Women.	Total.	Died.	No Effect.	Relapsed.	Cured.
Morphinism; morphine injected subcutaneously	16	22	38	2	5	3	28
Morphinism: morphine taken internally	1	2	3	...	...	1	2
Morphinism and alcoholism	1	...	1	...	...	...	1
Morphinism and cocainism	2	1	3	1	1	...	1
Cocainism	1	...	1	...	...	...	1
Opium internally	..	4	4	...	...	1	3
Chloralism	...	1	1	...	...	...	1
	21	30	51	3	6	5	37

Many of the cases were extremely grave and of long standing, and other treatment had been tried without success. One of the successful cases—a medical man—had taken morphine for eighteen years, and, during the last four, cocaine also. Another patient, Dr. Landgren, published his own case five years after he had been successfully treated by Wetterstrand. All Wetterstrand's cases, with the exception of one, were treated in private houses; and he found residence in a retreat quite unnecessary. The following are the points which he considered most important in order to ensure success:—the patient must be placed under the care of an absolutely trustworthy nurse; the doctor must gain the patient's confidence, and treat him as a person who is ill and deserving of sympathy, not as one who ought to be scolded for having given way to a bad habit; the morphine should not be stopped at once, but rapidly decreased, and the patient constantly informed of the diminution; all injections of water instead of morphine are wrong and ought to be scrupulously avoided. Fulda, Marot, and others report cases of long-standing morphinomania which have remained several years without relapse.

In many of my cases of hysteria and neurasthenia a drug-habit existed, especially where insomnia was a marked symptom. Some of these patients took morphine regularly, either by the mouth or hypodermically; and many of them were addicted to the use of chloral, sulphonal, etc. When treatment by suggestion removed the insomnia, the drug-habit was usually abandoned. The worst case of morphinism I have seen has now remained over fourteen years without relapse.

Vicious and Degenerate Children.—Treatment by suggestion frequently affords good results in vicious and degenerate children; and the following are the conclusions which Bérillon submitted to the International Congress of Hypnotism, Paris, 1889:—

(i.) Many carefully observed facts illustrate the therapeutic value of suggestion in the following diseases of children:—incontinence of urine and faeces, nervous tics, nocturnal terrors, chorea, onanism, blepharospasm; and many disturbances of the nervous system of a functional character.

(ii.) So far, no appreciable results have been obtained in cretinism, idiocy, and deaf-mutism.

(iii.) Suggestion is an excellent auxiliary in the education of vicious and degenerate children; and is especially indicated in cases of lying, cruelty, inveterate idleness, cowardice.

(iv.) Suggestion should be confined to cases in which the usual methods of education have failed, and medical men alone should employ it. It is not necessary for normal children: ordinary education ought to be sufficient. When, however, children are addicted to theft and other vicious habits, or are afflicted with disgusting infirmities, we ought to try to cure them by suggestion.

These conclusions were adopted unanimously by the Congress, and were transmitted to the Minister of Public Instruction and the Minister of the Interior.

Successful cases—ranging from undoubted moral insanity to those presenting one or more bad habits or tricks—are also cited by Liébeault, Voisin, and others. Many of the patients suffered from enuresis nocturna, either alone or associated with symptoms of degeneracy. In 1887, Liébeault recorded 77 cases, 45 being boys and 32 girls. The youngest was three, the oldest eighteen years of age, the average being about seven. In all, with the exception of 9, the malady dated from infancy. The results were as follows:—cured, 56; improved, 9; no result, 12.

Some of my successful cases, and I have had many, might have been justly classified as morally insane, the patients being addicted to self-abuse, purposeless theft, and lying. Others suffered from violent attacks of temper, nocturnal terrors, insomnia, enuresis nocturna, nail-biting, and various tics.

Sexual Perversion.—Krafft-Ebing, Schrenck-Notzing, and others report the successful treatment of sexual perversion, including cases of its grosser forms; and I have also obtained good results in these conditions.

Obsessions.—Hypnotic treatment has yielded brilliant results in various forms of obsession, such as agoraphobia, claustrophobia, *folie du doute.* The following, amongst others, cite successful cases:—Liébeault, Schrenck-Notzing, Hecker, Wetterstrand, de Jong, van Eeden and van Renterghem, Bernheim, Gorodichze, Voisin, Burot, Mavroukakis, Bourdon.

As far as my experience goes, the results in cases of obsession are better than in any other class of functional nervous disorder. The cases I have treated have been numerous, varied, and generally of long-standing. In some of them the obsessions had become insane delusions, and in several instances there had been attempts at suicide.

Neurasthenia.—In severe cases of neurasthenia the mental disturbance is often even more profound than in the milder forms of insanity. The whole character seems changed, and the patients suffer from varied and numerous delusions in reference to their physical and mental condition, and talk about these from morning to night. In 1893-94 Schrenck-Notzing published an account of 228 neurasthenic cases treated by himself, Brügelmann, Bérillon, Bourdon, Voisin, Burckhardt, Forel, Ringier, Ritzmann, Bourru, Burot, Stadelmann, von Corval, Michael, Drozdoivski, van Kozuchoivski, Neilson, Tuckey, Bernheim, van Renterghem, and Wetterstrand. The following were the therapeutic results:—

72 cases—equalling 31·6 per cent—recovered.
84 ,, ,, 36·8 ,, were much improved.
72 ,, ,, 31·6 ,, shewed no improvement.

Epilepsy.—Braid recorded a case of epilepsy in which there was no relapse after eight years; but amongst modern writers, few, with the exception of Wetterstrand, report cases as cured by hypnotic treatment. The latter asserted that some of his earlier cases had remained many years without relapse, and his general results are so startling that Forel said personal investigation alone enabled him to accept them.

Putting aside a case of epilepsy associated with dipsomania, in which over twelve years have passed without relapse, I have treated 10 cases by suggestion. Improvement occurred in 5 instances, but complete recovery in none. In one case of four years' duration, in which there were several attacks of *grand mal* per week and much impairment of intellect, there was a remission for twelve months; the patient then relapsed, but the fits ceased under renewed treatment.

Hysteria.—Possibly at least half of the cases recorded may be classed as hysterical; these range from isolated sensory or motor disturbances to the graver form of the malady, *la grande hystérie*, so seldom seen in England. For the purposes of this article only cases presenting more or less profound mental symptoms are deserving of notice. Bérillon states that for three years he watched the hypnotic treatment of *la grande hystérie* by Dumontpallier at the Pitié Hospital. In the majority of cases not only did the convulsive attacks disappear, but the other symptoms also. Some of these patients are now in excellent health, married and mothers of families, whilst others occupy important posts.

Voisin published a case of long-standing hystero-epilepsy with maniacal attacks. The patient recovered, and became wardrobe maid at the Salpêtrière. Burot cites a successful case of hysterical mania of two years' duration, with hallucinations of sound and delusions of persecution. Burckhardt, de Jong, Wetterstrand, van Eeden and many others also report good results in hysterical melancholia. A male patient with *grande hystérie* in my own practice recovered after hypnotic treatment.

Hypnotic Theories and Methods.—James Braid demonstrated that all so-called mesmeric phenomena were purely subjective in origin, and due to suggestion, instead of to some mysterious force or fluid. At first, Braid believed that it was necessary to put patients into an artificial sleep before treating them by suggestion, and he invented the terminology we still erroneously use. Later, he found that only one in ten of those he cured by suggestion passed into any condition resembling sleep, and he proposed to abolish his entire terminology, as it was misleading. Braid's later views were unnoticed, and, until recent times, the induction of artificial sleep was thought to be necessary. Thus, Liébeault long used Braid's earlier methods and called the condition *sommeil provoqué*. Bernheim insists that the so-called hypnotic state is always a conscious one, and explains that ignorance of this caused the fallacies of the Salpêtrière school. The patients, who were supposed to be asleep, really heard and responded to unconscious suggestions of the operator, and to these, not the physical means, the results were due. Consciousness is retained even in what is termed the most profound hypnotic condition. Here, the patients believe they have been asleep, because the state is followed by amnesia. Their lost memories, however, can always be revived by suggestion.

The essence of the whole condition is an increased suggestibility, which has followed the methods employed by the operator.

The modern conception in reference to so-called hypnotic phenomena is that they are due to the intelligent action of a secondary consciousness. This is the view held by William James, of Harvard, and only differs in one particular from Carpenter's theory of unconscious cerebration—the intellectual process is believed to be carried on consciously, but in another substratum of the personality, so to speak. According to Carpenter, much of our intellectual activity was essentially automatic and might take place unconsciously. German metaphysicians, from Leibnitz onwards, also believed that the mind might undergo modifications, without being itself conscious of the process, until the results presented themselves to the consciousness in the new ideas the process had evoked. As an example of this unconscious mental activity, Carpenter cited the spontaneous remembrance of some name we had vainly tried to recollect, and which suddenly flashed into consciousness when we were thinking of something different.

Method.—The following is the method I now adopt. I rarely begin treatment the first time I see a patient. After having satisfied myself that the case is a suitable one, I make a careful study of the patient's mental condition, and do my best to remove everything—fears, erroneous preconceived ideas, etc.—which might stand in the way of success. I then explain my methods to the patient; tell him about the secondary consciousness and its powers, and say: "Next time you come, we shall not talk about anything until after treatment. You will sit down in an arm-chair and close your eyes. While you are resting, I shall make suggestions of two kinds, but I do not want you to listen to them. You will always hear my voice, but I wish it to be a drowsy accompaniment to your restful thoughts. While I am making suggestions, try to concentrate your attention on some restful mental picture; its nature does not matter, as long as it *is* restful. This concentration is simply an artifice to turn your attention from my suggestions; for, if your normal consciousness is absorbed in this way, the suggestions more easily reach the secondary one." I always frankly tell the patient that I cannot explain why suggestion given in this particular systematised way often produces results far exceeding those obtained by the suggestions of ordinary life. I also explain that I do not possess any occult power; that I am simply going to try to arouse forces that are latent in the patient's own brain, and, further, that I cannot promise to cure him, as, even if the case is suitable, much depends on his ability to carry out my instructions.

The first set of suggestions refers to the conditions which I wish to create while the patient is in the arm-chair. I tell him that each time he comes he will find it easier to rest, to turn his attention away from me, and to concentrate it upon something restful. I have previously explained that I do not wish him to go to sleep, but that if he can get into the drowsy condition which precedes sleep, the suggestions are likely to be responded to more quickly. The other suggestions are curative and vary with each different case. These are begun at the first treatment, and I tell the patient beforehand that I make them, not because I believe they will be at once responded to, but because it is the repetition of this impression, made in this particular way, which gives it its power.

At first the novelty of the proceeding usually attracts the patient's attention, and prevents his turning his thoughts from my suggestions. After a few treatments, however, he is generally able to keep his attention on some restful mental picture, and often passes into a drowsy, day-dreamy state. Sometimes the condition apparently becomes one of slight natural sleep: the patient ceases to hear my voice for a moment or two, then drifts back to consciousness.

Formerly, not only did the operator attempt to obtain so-called hypnosis, but also tested its presence by suggesting to the patient that he was unable to open his eyes. Sometimes this succeeded, but, if not, the suggestion was repeated until responded to. Doubtless the success of these, and other inhibitory suggestions, helped to create the theory of hypnotic automatism, but, as I have frequently pointed out, hypnotic subjects never accept suggestions which are contrary to their moral sense. These experiments are objectionable, however, and I discarded them at a very early date, long before I changed my views as to the nature of so-called hypnosis. Although I suggested hypnosis, I never tested its existence by experiment: as the result of the methods I then used, the patients passed into a restful or drowsy condition and I made curative suggestions. The whole object of suggestive treatment ought to be the development of the patient's will-power and his control of his own organism. That idea, and that alone, should be instilled into his brain, and no experiment, however trivial, should be made which could possibly tend to make him believe that the operator was trying to dominate him.

Apparently, as the result of these methods, patients frequently develop an increased suggestibility and brilliant therapeutic results follow. I do not believe, however, that the methods in any way explain the phenomena, and I can see no logical connexion between the methods and their apparent results.

Dangers.—During twenty years' practice of treatment by suggestion I have not seen a single instance in which ill-effects, even of the most trivial description, have followed its use, despite the unstable mental condition of many of my patients. My experience is shared by others. Thus, Forel asserted that he, as well as Liébeault, Bernheim, Wetterstrand, van Eeden, de Jong, Moll, and the other followers of the Nancy school, had never seen a single instance in which mental or physical harm had been caused by the use of suggestion as a therapeutic agent. No complete record of their cases has been published, but the number certainly exceeds fifty thousand.

Therapeutic Results. Summary.—*Insanity.*—The question whether insanity in its graver forms can be cured by suggestion must still be regarded as an open one; but, in endeavouring to estimate the value of this form of treatment, we must not forget that our knowledge practically dates from Voisin's first case in 1880, and comparatively few attempts have been made to give the method a fair trial. Certain forms of insanity are undoubtedly curable, and it seems reasonable to suppose that treatment which can induce sleep without drugs, and remove excitement and other morbid mental conditions, may prove a valuable remedial agent.

Whilst no one disputes the genuineness of Voisin's results, the accuracy of his diagnosis is by no means universally accepted. In Forel's opinion, Voisin's so-called insane patients simply suffered from hysteria, which, with analogous mental troubles of a fugitive character, can be undoubtedly cut short by suggestion. Forel admits that he has been able to induce sleep by suggestion, and to obtain a temporary cessation of hallucinations; whilst in a case of dementia, with inactivity, vicious tendencies and intercurrent maniacal attacks, he succeeded in making the patient work, improved his moral condition, and arrested the maniacal attacks, which three years later had not returned. On the other hand, he has never succeeded in curing genuine insanity by suggestion. The brain, he says, is the instrument we employ in suggestion, and if the instrument itself be spoilt, we are no longer able to make use of it, or only to a very small extent, as a means of reacting upon itself.

In reply to this, Voisin, while admitting that suggestion could do nothing in somatic affections, such as general paralysis, cerebral softening, and the like, stated that many of his patients were genuinely insane, not simply hysterical. He asserted further that the successful treatment by suggestion of hysteria alone marked a therapeutic advance of no little importance. In asylums there were a large number of patients who suffered from hysterical insanity. They were violent and dangerous, and inspired both disgust and pity by their tendency to drink and steal, by their lies, dirtiness, and obscene and unnatural acts. Some, who had long been asylum inmates, now, thanks to suggestion, led active and useful lives.

It is to be noted also that, at a later date, Forel reported a successful case of hallucinatory insanity. The patient, who believed himself to be controlled by a spirit which entirely regulated his life, and forced him to do the most absurd things, was easily influenced by Forel; the hallucinations disappeared after the first treatment.

Epilepsy.—Although the cases just referred to, particularly Wetterstrand's, are undoubtedly encouraging, I do not think we are justified in regarding them as conclusive. In some instances, patients were reported as cured before sufficient time had elapsed to warrant this conclusion; whilst the description of others leaves some doubt as to the accuracy of the diagnosis. In France, hystero-epilepsy, a disease frequently cured by suggestion, is common, and might easily be mistaken for genuine epilepsy.

Other Diseases.—Apart from insanity and epilepsy, the therapeutic results must be regarded as satisfactory.

In estimating the results obtained by suggestion, it must not be forgotten that the majority of the cases which I have treated have been extremely unfavourable ones. Before being sent to me, the patients have generally been ill for years, and have become hopeless, as the result of the failure of other methods. Thus, it is not the specialist, whose practice is almost entirely confined to unfavourable cases, who is likely to get the best results. It is the general practitioner, who can choose suitable cases from amongst his own patients—patients whose confidence he has already gained. When the advantages of treatment by suggestion, and its freedom from danger, become more fully recognised, it will doubtless be employed in earlier stages of disease. When that day comes, the results should be still more striking.

Most of the diseases treated by suggestion—insanity, obsessions, bad habits, involuntary muscular movements and the like—are due to want of control in one form or another. The object of treatment by suggestion should therefore be the development of the patient's control of his organism and of his own will-power. To this control, when gained, we owe not only the cure of disease, but also often freedom from relapse. I have followed the after-history of many of the more interesting of my patients, and find that relapse has been rare. Some of my cases of dipsomania and obsessions have now remained well for eighteen years.

J. Milne Bramwell.

REFERENCES

1. Bérillon, E. "Les Applications de la suggestion à la pédiatrie et a l'éducation mentale des enfants vicieux ou dégénérés," *Rev. de l'hypnot. expér. et thérap.*, Paris, 1889-90, iv. 153; also *Compt. rend. du premier Cong. internat. de l'hypnot. expér. et thérap.*, 1889, Paris, 1890, i. 157.—2. *Idem.* "Valeur de la suggestion hypnotique dans le traitement de l'hystérie," *Rev. de l'hypnot. expér. et thérap.*, Paris, 1889-90, iv. 34.—3. Bourdon. "Jalousie morbide compromettant la vie, guérie par la suggestion," *ibid.*, 1888-89, iii. 141.—4. *Idem.* "Applications variées de la suggestion chez une hystéro-épileptique," *ibid.*, 1891-92, vi. 358.—5. *Idem.* "Applications de la psychothérapie aux neurasthénies graves et anciennes avec complications diverses: phobies, obsessions, hypochondriaque, polysarcie, etc.," *ibid.*, 1899-1900, xv. 365.—6. Bramwell, J. Milne. "Dipsomania and its Treatment by Suggestion," *Proc. Soc. Study of Inebriety*, 1900.—7. *Idem.* "Obsessions and their Treatment by Suggestion," *Liverpool Med.-Chir. Journ.*, 1909, xxix.—8. *Idem.* "On Imperative Ideas," *Brain*, London, 1895, xviii. 321.—9. Burckhardt. "Application de l'hypnotisme au traitement des maladies mentales," *Rev. de l'hypnot. expér. et thérap.*, Paris, 1889, iii. 56.—10. Burot, P. "Grande Hystérie guérie par l'emploi de la suggestion et de l'auto-suggestion," *ibid.*, 1886-87, i. 355.—11. *Idem.* "Manie hystérique avec impulsions et hallucinations guérie par la suggestion," *ibid.*, 1889, iii. 336.—12. Cruise, F. R. "Hypnotism," *Dublin Journ. Med. Sc.*, 1891, xci. 377.—13. David. "Hystérie convulsive; idées de persécution et de suicide; guérison," *Rev. de l'hypnot. expér. et thérap.*, Paris, 1891-92, vi. 236.—14. Delbœuf. "Quelques considérations sur la psychologie de l'hypnotisme à propos d'un cas de manie homicide guérie par suggestion," *ibid.*, 1892-93, vii. 200.—15. Farez, P. "Traitement psychologique du mal de mer et des vertiges de la locomotion (chemin de fer, omnibus, tramway)," *ibid.*, 1897-98, xiii. 136.—16. *Idem.* "Idées délirantes de persécution avec hallucinations auditives et visuelles consécutives à un traumatisme psychique chez un glycosurique. Traitement hypnotique et guérison des troubles mentaux, malgré la persistance de la glycosurie," *ibid.*, 1898-99, xiv. 296.—17. Forel, A. "Durch Spiritismus erkrankt und durch Hypnotismus geheilt," *Ztschr. f. Hypnot.*, Leipzig, 1893, iii. 299.—18. *Idem.* *Der Hypnotismus, seine psycho-physiologische, medicinische, strafrechtliche Bedeutung und seine Handhabung*, 2te Aufl., Stuttgart, 1891.—19. Gorodichze. "Un Cas de claustrophobie guéri par la suggestion hypnotique," *Rev. de l'hypnot. expér. et thérap.*, Paris, 1893-94, viii. 53.—20. Hecker, E. *Hypnose und Suggestion im Dienste der Heilkunde*, Wiesbaden, 1893.—21. de Jong (de la Haye). "Valeur thérapeutique de la suggestion dans quelques psychoses," *Compt. rend. du premier Cong. internat. de l'hypnot. expér. et thérap.*, Paris, 1889, 196.—22. von Krafft-Ebing, R. *Psychopathia sexualis*, transl. by C. G. Chaddock. Phila. and London, 1893.—23. Ladame. "La Traitement des buveurs et des dipsomanes par l'hypnotisme," *Rev. de l'hypnot. expér. et thérap.*, Paris, 1887-88, ii. 129.—24. Liébeault. "Monomanie suicide guérie par suggestion pendant l'état de sommeil provoqué," *Intern. Cong. of Exper. Psychol.*, London, 1892, 143.—25. *Idem.* "Traitement par suggestion hypnotique de l'incontinence d'urine chez les adultes et les enfants au-dessus de trois ans," *Rev. de l'hypnot. et thérap.*,

Paris, 1886-87, i. 71.—26. Marot. "Morphinomanie et suggestion ; guérison datant de trois ans et demi," *ibid.*, 1892-93, vii. 233.—27. Mavroukakis, A. "Les Neurasthéniques et la suggestion. Agoraphobie traitée avec succès par la suggestion," *ibid.*, 1892-93, vii. 374.—28. Myers, F. W. H. "The Subliminal Consciousness," *Proc. Soc. for Psych. Res.*, London, 1893, ix. 3, 26.—29. Neilson, H. "Le Traitement hypnotique de la dipsomanie," *Rev. de l'hypnot. expér. et thérap.*, Paris, 1892, vi. 14.—30. Pau de St. Martin. "Thanatophobie, agoraphobie et divers troubles nerveux traités par la suggestion," *ibid.*, 1899-1900, xv. 17.—31. van Renterghem et van Eeden. *Psycho-thérapie*, Paris, 1894.—32. Robertson, G. M. "Use of Hypnotism among the Insane," *Journ. Ment. Sc.*, London, 1893, xxxix. 1.—33. Roubinovitch. "Le Traitement des habitudes vicieuses par la suggestion," *Rev. de l'hypnot. expér. et thérap.*, Paris, 1887-88, ii. 364.—34. von Schrenck-Notzing. *Die Suggestionstherapie bei krankhaften Erscheinungen des Geschlechtsinnes*, Stuttgart, 1892.—35. Smith, R. P., and Myers, A. T. "On the Treatment of Insanity by Hypnotism," *Journ. Ment. Sc.*, London, 1890, xxxvi. 191.—36. Tuckey, C. Lloyd. *The Value of Hypnotism in Chronic Alcoholism*, London, 1892.—37. Velander (Jonköping). "Un Cas de mutisme mélancholique guéri par suggestion," *Compt. rend. du premier Cong. intern. de l'hypnot. expér. et thérap.*, Paris, 1889, 323.—38. Voisin, A. "De l'hypnotisme et de la suggestion hypnotique dans leurs applications au traitement des maladies nerveuses et mentales," *Rev. de l'hypnot. expér. et thérap.*, Paris, 1886-87, i. 4.—39. *Idem.* "Les Indications de l'hypnotisme et de la suggestion hypnotique dans le traitement des maladies mentales et des états connexes" (discussion by Forel and others), *Compt. rend. du premier Cong. intern. de l'hypnot. expér. et thérap.*, Paris, 1889, 147.—40. *Idem.* "Emploi de la suggestion hypnotique dans certaines formes d'aliénation mentale," *Communication faite au Congrès internat. de Psychologie*, Munich, 1896.—41. *Idem.* "De la dipsomanie et des habitudes alcooliques et de leur traitement par la suggestion hypnotique," *Rev. de l'hypnot. expér. et thérap.*, Paris, 1887-88, ii. 48.—42. Wetterstrand, O. G. *Der Hypnotismus und seine Anwendung in der praktischen Medicin*, Wien u. Leipzig, 1891.—43. *Idem.* "Die Heilung des chronischen Morphinismus, Opiumgenüsses, Cocaïnismus, und Chloralismus mit Suggestion und Hypnose," *Ztschr. f. Hypnotismus*, Leipzig, 1896, iv. 9.

J. M. B.

CRIMINAL LUNACY IN ENGLAND

By David Nicolson, C.B., M.D.

In his *History of the Criminal Law of England*, Sir J. Fitz-James Stephen defines Crime as "an act or omission, in respect of which legal punishment may be inflicted on the person who is in default, either by acting or omitting to act." The same authority describes the Criminal Law as that part of the law which relates to the definition and punishment of acts or omissions which are punished as being (i.) attacks upon public order, internal or external ; or (ii.) abuses or obstruction of public authority ; or (iii.) acts injurious to the public in general ; or (iv.) attacks upon the persons of individuals, or upon rights annexed to their persons ; or (v.) attacks upon the property of individuals or rights connected with, and similar to, rights of property.

The standard of conduct by which society expects members of the community to guide themselves is partly optional and partly obligatory. The observance of the optional rules of our everyday life may not always bring its own reward, but the power of inflicting punishment for transgressions of the obligatory rules constitutes the rigid means by which the law of the land is able to exert its authority in its endeavour to preserve and to safeguard public and personal rights. These punishable acts of transgression, or *crimes*, vary widely in their nature and in the amount and kind of injury which they inflict; the criminality finding its measure for the most part in the punishment awarded, regard being had in individual cases to previous conviction.

Speaking generally, serious crimes and minor crimes are respectively described as Indictable Offences and Non-indictable Offences. In the *Judicial Statistics*, Indictable Offences, which, with few exceptions, are tried at Assizes or Quarter Sessions, are taken as including "all serious offences which directly affect the person or property." They are 82 in number, and are divided into the following six classes:—(i.) Offences against the person. (ii.) Offences against property with violence. (iii.) Offences against property without violence. (iv.) Malicious injuries to property. (v.) Forgery and other offences against the currency. (vi.) Other Offences. Non-indictable Offences are tried summarily, and the great mass of them are acts which in their tendencies "are injurious to life or property or to the general welfare, but which cannot, as regards their immediate effect, be classified under the above specified heads." They include such offences as drunkenness, assaults, and vagrancy; and they also include offences against the game laws, the police regulations, and the Education Act.

Psychological Genesis of Crime.—In entering upon any systematic consideration of crime or criminality in its relation to normal mental life in individuals, we must, in the first instance, find some fairly stable estimate or standard of occurring crime, taken as a characteristic element in the constitution of our social life. I do not think we are likely to find a better standard than the 82 Indictable Offences upon which, as we have seen, is based the official classification of crimes, as given annually in the *Judicial Statistics, England and Wales.* This official classification is excellent in itself and well adapted for statistical comparisons as to the numerical amount and the character of crime in successive years; but as a preliminary to our present inquiry I found it necessary to rearrange the six classes into four groups, and ultimately to tabulate them under three primary divisions. Upon the returns given by the "Crimes committed" and the "Persons tried" in the five years 1889-93, I then marked out the percentage of persons tried in the various classes, groups, and divisions. I was careful, in carrying out this analytical condensation, to avoid making any such structural disruption in the classification as would dislocate the position of the official figures and percentages.

In the following table are given the crimes and percentage of cases in each of the three ultimate divisions:—

TABLE I.

Division.	Crimes.	Percentage of Cases in each Division.
I.	Violence to Person or Property .	15
II.	Sexual Offences . . .	10
III.	Offences against Property for spoil or gain	75

This simplification of crimes into three primary divisions discloses to us the composition of the psychological counterpart of classified crime. Taking "Crimes generally," this counterpart or equivalent comes out in the following proportions, the term "Propensity" (an extension of thought towards action) being used to express the outward activity in a criminal direction of three primary emotions or psychical states which are referred to under the generic term of "Dominant mental origin" (as regards crime).[1]

TABLE II.

Propensity (in Criminal Activity).	Dominant Mental Origin.	Percentage of Cases.
Thievish, etc. .	Acquisitiveness .	75
Malicious . .	Malice . . .	15
Lustful . .	Lust . . .	10

So far as crimes are concerned, it is shewn that acquisitiveness, malice, and lust are the three dominant seats of origin in the mind, and in this threefold centre, therefore, crime is to be regarded as having its genesis. Their contiguous and allied emotions and ideas come into play in varying proportions in different individual criminal offences. To take one instance: just as the "dominant" malice stretches away down through hatred, resentment, anger, spitefulness, and the like, so does crime against the person and property stretch away down from murder, through manslaughter or assault with intent, to cruelty and neglect, or to an act of incendiarism or of cattle-maiming. And so in the cases of acquisitiveness and of lust.

In Table III. is shewn the development of the scheme by which crimes (Indictable Offences) are traced to their origin in the minds of criminals. This scheme may, I hope, be of use in guiding us to a practical interpretation of the meaning or value of the word "Crime," which, as an abstract expression, is apt to be used vaguely and indefinitely. For instance, it is, I think, remarkable to find, in regard to crime, that as much as 75 per cent of it has reference to the acquirement of money or property by thievish and fraudulent means; or, in other words, that more than seven-tenths of our crime stands in some relation to the means used for earning a livelihood. The further question of the *criminality*—or the moral intensity in a criminal direction—of any indictable offence, has reference to the external circumstances in which the specific act is committed, and to the motive which more directly led up to it. The criminality of the man who steals some bread for his starving family is to be viewed in a very different light from that of the man who robs his master to pay for an outing (*vide* also p. 868).

---

[1] Throughout this article Indictable Crimes only are dealt with, unless it is otherwise so stated.

The criminal-like instinct for acquisitiveness or for violence is to be regarded as a factor of all mental life from its birth to its close, without limitation as to social status or as to the kind of ancestral or parental heritage or environment. This holds good, too, as regards the instinct for lustful indulgence, nature, however, having postponed for a few years the first development in individuals of the sexual passion, the feature of the mental life which leads up to the third dominant origin of crime. This threefold (criminal-like) instinct is a universal birthright which man shares with all animals; and it would be our natural and more or less prevailing characteristic through life, were it not for the gradual development in us of certain higher and inhibitory intellectual and volitional processes, such as prudence, reflection, and a sense of moral duty. These processes go to make up that self-controlling capacity which in the conflict of motives steers us aright and prevents the dominant emotion of the moment from exploding, or expending itself in some form of crime or vice. (*Vide* art. "Vice, Crime, and Insanity," p. 855.)

In proportion as this development is prevented, or stifled, whether from original brain defect or from lack of proper education and training, so is there a risk of the individual lapsing into criminal-mindedness or into actual crime. And this risk becomes intensified if the parents and their associates are people of vicious or drunken habits. Lastly, risk becomes compulsion when parents of the criminal class wilfully educate and train their children to a life of crime.

Whatever value we may attach, and rightly attach, to the influence of heredity as regards the quality of the brain in individuals, there can be no doubt of the value and influence of domestic environment, education, and training in moulding and forming individual character during the more plastic periods of infancy, childhood, and youth.

The so-called Science of Criminal Anthropology.—The factors with which we have been dealing prove stumbling-blocks in the way of "Criminal Anthropologists" and "Criminalists," who fail to give sufficient weight to them in building up their criminological science. Writers on

TABLE III.—The Psychological Genesis of Crime.

Crimes generally.

Number of Indictable Offences.	Class.	Description of Crimes.
26	I.	Offences against the person.
10	II.	Offences against property with violence.
14	III.	Offences against property without violence.
8	IV.	Malicious injuries to property.
4	V.	Forgery and offences against the currency.
20	VI.	Other offences.
82		

Group.	Description of Crimes.	Percentage of Persons tried.
I.	Offences against the person :	21·5
	(*a*) Crimes of violence.	12·2
	(*b*) Sexual Crimes.	9·2
II.	Offences against property for spoil or gain.	71·4
III.	Malicious injuries to property.	2·2
IV.[1]	Other Offences.	4·7

Division.	Propensity (in Criminal Activity).	Percentage of Cases.	Dominant Mental Origin (Primary Emotion).
I.	To violence to person or property.	15	Malice.
II.	To sexual acts.	10	Lust.
III.	To thieving, fraud, etc.	75	Acquisitiveness.

---

[1] Group IV., which contains such offences as extortion, perjury, piracy, indecent exposure, and poaching, is absorbed in the three divisions, and fractional portions in the percentages of cases are merged in the general totals.

"criminology" give us a copious and precise history of the anatomical configuration, the physiological eccentricities, the muscular anomalies, the complexion, the shape of the ear and the jaw, the tattoo marks, and so forth, in certain criminals. We get a striking and elaborated account of their many and fearful crimes, of their atrocious mental peculiarities and of their hideous moral obliquities. This analytical and biological process is applied by those who call themselves criminalists to a comparatively small group of criminals; and by implication, and even more directly, it is made applicable to criminals generally. The criminalist searches for anatomical, physiological, intellectual, and moral evils and obliquities in the structure and personality of the criminal; he finds them, he tabulates them. He does not seek for good; therefore he neither finds it nor tabulates it. If we are to give the devil his due, why not the criminal? The anthropological method of estimating the criminal must fail, because it does not include circumstance and motive in the computation, and because without these no standard of capacity, conduct, or responsibility can be regarded as trustworthy, or even possible. Again, if the criminalist tells us no more and no less than the truth, and if the criminal is what he would have us believe, our hopes for the betterment of the class by education and training, and for the reformation of the individual by punitory measures, prison discipline, industrial occupation, and other available means are crushed.

We have seen what a large proportion of crimes is due to acquisitiveness, and this close relationship of "crime" to the means used by criminals for obtaining a livelihood must not be overlooked; it is not for us to stamp "criminals" as lunatics or quasi-lunatics, or to place them on a special morbid platform of mental existence, merely because they prefer thieving, with all its concomitant risk, to more reputable, if more laborious, modes of maintaining themselves.

Who is to say that the professional thief who is caught picking a pocket and does his "three months hard" is in reality more of a criminal than the fraudulent tradesman or financier who swindles families wholesale during the week, and collects the offerings in church on Sunday? Where do the anatomical peculiarities of the latter class find a place on the criminal anthropologist's register? A criminal is a person who commits a crime, and not merely a person who is sentenced to imprisonment for committing a crime.

My objection to these criminological methods does not rest only on the injustice of crediting criminals generally with characteristics, anatomical, physiological, or mental, culled from the selected biographies of a few of the class under the guise of a so-called science. I object to it because it is not only useless but also misleading to us when we seek to apply it in detail in individual cases. I hope the day will never come when, in our official examination into the mental condition of suspected persons, or of persons lying in prison upon a criminal charge, we as medical men will be expected to produce our craniometer for the head measurements, and to place reliance upon statistical information as to the colour, size, or shape of any organ.[1] A man is sane or insane, criminal or lunatic, apart from and without regard to such sources of information. Each case must be taken on its own merits, and above all, and first of all, the man must be allowed to speak for himself, and to give his own account of the circumstances in which the act was committed and of the motives which led him to commit it (*vide* also p. 872).

Criminal Lunacy.—Having considered briefly some of the relationships existing between crime and criminality on the one hand, and the normal mental life in individuals on the other, we now pass on to a consideration of some points bearing on the crimes which are the outcome of mental unsoundness, and on criminal lunacy and criminal lunatics generally.

According to the statutory definition, "criminal lunatic" means any of the following persons:—

(*a*) Any person for whose safe custody during His Majesty's pleasure His Majesty or the Admiralty is authorised to give order; and

(*b*) Any prisoner whom a Secretary of State or the Admiralty has in pursuance of any Act of Parliament directed to be removed to an asylum or other place for the reception of insane persons.

It may here be observed that this statutory or official definition of a criminal lunatic has reference essentially to the responsible authority by whom certain classes of persons are dealt with; and differs from what may be called the technical or professional meaning of the term, in which crime and lunacy are the essential ideas. Technically, and in practice, the term "criminal lunatic" is not applied except to a person who has committed a crime and who is for that reason under special detention as a lunatic; and it matters not whether the priority of occurrence was with the insanity or with the criminal act, that is, whether the individual in question was insane when he committed the crime or was a criminal when he became insane. Socially, of course, and in some senses psychologically (as in the matter of treatment), it is not unimportant to distinguish between an individual who while insane commits a crime, and a criminal —and more especially a habitual criminal—who becomes insane while undergoing imprisonment; in short, between a criminal lunatic proper and an insane criminal.

In England and Wales on the 31st December 1907, there were, according to the *Judicial Statistics* for that year, 976 criminal lunatics under confinement. Of this total 736 were males and 240 were females, a proportion of 3 males to 1 female. With only 8 exceptions all had committed Indictable Offences.

The tabular statement on pp. 1024, 1025, taken from the *Judicial Statistics*, shews the offences committed by these criminal lunatics. It also gives their classification as to the stage or period at which the insanity was officially recognised.

With only 8 exceptions all the criminal lunatics of the country

---

[1] This, of course, is a totally different question from that of the utility of "anthropometry" in the identification of habitual criminals.

Table IV.—Criminal Lunatics, Offences, and Classification.

Offences.	Numbers detained on 31st December 1907.						
	Total Number.			Classification.			
				King's Pleasure Lunatics.		Secretary of State's Lunatics.	
	Total.	Males.	Females.	Insane on Arraignment.	Acquitted on ground of Insanity, or Special Verdict of Guilty but Insane.	Certified Insane before Trial.	Certified Insane after Trial.
Indictable Offences.							
1. Murder	433	270	163	97	255	42	39
2. Attempt to Murder . .	168	135	33	42	107	9	10
3. Threats or Conspiracy to Murder	8	8	...	4	4	...	...
4. Manslaughter . . .	31	26	5	10	7	5	9
5. Felonious Wounding . .	50	46	4	10	28	4	8
6. Endangering Railway Passengers	2	2	...	...	2	...	..
8. Malicious Wounding (Misdemeanours) . . .	46	41	5	7	37	2	...
9. Assault	4	4	..	1	3	...	.
11. Cruelty to Children . .	1	...	1	...	...	...	1
13. Child Stealing . . .	2	1	1	...	1	1	...
15. Concealment of Birth . .	2	...	2	1	1	...	...
16. Unnatural Offences . .	13	13	...	9	2	...	2
17. Attempts to commit Unnatural Offences . .	6	6	...	2	3	..	1
18. Indecency with Males . .	2	2	...	1	1	...	...
19. Rape	6	6	...	5	...	1	...
20. Indecent Assaults on Females	19	19	...	13	5	...	1
21. Defilement of Girls under 13	12	12	...	6	1	...	5
22. Defilement of Girls under 16	3	3	...	1	1	1	...
28. Burglary	12	12	...	1	3	3	5
29. Housebreaking . . .	7	7	...	1	1	...	5
30. Shopbreaking . . .	11	11	...	1	1	1	8
32. Entering with intent to commit Felony . . .	3	3	...	2	...	...	1
34. Robbery	6	6	...	1	...	...	5
35. Extortion by Threats to Accuse	1	1	...	...	...	...	1
36. Extortion by Threats . .	1	1	...	...	...	...	1
37. Larceny of Horses and Cattle	3	3	...	...	2	...	1
38. Larceny from the Person .	4	4	...	1	...	...	3
39. Larceny in House . .	1	1	...	1	...	...	...
42. Larceny of Post Letters .	1	1	...	...	..	...	1
44. Simple Larceny and minor Larcenies . . .	18	14	4	7	5	1	5
45. Obtaining by False Pretences	12	10	2	3	1	2	6
48. Other Frauds . . .	2	1	1	1	1	...	...
49. Receiving Stolen Goods .	1	1	...	...	...	...	1
51. Arson	35	33	2	10	12	3	10
53. Killing and Maiming Cattle	2	2	...	1	1	..	...
54. Malicious Use, etc., of Explosives	1	1	...	...	1	...	...
58. Other Malicious Injuries .	7	6	1	4	3	...	...
59. Forgery and Uttering (Felony)	2	2	...	..	2	..	...
62. Uttering Counterfeit Coin .	1	...	1	...	...	1	...
63. High Treason . . .	2	2	...	...	2	...	...
70. Perjury	...	...	...	...	...	...	...
Carry forward . . .	941	716	225	243	493	76	129

Table IV.—*Continued*

Offences.	Numbers detained on 31st December 1907.						
	Total Number.			Classification.			
				King's Pleasure Lunatics.		Secretary of State's Lunatics.	
	Total.	Males.	Females.	Insane on Arraignment.	Acquitted on ground of Insanity, or Special Verdict of Guilty but Insane.	Certified Insane before Trial.	Certified Insane after Trial.
Brought forward	941	716	225	243	493	76	129
76. Libel	3	1	2	2	...	...	1
78. Indecent Exposure	1	...	1	1	...	...	...
80. Other Nuisances	...	...	...	...	...	...	...
81. Habitual Drunkenness	11	2	9	...	...	1	10
82. Suicide (Attempting to Commit)	12	9	3	3	4	3	2
Total, Indictable Offences	968	728	240	249	497	80	142
NON-INDICTABLE OFFENCES.							
Assaults :—							
Aggravated	...	...	...	...	...	...	...
On Constable	...	...	...	...	..	...	...
Common	...	...	...	...	...	...	...
Cruelty to Children	...	...	...	..	...	...	...
Intoxicating Liquor Laws, Offences against :—							
Drunkenness, Simple	...	...	...	...	...	...	...
Drunkenness, with Aggravations	1	1	...	...	...	...	1
Malicious Damage :—							
Other Offences	...	...	...	...	...	...	...
Military and Naval Law, Offences against :—							
Navy	1	1	...	...	..	...	1
Police Regulations, Offences against :—							
Town Police Clauses Acts	1	1	...	...	...	..	1
Poor Law, Offences against :—							
Neglecting to Maintain Family, etc.	...	...	...	...	...	..	...
Misbehaviour by Paupers	1	1	...	...	...	...	1
Stealing or Destroying Workhouse Clothes	...	...	...	...	...	...	...
Prevention of Crimes Acts :—							
Offences by Licence Holders	..	...	...	...	...	...	...
Unlawful Possession	..	...	...	...	..	...	...
Vagrancy Acts, Offences against :—							
Begging	3	3	...	...	...	...	3
Sleeping Out	...	...	...	...	...	...	..
Frequenting	...	...	...	...	...	...	...
Total, Non-Indictable Offences	7	7	...	...	..	...	7
Surety Prisoners	1	1	...	—	—	—	1
Grand Total — Males	...	736	...	193	352	57	133
Grand Total — Females	...	...	240	56	145	23	17
Grand Total — Males & Females	976	736	240	249	497	80	150

committed Indictable Offences, and they are classified under two heads, "King's pleasure lunatics" and "Secretary of State's lunatics."

"King's pleasure lunatics" are criminal offenders of unsound mind not sentenced to any term of imprisonment, but ordered by a Court of Law to be detained during His Majesty's pleasure. King's pleasure lunatics are classified as having been either (*a*) found insane on arraignment, or (*b*) acquitted on the ground of insanity or (since "Trial of Lunatics Act," 1883) found guilty but insane; the numbers being 249 of the former to 497 of the latter.

"Secretary of State's" lunatics are criminal offenders of unsound mind who are detained upon the authority of the Secretary of State, having been certified insane (*a*) before trial or (*b*) after trial and sentence (to death, penal servitude, or imprisonment); the numbers being 80 of the former to 142 of the latter.

All criminal lunatics are detained for indefinite periods, with the exception of those undergoing sentences to penal servitude or shorter terms of imprisonment; the latter at the expiration of their sentence cease to be "criminal" and become "pauper" lunatics.

The following table gives the results of analytical inquiry into the mental origin of the crimes (Indictable Offences) of criminal lunatics:—

TABLE V.—Psychological Genesis of Crime.

Crimes of Criminal Lunatics.

Division.	Propensity (in Criminal Activity).	Percentage of Cases.	Dominant Mental Origin (Primary Emotion).
I.	To Violence to Person or Property	86	Malice.
II.	To Sexual Acts	5	Lust.
III.	To Thieving, Fraud, etc. . .	9	Acquisitiveness.

The following interesting and instructive comparison shews that the criminal tendency in the sane is towards *plunder*, whilst in the insane it is towards *violence*:—

TABLE VI.

Division.	Percentage: Crimes generally.	Percentage: Crimes of Criminal Lunatics.
I. Violence .	15	83
II. Sexual Acts .	10	7
III. Thieving, etc.	75	10

Population of Broadmoor Criminal Lunatic Asylum.—Of the 968 criminal lunatics (Indictable Offences), as many as 792 were under detention in Broadmoor Asylum. The remaining 176 consisted of 47 penal servitude prisoners detained in the lunatic wards of Parkhurst Prison and 129 persons sentenced to imprisonment for minor offences who were distributed throughout 51 county and borough asylums. These will not be further referred to.

The statistics referring to Broadmoor are taken from the report on that Asylum for the year 1908.

Table VII. on p. 1028 gives the crimes and classification of the patients in the Asylum on the 31st December 1908.

A glance at the table shews that out of the 801 inmates as many as 694 inmates were under detention for grave crimes of personal violence, namely, murder, manslaughter, and attempt to murder; leaving only 107 under detention for other offences.

Of the 801 patients, 457 had committed homicide (429 cases of murder and 28 of manslaughter). Of these, 284 were men and 173 were women. With few exceptions the death caused by the women was that of their own child or children.

The proportion of cases of homicide for the whole population was 57 per cent, for the male inmates alone 49·3 per cent, and for the female inmates alone 77·2 per cent. If we take the whole 968 criminal lunatics (Indictable Offences) in Broadmoor and other asylums, the proportion of homicidal cases is 47·9 per cent, very nearly one-half.

The Broadmoor population, at the end of 1908, included 41 inmates who were certified to be insane while undergoing sentences of penal servitude or imprisonment, leaving 760 patients who may be regarded as having been insane at the time they committed their crimes.

The following is the numerical list of these crimes :—

Murder	413
Manslaughter	21
Attempt to murder	228
Other offences	98
	760

[TABLE

TABLE VII.—Crimes and Classification of Criminal Lunatics in Broadmoor on the 31st December 1908.

Crimes.	Total Number remaining in the Asylum on the 31st of December 1908.			Classified with reference to the period at which Insanity was recognised.																	
				Certified to be Insane whilst awaiting Trial or Judgment.			Found Insane by Jury on Arraignment.			Acquitted on the ground of Insanity, or found Guilty but Insane (in terms of "Trial of Lunatics Act, 1883").			Reprieved on the ground of Insanity.			Certified to be Insane whilst undergoing sentences of Penal Servitude.			Certified to be Insane whilst undergoing shorter Terms of Imprisonment.		
	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.
Murder	261	168	429	23	16	39	57	42	99	149	108	257	17	1	18	15	1	16	...	...	...
Attempt to murder, maim, etc.	198	39	237	9	...	9	42	9	51	138	29	167	...	...	...	[1]9	...	9	...	1	1
Threats or conspiracy to murder	8	...	8	...	...	...	4	...	4	4	...	4	...	...	...	...	...	...	...	...	...
Manslaughter	23	5	28	4	1	5	9	1	10	4	2	6	...	...	...	6	1	7	...	...	...
Endangering railway passengers	2	...	2	...	...	...	...	...	...	2	...	2	...	...	...	...	...	...	...	...	...
Assault, common	3	...	3	...	...	...	...	...	...	3	...	3	...	...	...	...	...	...	...	...	...
Concealment of birth	...	1	1	...	...	...	...	...	...	...	1	1	...	...	...	...	...	...	...	...	...
Desertion of child and child-stealing	1	1	2	...	1	1	...	...	...	1	...	1	...	...	...	...	...	...	...	...	...
Unnatural offences and attempts	15	...	15	...	...	...	8	...	8	6	...	6	...	...	...	1	...	1	...	...	...
Rape	5	...	5	1	...	1	4	...	4	...	...	...	...	...	...	...	...	...	...	...	...
Defilement of girls and indecent assaults on females.	11	...	11	1	...	1	7	...	7	2	...	2	...	...	...	1	...	1	...	...	...
Burglary and housebreaking	9	1	10	3	...	3	4	...	4	1	...	1	...	...	...	1	1	2	...	...	...
Larceny of horses and cattle	2	...	2	...	...	...	...	...	...	2	...	2	...	...	...	...	...	...	...	...	...
Larceny and petty thefts	7	1	8	...	...	...	4	1	5	2	...	2	...	...	...	1	...	1	...	...	...
Obtaining goods by false pretences	5	2	7	1	1	2	2	...	2	1	...	1	...	...	...	1	1	2	...	...	...
Arson and malicious burning	18	2	20	2	...	2	8	...	8	8	2	10	...	...	...	...	...	...	...	...	...
Killing and maiming cattle	2	...	2	...	...	...	1	...	1	1	...	1	...	...	...	...	...	...	...	...	...
Forgery and uttering (felony)	2	...	2	...	...	...	...	...	...	2	...	2	...	...	...	...	...	...	...	...	...
Treasonable and seditious offences	2	...	2	...	...	...	...	...	...	2	...	2	...	...	...	...	...	...	...	...	...
Libel	1	1	2	1	...	1	...	1	1	...	...	...	...	...	...	...	...	...	...	...	...
Attempted self-murder	1	3	4	...	...	...	...	1	1	1	2	3	...	...	...	...	...	...	...	...	...
Under the Vagrant Laws	1	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	1
Total	577	224	801	45	19	64	150	55	205	329	144	473	17	1	18	35	4	39	1	1	2

[1] Of these one received a conditional pardon.

The following are the principal crimes in the 98 cases in which the offences were other than murder, manslaughter, and attempt to murder, and in which the offenders were insane at the time of the act:—sexual offences, 29; arson, 20; burglary, housebreaking, and larceny, 22.

The following are the principal offences of the 41 insane convicts ("insane criminals") under detention in Broadmoor on the 31st December 1908:—

Murder	16
Manslaughter	7
Attempt to murder	10
Sexual offences	2
Larceny, etc.	6
Total . . .	41

Murder Cases.—The classification of the cases of murder by insane persons with reference to the stage at which their insanity was officially recognised gives the following figures. It may be observed that the death or discharge of the total murder cases admitted did not to any great extent alter the percentages in each class of those remaining under detention.

TABLE VIII.[1]—Murders by Insane Persons.

Class.	Stage at which Insanity was officially recognised.	Murder Cases under detention in Broadmoor on 31st December 1908.		Total number of Murder Cases admitted into Broadmoor from date of opening in 1863 till end of 1908.	
		Total.	Percentage.	Total.	Percentage.
I.	Certified to be Insane whilst awaiting Trial or Judgment	39	9·4	100	11·0
II.	Found Insane by Jury on Arraignment . . .	99	23·97	202	22·4
III.	Acquitted on the ground of Insanity or found Guilty but Insane (in terms of "Trial of Lunatics Act, 1883")	257	62·2	555	61·5
IV.	Reprieved on the ground of Insanity	18	4·35	44	4·88
		413	100	901	100

Of the 413 insane persons who committed murder, 246 were men and 167 were women.

---

[1] This table does not include murder cases in which the death penalty was commuted to penal servitude for life, and in which the convict was transferred to Broadmoor from a convict prison.

Table IX. gives the degree of relationship of the persons killed to the lunatics who took their life.

TABLE IX.—Broadmoor Homicide Cases. Degree of Relationship or otherwise of Persons killed.

Degree of Relationship or otherwise of those whose Deaths were caused.	In Asylum 31st December 1908.		
	M.	F.	Total.
Father	2	...	2
Father and another person	1	...	1
Step-father	2	...	2
Mother	15	2	17
Both parents	1	...	1
Husband	...	2	2
Wife	60	...	60
Wife and child or children	8	...	8
Own child or children	48	149	197
Own child and step-daughter	1	...	1
Own child and police officer	1	...	1
Brother or brothers	6	...	6
Brother-in-law	3	...	3
Sister	3	...	3
Sister and her child	2	...	2
Sister-in-law	1	...	1
Uncle	2	...	2
Aunt	2	...	2
Nephew	4	1	5
Niece	2	3	5
Grandchild	2	2	4
Man or woman with whom cohabiting	6	1	7
Woman to whom engaged	4	...	4
Fellow-patients in asylums	18	2	20
Fellow-inmates of workhouses	4	...	4
Fellow-prisoner	1	...	1
Attendant	5	...	5
Police officer in charge	1	...	1
Surgeon attending	1	...	1
Other persons while under care as lunatics	2	1	3
Other persons	76	10	86
Total	284	173	457

It will be seen that 149 women and 48 men took the lives of their own child or children, 15 men and 2 women killed their mother, 60 men killed their wife, and 8 their wife and child or children. Eighteen men and 2 women killed fellow-patients in asylums, and 5 men killed asylum attendants.

Amongst the female inmates there were only 24 cases out of a total of 173 in which the person killed was other than their own child. These figures all refer to inmates of Broadmoor.

With the view of arriving at a comparative statement of the degree of relationship in cases of murder by men, I have drawn up the following table which shews the relationship of the person killed in 200 cases of men executed, and 200 cases of men found insane. The table also shews the mode of killing in these cases; but the information arrived at is of a negative character and to the effect, as one would have expected, that the mode of killing affords no guide to the insanity or otherwise of the murderer—unless perhaps it may be noted that poison is administered in 7 cases by sane men, in 2 cases by insane men.

TABLE X.—Murder Cases: shewing Relationship of Person killed and Mode of Killing.

Relationship.	200 Men Executed.	200 Men found Insane.	Mode of Killing.	200 Men Executed.	200 Men found Insane.
Father	2	5	Cutting throat	51	67
Mother	1	11	Shooting	42	40
Mother or sister	0	7	Beating on head, etc.	52	48
Wife	52	45	Stabbing, cutting, etc.	23	23
Own child or children	7	43	Strangulation or suffocation	10	6
Other ,, ,,	11	11			
Other adult relatives	3	6	Drowning	4	5
Women with whom cohabiting	27	6	Poisoning	7	2
Other women	32	16	Kicking, etc.	4	3
Policeman, warder, gamekeeper, etc.	26	3	Other modes	7	6
Other men	39	47			
	200	200		200	200

Where close blood-relationship existed, insanity was established in the great majority of cases; thus, of 76 men who murdered their father, mother, brother, sister, or own child, as many as 66 were found to be insane, a ratio of more than 6 to 1. Cases of wife-murder supply some of the most complex problems in medical jurisprudence when the question of insanity is raised, because of the difficulty that often exists in proving satisfactorily whether the jealousy which in most cases has led to the murder has any foundation in fact, or was merely the outcome of insane delusion. And curiously enough the results shew a very even balance; for of 97 cases of wife-murder (a large proportion, be it noted, out of 400 murders) 52 men were executed and 45 were found to be insane. Whilst, therefore, there would appear to be a 6 to 1 element of presumption in favour of insanity where a close blood-relation is murdered, there is rather more than an even chance in favour of execution for a wife-murderer as compared with a verdict of insanity. Again, where men murder women who are not their wives, their chances of being executed and not found insane are very nearly 3 to 1. Where a policeman or warder, or other person in authority, is murdered, a verdict of insanity would appear to be very unlikely; whilst if one man murders another man, the chances are slightly in favour of his being found insane, as compared with the chances of his being hanged for his crime. It must be noted, however, that all these proportions refer to a verdict of the insanity, as compared with the execution, of the individuals; and that they do not take into account the fairly numerous cases in which for various reasons a murderer is neither found insane nor executed, but is sent to penal servitude for life.

The Discharge of Criminal Lunatics.—The Criminal Lunatics Act, 1884, sect. 5 (2), provides that "a Secretary of State may absolutely discharge any criminal lunatic, and may also discharge any criminal lunatic conditionally, that is to say, on such conditions as to duration of such discharge and otherwise as the Secretary of State may think fit."

Since the opening of the Asylum in 1863, up to the end of 1908, 184 out of about 940 murder cases admitted into Broadmoor were discharged conditionally on Secretary of State's Warrant to the care, in nearly all the cases, of relatives who undertook to report if any signs of a relapse shewed themselves. As would naturally be expected, the great bulk of these, numbering 126, were women who, in various circumstances, but mostly at puerperal or lactational periods, had taken the lives of their children. It would, on the one hand, be too terrible if Broadmoor were to be made a place for the abandonment of all hope of discharge, whilst, on the other, the utmost care has to be taken in order that the persons and property of the community should be safeguarded. Each case is considered on its own merits, and it is not until after careful and prolonged observation of the individual on the part of the responsible authorities that a favourable decision is arrived at in the matter of release; and stringent conditions are imposed upon those into whose care recovered cases are sent. Official cognizance of those discharged is kept up for years, and it is very satisfactory to be able to record that in no instance has the crime been repeated by homicides who have been set at large.

Of the 184 persons (58 men and 126 women) discharged recovered from Broadmoor (murder cases), 42 (12 men and 30 women) had to be re-admitted on relapse, and in several of these cases the individuals came back of their own accord, being afraid to trust themselves, or feeling unequal to the battle of life. Two or three other discharged patients found their way into ordinary asylums on relapse, but the number of relapses affords satisfactory testimony of the care exercised in discharging cases; whilst the safe return to asylum of relapsed cases shews the vigilance exercised in carrying out the conditions of release by those who undertake the duty of supervision.

The following are the chief points that have to be considered and weighed before the Secretary of State sanctions the release of a criminal lunatic:—(i.) The crime, its nature, and the circumstances in which it was committed. (ii.) The insanity, its character and history; whether hereditary or due to any special cause, and whether first attack. (iii.) The duration of the attack of insanity. (iv.) Whether relapse occurred, and if so, how brought about. (v.) The length of time since last attack, or since last active indication of insanity. (vi.) The completeness of the recovery, and whether there remain any after mental effects, such as weak-mindedness, undue irritability, or suspiciousness. (vii.) The likelihood of remaining well if out in the world. A person who has committed murder and been for years in an asylum is somewhat handicapped in starting social life again, and may not unnaturally shew some sensitiveness to his position. (viii.) Capacity for earning livelihood or contributing to own maintenance; industrious disposition or otherwise; whether a former master or other person has offered employment. (ix.) Disposition to drink, often a difficult inquiry. (x.) The home he goes to, and the kind of person or persons who undertake to look after him and report periodically, usually every three months to begin with, as to his condition, or immediately in the event of signs of relapse shewing themselves.

In proportion as the answers to all these points are satisfactory would a criminal lunatic's chance of conditional discharge be likely to receive favourable consideration.

In Table XI. will be found a complete and interesting summary of the changes occurring in the population of the Criminal Lunatic Asylum at Broadmoor since it was opened in 1863 up to the end of 1908. It records the admissions, discharges, deaths, recoveries, and escapes. Taking the five years ending with 1908 as representing a normal period, the average annual admissions (all forms of crime included) were 51 in number. For the whole forty-five years ending with 1908 the average annual number of patients discharged recovered was 7·8; while the average annual percentage of deaths on the average number resident was, for the same period, 2·9.

The Insanities of Criminal Lunatics.—So far as this country is concerned, I am not aware that there are any trustworthy statistics on the insanities of criminal lunatics; but in any case it is not within the scope of this article to do more than refer in brief and general terms to the subject.

The tendency — I might say the essential characteristic — of all lunacy is to set at naught first and most frequently the *optional* and then the *obligatory* rules of conduct; and the object of all lunacy legislation from a public standpoint is to prevent the lunatic from becoming actually dangerous to himself or others—to prevent him, in short, from becoming a criminal lunatic. No stronger confirmation of the truth of this is needed than that as much as 86 per cent—or let us say the great bulk—of the crimes of criminal lunatics are crimes of violence.

The criminal group of lunacy cases is characterised by the fact that the mental condition of the individual, his sanity or otherwise, has to be estimated primarily in its relation to an act of criminal import committed by him. This criminal act introduces the idea of responsibility and punishability, and the consequences of a finding of sanity or insanity differ materially from the consequences in cases into which the criminal element does not enter. Criminal lunacy cases may be divided into non-capital and capital. In non-capital cases there is the alternative of prison

TABLE XI.—Shewing the Admissions, Discharges, Deaths, and Escapes, with the Mean Annual Mortality and the Proportion of Recoveries since the opening of Broadmoor Asylum.

Years.	Admitted.									Of each Year's Admissions discharged, died, or otherwise disposed of in the Year 1908.															Total discharged, died, or otherwise disposed of, of each Year's Admissions from the opening of the Asylum to 31st December 1908.															Remaining of each Year's Admissions on the 31st December 1908.		
	New Cases.			Re-admissions.			Total Admissions.			Recovered and discharged.			Recovered and removed to Prisons.			Transferred to other Asylums, Workhouses, or Private Care.			Died.			Escaped during the Year and not recaptured.			Recovered and discharged.			Recovered and removed to Prisons.			Transferred to other Asylums, Workhouses, or Private Care.			Died.			Escaped and not recaptured before 31st Dec. of Year of Escape.					
	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.	M.	F.	T.
From 27th May 1863 to 31st Dec 1898.	1741	533	2274	60	23	83	1801	556	2357	..	..	..	..	..	..	1	..	1	23	1	24	..	..	..	157	143	300	94	18	112	683	158	841	569	138	707	6	1	7	292	98	390
1899 . .	30	8	38	3	3	6	33	11	44	1	1	2	..	..	..	..	..	..	..	..	..	..	..	..	8	2	10	..	1	1	6	1	7	6	1	7	..	..	..	13	6	19
1900 . .	32	7	39	2	2	4	34	9	43	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	2	1	..	1	7	1	8	3	1	4	..	..	..	23	5	28
1901 . .	29	21	50	..	3	3	29	24	53	..	..	..	..	..	..	1	..	1	..	..	..	..	..	..	4	7	11	..	1	1	3	..	3	5	2	7	..	..	..	17	14	31
1902 . .	93	18	111	3	2	5	96	20	116	..	..	..	..	..	..	1	..	1	..	..	..	..	..	..	4	7	11	3	1	4	38	1	39	8	..	8	..	..	..	43	11	54
1903 . .	54	15	69	5	5	10	59	20	79	1	2	3	..	..	..	1	..	1	..	..	..	..	..	..	3	3	6	6	2	8	8	1	9	5	1	6	..	..	..	37	13	50
1904 . .	32	18	50	2	1	3	34	19	53	..	1	1	..	..	..	..	..	..	1	..	1	..	..	..	4	3	7	..	1	1	..	..	..	2	1	3	..	..	..	28	14	42
1905 . .	31	17	48	4	3	7	35	20	55	..	2	2	..	..	..	..	..	..	2	..	2	..	..	..	2	3	5	1	..	1	..	..	..	3	1	4	..	..	..	29	16	45
1906 . .	36	10	46	1	2	3	37	12	49	..	..	..	..	..	..	1	..	1	..	..	..	..	..	..	..	1	1	1	..	1	1	..	1	1	1	2	..	..	..	34	10	44
1907 . .	32	15	47	1	3	4	33	18	51	2	1	3	..	..	..	..	..	..	..	..	..	..	..	..	2	1	3	..	..	..	..	..	..	..	..	.	..	..	..	31	17	48
1908 . .	29	17	46	1	3	4	30	20	50	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	30	20	50
Total .	2139	679	2818	82	50	132	2221	729	2950	4	7	11	..	..	..	5	..	5	26	1	27	..	..	..	184	172	356	106	24	130	746	162	908	602	146	748	6	1	7	577	224	801

or asylum, whilst in capital cases (that is, murder cases) the death penalty brings to the medical psychologist a grave sense of responsibility which, if undertaken, must be carried out fully and honestly as a matter of duty, without sentiment or fads. The considerations which have to be weighed in criminal cases by the medical man are complicated by the possible presence of simulated insanity: and the graver the case and its consequences the greater the incentive to malingering. In cases in which the plea of insanity is raised we have to satisfy ourselves whether any abnormal, eccentric, or insanelike behaviour is the genuine and spontaneous outcome of the man's mental operations, or whether it is due to the feigning of a sane person seeking to avoid punishment for a criminal offence. In murder cases my experience has led me to be extremely suspicious of a man who asserts complete obliviousness of the murder and of the series of purposive acts connected with it.

What, we may ask, is the mental attitude of lunatics who commit crimes of violence? Putting aside the small percentage of violence to property committed by lunatics, consisting mostly of arson, the outcome of ill-conditioned imbecility, it will be found that personal violence by a lunatic is mostly referable either to mental exaltation or to mental depression in one or other of their varied forms, accompanied by delusion or hallucination.

In conditions of *mental exaltation or mania*, the delusion which leads to the violence is usually one of suspicion, persecution, or conspiracy. In some cases the insane mental excitement is comparatively transient, but leaves the delusion well marked and active; and these are cases which prove very difficult when an effort is being made to estimate the mental condition, *qua* sanity or insanity, of an individual at the time he committed a criminal act (not unusually murder or attempted murder). Given a case of wife-murder, the sincerity and circumstantiality of the prisoner's declarations as to her infidelity are in themselves no trustworthy guide either as to the fact about her or as to the existence or non-existence of insane delusion in him. The scope of the inquiry has to be extended in the direction both of the circumstances connected with the crime and of the prisoner's general mental condition up to and at the time he committed it. The possibility of imposture and feigning being borne in mind, the experienced medical man will be careful to commence his examination by dealing with general questions before he deals with the particular question as to the prisoner's reasons for committing the act.

In conditions of mental depression or *melancholia*, the delusion consists very frequently in an insanely exaggerated extension of religious feeling, whereby the patient comes to believe that she (for the case is more frequent among women) has committed "the unpardonable sin," is deserted by God, and is condemned to eternal damnation for the commission of fancied crimes and disgusting enormities. This delusion, with its accompanying sense of unworthiness, leads up, through a belief in her unfitness to live, to active suicidal ideas, in the contemplation of which she becomes strongly imbued with the feeling that she must not leave her child or children, more especially those of whom she is fondest, behind her to have to live the horrible life that is in store for them. She takes their lives, and in doing so her mind may come to a realisation of what she has done, and she may stop short at the intended self-destruction. Or, again, her delusions may include the fixed belief that her children are not hers; and an auditory hallucination may bring to her God's voice telling her to destroy them. Delusions of this type have many phases in their active outward expression; but the condition is characterised by despondency, apprehensiveness, self-absorption, and self-condemnation, and there is little difficulty in recognising its genuineness.

Epilepsy is not found to exist so frequently among criminal lunatics as might be expected. Out of a population of 801 criminal lunatics in Broadmoor, not more than 40 are classed as epileptic, a proportion of 1 in 20, or 5 per cent; the explanation, no doubt, being that persons suffering from epilepsy are, when at large, kept under observation and thereby prevented from committing criminal acts. Broadmoor has only 1 general paralytic and 6 paralytics amongst its population.

Drink is doubtless a potent factor, either directly or indirectly, in the production of crime and insanity; but it is very difficult in the consideration of its influence accurately to estimate how far it is the original, or, it may be, sole cause of the one or the other; or how far the drinking habit or drunkenness is more or less the effect of pre-existing mental weakness or nervous instability. Each case has to be considered separately; and whilst excessive indulgence in drink cannot be held to absolve from responsibility for criminal acts committed under its influence, there are cases, no doubt, in which the absolute rigidity of the rule may to some extent be justifiably departed from. I have no doubt whatever that it frequently happens that an ordinary lunatic or weak-minded person is converted into a criminal lunatic by the toxic action of alcohol upon his brain.

Causes of Criminal Lunacy.—The doctrines or hypotheses of the causation of insanity in general must be held to apply to criminal as well as to non-criminal insanity; and a study of the reasons why a lunatic becomes a criminal in one case and not in another, really resolves itself into a study of the circumstances in which a certain proportion of lunatics are allowed to become criminal lunatics.

One day before I left Broadmoor a friend of mine, one of Her Majesty's Inspectors of Schools, asked how the lunatics were getting on. I replied, "Oh, much as usual." He then volunteered his opinion (as so many do who have avoided any knowledge on the subject) on the "responsibility and crime" question by saying, "I would hang all lunatics who commit murder." "Well, but," I said, "if you had a grown-up son who had the misfortune to become insane, and if you neglected him, and if he by reason of his insanity and your neglect murdered some one, do you think hanging, then, the proper treatment for him?" "Ah," he said good-naturedly, "that is a home-thrust, and I'll say no more about the matter." And so it is: society stands *in loco* *parentis* to the lunatic, and perfect government would ideally imply a perfect supervision and care of all lunatics. Meanwhile our lunatics are to a large extent allowed to become criminal lunatics. That our criminal lunacy is largely preventable there is no doubt, and the responsibility for its occurrence is often traceable either to (*a*) *delay in certifying* an insane person for lack of evidence of insanity, or from fear of persecution or annoyance, or in a fatal deference to the pleadings of relatives; or to (*b*) *dilatoriness in removal* of certified lunatics to an asylum; or to (*c*) *premature discharge from an asylum.*

The number of murders and attempts to murder that result from the condition of things here mentioned is very considerable. There are walking about in our midst plenty of potential criminal lunatics whose insanity indeed is not unrecognised; nobody knows when it may be his turn to enact the part of victim in a realistic drama of blood.

Procedure in Criminal Cases in which Insanity is present or pleaded.—There are six stages at which a criminal offender may be officially recognised as insane and placed under detention as a criminal lunatic:—(i.) While awaiting trial. (ii.) On arraignment. (iii.) When found guilty of the act, but insane at the time it was committed. (iv.) While awaiting judgment. (v.) When reprieved on the ground of insanity. (vi.) While undergoing a term of penal servitude or shorter term of imprisonment.

Stage 1.—When the accused person is found to be insane while awaiting trial or under remand, and when the insanity is of such a character that he cannot properly be treated in prison, it becomes a necessary provision that he should be transferred to an asylum before his health suffers unduly. The individual on becoming sane is liable to be sent back to prison for trial, but this course is very seldom pursued.

Stage 2.—Here the accused person, if found by the jury to be insane and unfit to plead to the indictment, is ordered to be detained during His Majesty's pleasure. This is a wise provision; but it is extremely desirable that the prisoner, although insane, should be allowed to plead if he be at all capable of understanding the position he is in; for a trial gives him an opportunity of speaking through his counsel, and enables a decision to be arrived at as to his guilt or innocence. In one case a lunatic escaped from an asylum and killed a stranger on the roadside. At his trial he was able to shew that he acted in self-defence and he was acquitted and discharged. His friends were waiting and removed him back to the asylum.

Stage 3.—After the trial of Roderick Maclean for shooting at Queen Victoria, the "Trial of Lunatics Act, 1883" abolished the old verdict of acquittal on the ground of insanity in favour of a special verdict of guilty but insane at the time of the act. If in some senses this verdict is less logical than the old one, it has the advantage of definitely asserting the guilt of the prisoner. This stage is the only one where, in the process of making a criminal lunatic, the legal dictum (arising out of the Macnaghten case) of a "knowledge of right and wrong" is submitted to the jury in limitation of the area of insanity which ought to suffice, according to the law, in establishing the irresponsibility of the individual. The dictum is not a satisfactory one; and if it were insisted upon to the bitter end, it would have been the means of hanging more than half the women and many men now in Broadmoor as criminal lunatics.

After many wordy contests and much, perhaps needless, wrangling, I think the outcome has been that there is a general consensus of opinion that the legal dictum is too rigid on the one hand, and that medical opinion is too elastic on the other.

In 1894 a Committee of members was instructed by the Medico-Psychological Association to investigate and report on this most difficult and delicate question. The report of this Committee was submitted at the annual meeting of the Association in July 1896. After discussion it was unanimously agreed (quoting from the official account in the *Journal of Mental Science* for October 1896) that the "Report as amended be received and adopted by the Association." The concluding amended paragraph of the Report reads as follows:—"Under the circumstances disclosed by their investigation, your Committee, while not approving the doctrines and definitions contained in the Judges' answers to the House of Lords in 1843, are at present unable to make any recommendations for the amendment of the law."

Stage 4.—It very seldom happens that insanity intervenes between conviction and judgment. There have been only two such cases at Broadmoor.

Stage 5.—The Criminal Lunatics Act, 1884, sect. 2 (4), provides that in the case of a prisoner under sentence of death, if it appears to a Secretary of State, either by means of a certificate signed by two members of the visiting committee of the prison in which such prisoner is confined, or by any other means, that there is reason to believe such prisoner to be insane, the Secretary of State shall appoint two or more legally qualified medical practitioners, and the said medical practitioners shall forthwith examine such prisoner and inquire as to his insanity, and after such examination and inquiry such practitioners shall make a report in writing to the Secretary of State as to the sanity of the prisoner, and they, or the majority of them, may certify in writing that he is insane.

Every opportunity is given in this section of the Act for the question of insanity to be raised after sentence of death has been passed; and the Home Secretary is empowered to cause further medical inquiry to be made into the actual mental condition of the convict. If the prisoner under sentence of death is found to have been insane at the time the crime was committed, and if he is certified to be insane at the time of the statutory inquiry, he is removed to Broadmoor on the Secretary of State's Warrant. But if the prisoner is found to have been insane at the time he committed the crime, and if no certificate of his insanity at the time of the inquiry can be given, the death sentence is commuted to one of penal servitude for life.

It may be to some persons a matter of surprise that in not more than 44 cases (42 men and 2 women) has the death sentence been reprieved on the grounds of insanity and the prisoner sent direct to Broadmoor; and this over a period of upwards of 45 years, during which time close upon 940 criminal lunatics who have committed murder were admitted into Broadmoor.

Stage 6.—Forty-one prisoners undergoing penal servitude (convicts) or shorter terms of imprisonment were under detention as criminal lunatics in Broadmoor on 31st December 1908. Of these, 33 had committed crimes of violence to the person, including 12 who had committed murder. The rest of the penal servitude (convict) criminal lunatics are detained in the lunatic wards of Parkhurst Prison.

The "Criminal Appeal Act, 1907" is described as "An Act to establish a Court of Criminal Appeal and to amend the Law relating to Appeals in Criminal Cases"; and it will have the effect of relieving the Home Secretary of some of his unenviable responsibility in cases in which the death penalty has been pronounced. In this sense it may be regarded as an adjunct to stage 5 of the procedure which I have described as holding good in dealing with criminal lunacy cases. The Act makes the following provision, section 5, subsection 4: If on any appeal it appears to the Court of Criminal Appeal that, although the appellant was guilty of the act or omission charged against him, he was insane at the time the act was done or omission made so as not to be responsible according to law for his actions, the court may quash the sentence passed at the trial and order the appellant to be kept in custody as a criminal lunatic under the Trial of Lunatics Act, 1883, in the same manner as if a special verdict had been found by the Jury under that Act. The only case that has come up under this subsection is that of James Jefferson, who appeared in July 1908 against a conviction for murder; and the record is sufficiently interesting and important to justify its being given in some detail—

> Jefferson was tried at Leeds before Mr. Justice Bigham, and three medical experts gave evidence as to his insanity. The *Times* (20th July) gives the following account of the concluding portion of the proceedings:—"In answer to the learned Judge, they (the medical men) agreed that he knew he was killing the woman. But on the question whether he knew he was doing wrong their answers differed. Dr. Edgerley said he thought that the prisoner imagined that murdering the woman would be of some advantage to him, by ridding him of a persecutor, and that this delusion would probably be so strong in his mind that all idea of right and wrong would be excluded. Dr. Exley said: 'I think he knew he was doing wrong, but I think he had no idea how wrong.' Mr. Justice Bigham directed the Jury as follows on the question of insanity: 'If the prisoner knew that he was doing wrong, it does not matter that he knew how wrong. If he knew he was doing wrong, it does not matter that he suffered from delusions or hallucinations. A man commonly described as a lunatic may be as guilty of murder as any of you. You have to determine whether he knew he was doing wrong. It is for the prisoner to satisfy you by his evidence beyond all reasonable doubt.' A Juror: 'If there is any doubt, is he not entitled to the benefit

of it?' Mr. Justice Bigham: 'No, it is the other way on, he must satisfy you beyond all reasonable doubt that he did not know he was doing wrong.' The Juror: 'If he knew he was doing wrong, but was insane, how then?' Mr. Justice Bigham: 'If he knew he was doing wrong it does not matter how insane he was, he is guilty.' The Jury, after a retirement of an hour and a half, found the prisoner *Guilty*, and he was sentenced to death." The case came before the Court of Criminal Appeal (consisting of Mr. Justice Lawrance, Mr. Justice Ridley, and Mr. Justice Darling), and the decision arrived at was as follows:—(*Times*, 31st July 1908): "Mr. Justice Lawrance, in giving judgment, said that there was no doubt that the verdict given was unsatisfactory, and in his judgment it ought not to stand. He had read the evidence given by the doctors, and it appeared that there was strong evidence called before the Jury which shewed that this man was not in such a state of mind as to make him responsible for his act. The verdict given being unsatisfactory, he thought they ought to say that the verdict which the Jury should have returned was that the man was insane when he committed the act. He hoped the case was not one of those in which the Jury returned the verdict they found because they knew there was a Court of Appeal behind them to which recourse might be had. The verdict would be set aside, and the order would be that the prisoner should be detained as a criminal lunatic during His Majesty's pleasure."

The verdict wrung from an unwilling Jury was here set aside without reference to the principle of a knowledge of right and wrong as a test of responsibility, and the old-fashioned dictum may now be said to have got its quietus.

DAVID NICOLSON.

ENGLISH LAW AND PRACTICE OF LUNACY

By T. OUTTERSON WOOD, M.D., and G. H. SAVAGE, M.D., F.R.C.P.

THE LAW OF LUNACY.—The lunacy legislation of this country, despite the Acts of 1890 and 1891, remains in an unsettled state; and the care and treatment of the insane is burdened with vexatious and unnecessary restrictions. Not only are the steps required for the placing of a person of unsound mind under legal care complicated and clumsy, but they result in many cases in a delay of that early treatment which is so important in cases of mental disease. The following is a brief abstract of the law which enables a person of unsound mind (not a pauper) to be placed under care and treatment as a private patient, either in an asylum (private or public) or hospital for the insane; or under what is technically designated "Single Care,"—that is, under certificates in the house of a medical man or other person.

Urgency.—In cases of urgency any person (but if possible a relative of the patient) who is twenty-one years of age may sign an order for the admission of an insane person into a lunatic asylum or hospital for mental diseases, or into single care (Form 1). Such order can only be given if the patient has been seen by him (or her) within two days before the date of the order; and it must be accompanied by a certificate of a qualified registered medical practitioner, who, besides stating the facts indicating insanity at the time of examination, must also certify that it is necessary, either for the welfare of the patient or the safety of the public, that the patient shall be removed at once for proper care and treatment (Form 2). He must also give his reasons in writing for arriving at that conclusion. (See "Statement" accompanying Urgency Order, which is part of the medical certificate.) He must further certify also that the patient is in a fit state of bodily health to be removed. The medical practitioner certifying must have seen and examined the patient personally not more than two clear days before his reception, and must state in his certificate the place and date of such examination.[1]

An Urgency Order remains in force for seven days, or, if a petition for a Reception Order is pending, until the petition is disposed of; and, during this time, the necessary formal proceedings must be taken to ensure the legal detention of the patient beyond that period; otherwise the patient will have to be discharged. The further steps necessary for detaining the patient are as follows:—A *petition* for a Reception Order, accompanied by the certificates of two qualified and registered medical men, and a "*statement of particulars*" must be laid before a "Judicial Authority," who is either one of the justices specially appointed by the Quarter Sessions to act as a judicial authority in these cases, or a County Court Judge, or a Stipendiary or Police Magistrate; the order will not, however, be invalid, though signed by a justice who is not specially appointed, if approved and signed by one of such specially appointed justices within fourteen days after its date. If possible the petition must be presented by a husband, wife, or relative of the alleged lunatic. If not so presented, the reason why not must be stated.

The person presenting the petition must have seen the patient within fourteen days, and shall undertake to visit the patient duly, or appoint a deputy to do so. Whenever practicable, one of the medical certificates should be signed by the usual medical attendant of the patient.

If the justice before whom the petition is laid shall be satisfied by the documentary evidence that the person is of unsound mind he may make the *order* forthwith; or, if not satisfied, he may appoint a time, not more than seven days after the presentation of the petition, for the further consideration of the matter; he may, if he think it necessary, visit the patient.[2] If the petition is dismissed the justice shall deliver to the

[1] The forms for cases of urgency only are given here. The necessary forms in ordinary cases can be obtained from the institution for the insane whither the patient is to be removed, or from Messrs. Shaw & Sons, Law Stationers, 7 Fetter Lane, London, E.C. The Urgency forms may be copied in manuscript if time presses.

[2] A justice often fails to understand that the Act does not desire him to visit the patient unless in his opinion there be some special reason for his so doing—such as an inconclusive medical certificate, or some facts in the case which in his opinion call for further inquiry. It

petitioner in writing his reasons for dismissing it, and shall send a copy of this document to the Commissioners in Lunacy.

The *Reception Order* of a justice is sufficient authority to take the patient to, and to receive him in, an asylum, or to detain him there if he has already been removed on an Urgency Order.[1] All these necessary documents must be delivered to the petitioner, who, in turn, must deliver them to the person receiving the patient. A Reception Order has to be kept operative for the detention of a lunatic by reports and certificates of continued insanity, sent by the medical officer to the Commissioners in Lunacy not more than one month, or less than seven days, from the end of the first, second, and third years from its date; and thereafter at intervals of five years.

If a private patient has been received into legal care, either in an asylum or private house, without having been seen by a judicial authority, he shall have the right to see one; and the medical attendant is bound to give him notice of his right, in writing, within twenty-four hours of his reception, unless he (the person receiving the patient) sign a certificate that the exercise of such right would be prejudicial to the patient. The patient is required to exercise his right within seven days, and he must express his desire to do so on Form 7, which will be supplied to him.

Inquisition.—Another method of obtaining legal power to detain a person as of unsound mind is to have him proved so by "Inquisition"; this is generally resorted to when the patient is wealthy, his insanity is clearly marked, and he is not likely to be cured; or when owing to his infirmity business concerns are at a standstill. The steps to procure an inquisition (*de lunatico inquirendo*) are for a relative or some responsible person to instruct a solicitor, who, after obtaining affidavits from two medical men testifying to the insanity of the patient, makes application to the Judge in Lunacy for an inquiry. The Commissioners in Lunacy can also report to the Lord Chancellor that the property of a lunatic is not duly protected, or his income not properly applied, and such report has the effect of an application for an inquisition. The inquiry is held before a Master in Lunacy, who acts as judge; the patient may be defended by counsel and be tried by a jury if he wishes; and he may be present at the inquiry. A person found insane by inquisition becomes a "Chancery patient," and two persons are appointed, one as committee of his estate, and the other as committee of his person. The committee of his person can, with the consent of the Lord Chancellor's visitors, remove the patient

happens not infrequently that from a mistaken sense of duty, or prompted by his clerk, the justice makes a visit as a matter of ordinary duty. In this case the justice, being unskilled in diagnosis, may fail to recognise the insanity of a properly certified patient, and thus may bar himself from signing an order. In this way dangerous lunatics have often been set at large, to their own grievous harm or to the harm of others. The words of the Section are that the judicial authority "shall consider" (on the documents) "whether it is necessary for him personally to see and examine the alleged lunatic. . . . If not so satisfied" ("that an order may properly be made forthwith") he may appoint a time for consideration and further inquiry. Finally, "if not satisfied with the evidence . . . of the medical certificates, he may, if he thinks it necessary to do so, visit the alleged lunatic," etc.

[1] It must be acted on within seven clear days from its date.

from place to place. The committee of his estate manages his affairs under the supervision of the Judge in Lunacy.[1]

Lunatic not under Proper Care or Control, or Ill-treated.—Again, a person of unsound mind who is not a pauper, and not wandering at large, but who is not under proper care and control, or is cruelly treated and neglected, can be ordered by a specially appointed justice to be visited and examined by two medical men, and the justice, if satisfied that the patient is a lunatic, and is not under proper care and control, or is neglected or cruelly treated, may order him to be sent to an asylum. The law provides that every constable, relieving officer, or parish overseer shall give information to such justice upon oath, within three days of obtaining the knowledge, that such alleged lunatic is within his district or parish, and is not under proper care and control, or is cruelly treated.

Medical Certificates. — It should be borne in mind that medical certificates of insanity must be based upon facts observed at the time of the examination, and when filling up the certificate these facts must be clearly stated. The certificate must not be based only upon facts communicated by others; and the medical man must be extremely careful not to act hurriedly, or without due care. It is always wise to spend a considerable time in ascertaining, as nearly as may be, not only the exact mental condition of the patient, but the physical condition also. By so doing the medical practitioner will be protected by law, and no charge of acting without good faith and reasonable care can successfully be brought against him.

Single Patients.—When any medical man or other person undertakes the care and treatment of a patient in his own house the case must be admitted under the same formalities, as regards Reception Order and medical certificates, as if he were being admitted into an asylum.[2] As a rule, only one such certified case can be received, but the Commissioners in Lunacy have the power, in special circumstances, to permit more than one, and their sanction must be obtained.

The law bearing upon the admission of cases of incipient or uncertifiable insanity into single care is now before the Legislature, and it is proposed that a doubtful or uncertifiable case may be taken in for a definite period upon the application of a relative or friend, accompanied by one medical certificate (see also following paragraph).

Commissioners' Rules.—In addition to the Act of Parliament, the Commissioners in Lunacy are empowered to make Rules for the guidance of those having charge of the insane, and these Rules are legally as binding as the Act itself. Therefore any medical practitioner who takes care of a certificated insane person must apply to the Commissioners in Lunacy for a copy of their Rules, so that he may inform himself of their requirements. These Rules specify the notices to be given to the Commissioners and others, the books and records to be kept, and the regulations to be observed by those having charge of lunatics. It is illegal for any person, whether a medical man or not, to take charge of an insane person for payment, except under a proper Reception Order.

---

[1] The Commissioners in Lunacy, moreover, have now considerable powers of inquiry into the property of insane persons, Sect. 50, subs. (2) 53, Vict. ch. 5.

[2] The necessary forms can be procured from Messrs. Shaw, Law Stationers, 7 Fetter Lane, London, E.C.

Mechanical restraint must not be employed without conforming to the requirements of the Lunacy Acts, and the Commissioners in Lunacy have the power to determine, by regulation, what mechanical means of restraint may be used; a record of the use of any such restraint, its kind and duration, must be carefully kept from day to day in every case in which it is employed, and a copy thereof must be forwarded to the Commissioners quarterly.

The power of discharging a private patient (not being a Chancery patient) is given in the first instance to the petitioner for the Reception Order; failing him to relatives, or the person who pays for the patient; but powers of discharge are also given to the Commissioners, and to Justices who are Visitors of Provincial Licensed Houses.

Medical Attendant.—A medical practitioner who certifies to the insanity of one of his patients is not allowed to act as his medical attendant while he is under certificates. This is a curious provision of the law, for care is taken in the Act that the usual medical attendant, whenever practicable, shall sign one of the certificates, though by so doing he is precluded from attending him while he is detained under his certificate. The patient and his family may be anxious to retain the services of their own doctor; but, if he sign one of the certificates, he is legally disabled from attending him.

53 Vict. cap. 5, Schedule 2.

FORMS 4, 2, 8, and 9.

Form of Urgency Order for the Reception of a Private Patient with Medical Certificate and Statement accompanying Urgency Order.

I, the undersigned, being a person twenty-one years of age, hereby authorise you to receive as a Patient into your (*a*)..........
(*b*) ....
as a (*c*) .... whom I last saw at ....
on the (*d*) .... day of .... 1 ....

I am not related to or connected with the person signing the Certificate which accompanies this Order in any of the ways mentioned in the margin (*e*). Subjoined (or annexed) hereto is a Statement of Particulars relating to the said ....
....

(Signed)
Name and Christian name at length } ....
Rank, Profession, or Occupation (if any) } ....
Full postal address ....
How related to or connected with the Patient } ....

Dated this .... day of .... 1 ....
To (*f*) ....
....

(*a*) House, or hospital, or asylum, or as a single patient.

(*b*) Name of patient.

(*c*) Lunatic, or an idiot, or a person of unsound mind.

(*d*) Some day within two days before the date of the Order.

(*e*) Husband, wife, father, father-in-law, mother, mother-in-law, son, son-in-law, daughter, daughter-in-law, brother, brother-in-law, sister, sister-in-law, partner, or assistant.

(If not the husband or wife, or a relative of the patient, the person signing to state as briefly as possible—(1) Why the Order is not signed by the husband or wife, or a relative of the patient. (2) His or her connexion with the patient, and the circumstances under which he or she signs.)

(*f*) Superintendent of the —— asylum, —— hospital, or resident licensee of the —— house (describing the asylum, hospital, or house by situation and name).

Lunacy 4 and 2
(53 Vict. cap. 5, s. 11).

STATEMENT OF PARTICULARS REFERRED TO IN THE ANNEXED ORDER.

If any Particulars are not known, the Fact is to be so stated.

(Where the patient is in the petition or order described as an idiot, omit the particulars marked *)

The following is a Statement of Particulars relating to the said . . . .
Name of patient, with Christian Name at length . . . . . Sex and age . . . . .
*Married, single, or widowed . . . . . *Rank, profession, or previous occupation

(if any) . . . . . *Religious persuasion . . . . . Residence at or immediately previous to the date hereof . . . . . *Whether first attack . . . . . Age on first attack . . . . . When and where previously under care and treatment as a lunatic, idiot, or person of unsound mind . . . . . *Duration of existing attack . . . . . Supposed cause . . . . . Whether subject to epilepsy . . . . . Whether suicidal . . . . . Whether dangerous to others, and in what way . . . . . Whether any near relative has been afflicted with insanity . . . . . Names, Christian Names, and full postal addresses of one or more relatives of the patient . . . . . Name of the person to whom notice of death is to be sent, and full postal address, if not already given . . . . . Name and full postal address of the usual medical attendant of the patient . . . . .

When the petitioner or person signing an urgency order is not the person who signs the statement, add the following particulars concerning the person who signs the statement :—

Signed : Name, with Christian Name at length . . . . . Rank, Profession, or Occupation (if any) . . . . . How related to or otherwise connected with the patient . . . . .

---

53 Vict. cap. 5.—Form 9.

(*i*) If any urgency certificate is required, it must be added here.

(*i*) *Statement accompanying Urgency Order.*

I certify that it is expedient for the welfare of the said .... (or for the public safety, as the case may be) that the said ....
should be forthwith placed under care and treatment.

My reasons for this conclusion are as follows : ....
....

(*k*) Strike out this clause in case of a private patient whose removal is not proposed.

4. The said...appeared to me to be (or not to be) in a fit condition of bodily health to be removed to an asylum, hospital, or licensed house (*k*).

5. I give this certificate, having first read the section of the Act of Parliament printed below.

Dated this ....day of ....
One thousand ....hundred ....
(Signed) ....
of (*l*) ....

(*l*) Insert full postal address.

Extract from Section 317 *of the Lunacy Act,* 1890.

Any person who makes a wilful misstatement of any material fact in any medical or other certificate, or in any statement or report of bodily or mental condition under this Act, shall be guilty of a misdemeanour.

T. Outterson Wood.

Testamentary Capacity.—The general idea of legal capacity is that the testator must, at the time of making his will, have had a knowledge of his property and of his kindred, memory sufficient to know his proper relations to those about him, freedom from delusions affecting his property and his friends, and sufficient physical and mental power to resist undue influence. A person may be unable to make a just will, because he believes (falsely) that he is without means, or that his relatives have turned against him. In melancholic states, therefore, a man may be incapable of making a just will; on the other hand, though suffering from profound melancholia, he may make a will which is consistent with intentions expressed during the healthy period of his life. Defect of memory is a very serious bar to will-making, but if the person recognise his loss of memory and act in a way consistently with his past expressed intentions, the will may be upheld: or, again, a man with very defective recent memory may have an accurate judgment on business matters placed before him; thus, even though he forget as soon as he has given his judgment, yet if this judgment is consistent with his previous intentions, it might be taken as a legal expression of such intentions. In one case a patient with great loss of recent memory, when asked as to his wishes concerning certain property, always gave the same answer, though he forgot he had already done the same half-a-dozen times. If the testator does not recollect the number of his children, or whether they are living or dead, he may easily make a bad will; or there may be some loss of memory leaving in him a false impression of the conduct of some person: for example, an eldest son for some years was a cause of worry and anxiety to his father, and the father intended to disinherit him; later, however, he reformed and was forgiven; but with old age the father forgot the last stage, and acted on the assumption that his son was still a waster. In some rare cases following shock or injury, a partial defect of memory may interfere with due capacity; thus, it has occasionally happened that a man after an acute illness or concussion of the brain has forgotten that he was married, and would not acknowledge his wife.

Delusions are looked upon as very strong points against testamentary capacity; but, unless the delusions have some direct bearing on the property, or on the relations of the testator to his friends or to society, a jury will not pay much attention to them. That a man believes the moon to be of green cheese, or that he is himself a bottle of pickles, will not necessarily affect his judgment as to his property; although even here it is not always safe to say that a notion which in a sane person would not affect conduct, might not change the conduct of an insane person.

Delusions as to property, and as to the conduct of friends and kindred, are among the strongest points against the testamentary capacity of an individual.

Besides loss of memory and the presence of delusions we have to consider various forms of mental weakness. Idiocy and a marked degree of congenital or acquired imbecility will be held to bar testamentary capacity ; but there are many young men and women, morally defective, who if they come into property may rapidly dissipate it ; yet they are not certifiably insane, or, in the eye of the law, incapable of making a will. In prospect there is legislation for the protection of the feeble-minded. The plea of undue influence, however, may be raised in such cases ; particularly if some such youth has been dominated and directed by a woman.

Mental weakness resulting from previous attacks of insanity, recurring epileptic fits or apoplexy, may incapacitate the patient ; but the jury will be more influenced in such cases by the nature of the will than by any medical evidence as to the testator's capacity.

In my experience litigation is most common in respect of the wills of persons who have been intemperate, and of those who have had apoplectic seizures. In the former cases the evidence, however true, may be very conflicting ; as in the presence of some witnesses the testator may have been sober, and in the presence of others he may have been drunk. In drunkards, too, degeneration may be very rapid ; so that a fairly reasonable man may fall into mental confusion in a few days, and wholly lose his memory. That a man has been long subject to epilepsy may be a ground for doubting full mental capacity ; but this will not avail much if the will itself is fairly reasonable.

After apoplectic seizures more or less mental defect is generally seen. The most common and conspicuous affliction is aphasia. Lawyers and some juries have got a vague general idea that a person may have speech-defect without any real intellectual loss, and it will be useless to refer to aphasia as a sign of mental defect if a fairly reasonable will is propounded. There is, in my opinion, always some mental failure with the aphasia, but this varies very greatly, and in examining such cases with a view of testing their capacity great care and patience are required. On the other hand, lawyers may endeavour to establish capacity when a testator afflicted with aphasia can express himself as satisfied with what is read to him ; yet in many cases consent is only automatic ; a mere "Yes" to a question does not necessarily import understanding. Aphasia, then, may be present with or without testamentary capacity ; yet, as in many cases of aphasia the position of medical men called in to advise on testamentary capacity may be a very difficult one, I will set down here also the opinions of three other authors who have given special attention to the subject.

Dr. William Elder (*Brit. Med. Journal*, 1898, ii. 583) concludes : (i.) That organic disease of the brain may render a patient incapable of making a will, and that some form of aphasia may be produced also as one of the symptoms of the organic disease. (ii.) That some forms of aphasia may render a patient incapable of will-making. (iii.) That auditory aphasia, if at all well marked, incapacitates a patient from will-making. (iv.) That some other forms of aphasia, such as pictorial word-blindness, pictorial motor aphasia, and agraphia, may render a patient incapable of making a will, not necessarily from being mentally incapable, but from the difficulty of carrying out the legal formalities. (v.) That these difficulties in carrying out the legal formalities necessarily vary according to the law of the particular country. (vi.) That patients in simple uncomplicated cases of infrapictorial auditory, infrapictorial visual, and infrapictorial motor aphasias are capable of valid will-making.

Dr. Clouston (*Brit. Med. Journ.*, 1898, ii. 585) gives the following advice: (i.) Test whether the document is his own and has not been suggested by others; (ii.) put the contrary proposition, or a different proposition twice at least as to sums disposed of, and as to the persons to whom the money or property is left: no will can be valid when these two points have not been carefully gone into.

Sir W. Bateman (*Aphasia*, 2nd ed., 1890) wrote: "Aphasia does not necessarily entail testamentary incompetency; in fact, wills, or other legal documents, should as a rule be recognised as valid when the parties understand fully what is put before them, and can express assent or dissent with certainty, whether by articulate, written, or gesture language."

Much has been made of lucid intervals, and therefore we must not lose sight of them. Many patients in asylums suffering from recurring attacks of mania or melancholia, and some suffering from *folie circulaire* or epileptic insanity, may be quite sane for long periods, or sane enough to make a will; though they might not be considered capable of committing a crime, they might be capable of disposing of property. Even in some chronic and incurable patients an acute illness may cause temporary sanity. In general paralysis of the insane there may be complete remissions, and the wills of some general paralytics have been established on the ground of such remissions. I think this has been carried too far, for, though remissions may occur, we do not expect them in the last stage of the disease.

Certain wills made by patients in asylums and even patients who were lunacy Wards in Chancery have been accepted by the Courts.

The present bent of an English jury is to find for any will, not in itself unreasonable, if it express the wishes of the testator approximately. It has been established that a patient in private care, under lunacy certificates, may make a valid will; that a patient in an asylum may likewise make a good will; and that even a "Chancery patient" may be competent to dispose of his property.

G. H. SAVAGE.

INDEX

Abscess of the brain, 250-265 ; age and, 253 ; cerebellar, 136, 264 ; cerebral tumour and, 260 ; convulsions in, 259 ; diagnosis, 256-265 ; ear disease and, 250-254, 256-257, 259, 263, 265 ; etiology, 250-253 ; fever in, 255 ; leucocytosis in, 258, 260 ; meningitis and, 258 ; morbid anatomy, 250-253 ; nose disease and, 253, 265 ; prognosis, 262 ; pulse in, 255 ; pyaemic, 251, 254, 262 ; sex and, 253 ; sinus phlebitis and, 259 ; symptoms, 253-256 ; temporo-sphenoidal, 263 ; treatment, 262-265

Abscess, cerebellar, 136, 264 ; cerebral, 250-265 (*see above*) ; frontal, 45, 64 ; medullary, 156 ; occipital, 86 ; of the crura cerebri, 117 ; periarticular, in meningococcic meningitis, 179 ; pontine, 126 ; temporal, 94-95 ; temporo-sphenoidal, 263

Absinthe, and the cerebral circulation, 14 ; and epilepsy, 508

Accoucheur's hand in tetany, 210, 589

Acne, due to bromides, 515

Acquisitiveness and criminal lunacy, 1019

Acromegaly and the hypophysis, 162

Acrophobia in tic, 625, 628

Actinomycosis, meningeal, 166

Adenoids, and night-terrors, 809, 816, 817 ; and stammering, 572

Adhesions in hemiplegia, 302

Adolescence and puberty, insanity in, 927-930

Adrenalin and the cerebral circulation, 11-12

Aerophagy in neurasthenia, 760

Agglutination-test for the meningococcus, 172

Agnosia, 447-452 ; apraxic, 80, 83, 447-452 ; auditory, 439, 450 ; ideational, 451 ; gustatory, 440, 450 ; object, 450 ; olfactory, 440, 450 ; tactual, 439 ; verbal, 386, 397, 429, 450 ; visual, 83, 89, 439-440

Agoraphobia, 864, 970 ; hypnotism in, 1011 ; in neurasthenia, 749 ; in tic, 625, 628

Agraphia, 405, 409, 413, 416-418, 451 ; complete, 417 ; in cerebral abscess, 264 ; partial, 417 ; prognosis, 417 ; testamentary capacity in, 1048

Akinesia algera, 717

Albuminuria, and insanity, 908 ; in medullary lesions, 159 ; in pontine lesions, 134

Alcoholic excess, and criminal lunacy, 1036 ; and idiocy, 875 ; and insanity, 833, 909, 912-920 ; and vice, crime, and insanity, 856-857 ; diagnosis from cerebral haemorrhage, 330 ; in epilepsy, 508 ; in general paralysis, 350, 377

Alexia (word-blindness), 89, 386, 395

Alphabet, the, and stammering, 570

Alternation in insanity, 976

Altruism in epileptics, 956

Amaurosis (*see also* Idiocy), amaurotic family, 468-473 ; in infantile diplegia, 459

Amblyopia, and amaurotic family idiocy, 468-471 ; and hysteria, 707 ; and lesions of the angular gyrus, 81 ; and lesions of the corpora quadrigemina, 121 ; and lesions of the optic thalamus, 114 ; in cerebral haemorrhage, 325 ; in intracranial tumour, 275 ; in polioencephalitis, 246

Amblyopia, crossed, in cortical lesions, 84 ; hysterical, 84, 707

Amenorrhoea and intracranial tumour, 280

Ametropia, and chorea, 526 ; and intracranial tumour, 279 ; and neurasthenia, 743, 745, 750, 769

Amnesia, alcoholic, 917 ; hypnotic, 1012 ; hysterical, 691, 715

Amyl nitrite, and the cerebral circulation, 22 ; in epilepsy, 504

Anaemia and intracranial tumour, 279

Anaemia, cerebral, 23-28 ; and neurasthenia, 769

Anaesthesia (*see also* Sensation, Impairment of), and the falciform lobe, 76 ; cortical, 67-71 ; hysterical, 67, 74, 697-699

Anaesthetics and insanity, 907

Anarthria, 397, 399 ; spastic, 400

Aneurysms, miliary, and cerebral haemorrhage, 314

Anger and melancholia, 970
Anidrosis in thrombosis of the posterior cerebellar artery, 160
Ankle-clonus, in hysteria, 701, 704; in lesions of the optic thalamus, 113; in neurasthenia, 764, 771
Anophthalmos, cortical atrophy in, 88
Anorexia nervosa, 710, 722-723, 747, 758
Anosmia in frontal tumour, 45, 49, 50
Anthropology, criminal, so-called, 1020
Anuria, hysterical, 713
Apathy, mental, in cerebral syphilis, 337
Aphasia, 385-446 (Figs. 20-24): agnosia, verbal, 386, 397, 429; agraphia and, 405, 409, 413, 416-418; alexia and, 386, 395; amnesia and, 90-97, 386, 410, 426, 428; anarthria and, 397, 399; and testamentary capacity, 1048; aphemia and, 405-409; aphthongia and, 400; apraxia and, 396, 407, 447-450; auditory and visual word-centres and, 424-443; auto-intoxication and, 419; Broca's, 64, 385, 395, 397, 402, 408, 410-424; cerebral lesions and, 394; commissural, 444; commissural defects and, 443-445; concepts and, 391-394; convulsions and, 319; diaschisis and, 395, 411; dysarthria and, 399-405; embolism and, 418; hypnosis and, 419; hysteria and, 420-422; idioglossia and, 400; infrapictorial, 1049; in cerebral abscess, 258; in cerebral haemorrhage, 298, 324, 329; in cerebral syphilis, 338; in hemiplegia, 62-75, 93, 324; in meningitis, 211, 216; in recurrent paralysis, 605; in tumour of the brain, 277; incitation of the speech-centres and, 391; insula and, 97; intermittent claudication and, 418; lalling and, 400; Marie's views on, 396-398; motor, 64, 385, 424, 448; mutism in, 408, 420-422; paraphasia and, 427; percepts and, 391-394; poisoning and, 418; pseudo-mutism and, 419; pure or subcortical, 388, 405-409, 431; right cerebral hemisphere and, 390; sensory, 83, 385, 397, 425; speech-apraxia and, 386; speech-defects and, 398-414; speech-memories and, 396-398; stammering and, 400; subcortical, 398, 405-409, 431; symptoms, 414-424; temporary, 418-424; transcortical, 388; visual, 1049; Wernicke's, 90-97, 385; word-centre defects and, 424-443; word-images and, 391; word-memory and, 387 (Fig. 20), 396; writing centre and, 391, 412, 416-418; Wyllie's views on, 422-424, 442
Aphasia of the limbs, 50
Aphemia, 405-409; distinction from spastic anarthria, 406
Aphonia, hysterical, 424, 708
Aphthongia and aphasia, 400
Apoplexy, 320 (*see also* Cerebral Haemorrhage, 307-334); general paralysis and, 379; ingravescent, 321
Apoplexy, symptoms due to cerebral anaemia, 33, 320-321
Apraxia, agnostic, 449 (*see* Agnosia, 450-452); in angular lesions, 83; in parietal lesions, 80
Apraxia, ideational, 452
Apraxia, motor, 326, 447-452; agnosia and, 447; corpus callosum and, 100; diagnosis, 450; in frontal tumour, 50; motor aphasia and, 64, 385, 424, 448; pathology, 448; symptoms, 449
Apraxia, sensory, 449 (*see* Agnosia, 450-452); speech-, 386
Aqueous-humour, secretion of the, 12
Areas of the brain (*see also* Gyrus *and* Lobe), arm, 53; association, 40, 950; Broca's, 74, 97; calcarine, 80-81, 86; face, 53; frontal, 40-50 (Figs. 5 and 6); kinaesthetic, 71, 73; larynx, 53; leg, 52; motor and kinaesthetic, 71; mouth, 53; pericalcarine, 86; peristriate, 86; post-frontal, 40; posterior association of Flechsig, 79; pre- and post-central, 51-76, 950; prefrontal, 40; Rolandic, 51-76 (Figs. 7-10); striate of Elliot Smith, 86; tongue, 53; visual, 88; Wernicke's, 97
Arithmomania in tic, 628
Arsenic, and insanity, 912; in cerebral syphilis, 345; in chorea, 549; in epilepsy, 515, 516; in tic, 634
Arteries, cerebral, the, 11-20 (Figs. 1 and 2); 311-312
Arteriosclerosis, and cerebral haemorrhage, 308-310, 313; vasomotor nerves and, 11, 13
Arthrogryposis and tetany, 578
Articulation, mechanism of normal, 569
Articulation, impairment of, in extra-cerebellar tumour, 148; in pseudo-bulbar paralysis, 106-108; in Rolandic lesions, 64
Asphyxia and the cerebral circulation, 10-12; and night-terrors, 809
Astasia in cerebellar lesions, 137-138, 146
Astereognosis, 447, 450; and cerebellar disease, 149; and cortical lesions, 67, 70-74, 80; and lesions of the optic thalamus, 114; and parietal tumour, 275
Asthenia and lesions of the cerebellum, 138, 144
Asymboly, 447-450
Ataxia, cerebellar, 140, 399; cerebral, in chorea, 399; cortical, 450; in cerebral tumour, 275; in general paralysis, 373-375; in lesions of the corpora quadrigemina, 120; in lesions of the crura cerebri, 118; in lesions of the optic thalamus, 112-113; in polioencephalitis, 247
Ataxia, hereditary cerebellar of Marie, 142
Ataxia, social, in general paralysis, 358, 359
Atheroma in cerebral haemorrhage, 293, 313, 315

Athetosis, and lesions of the optic thalamus, 112-113 ; double, 456, 460 ; in cerebral thrombosis, 299 ; in idiocy, 883 ; tic or, 633
Atonia and lesions of the cerebellum, 138, 144
Atrophy, olivo-ponto-cerebellar, 140-141, 143
Atrophy, optic, in disease of the hypophysis, 162 ; in frontal tumour, 46
Atropine in tic, 635
Auditives and visuals, 389, 426, 433, 434
Aura, the epileptic, 494-500, 947-952 ; auditory, 94, 951 ; cephalic, 498, 951 ; epigastric, 498, 951 ; gustatory, 498, 951 ; in Jacksonian epilepsy, 274 ; intellectual, 277 ; olfactory, 96, 498 ; psychical, 499, 951 ; somnolence, 500, 951 ; trunk, 497 ; visceral, 497, 951 ; visual, 499, 951
Aura, the hysterical, 694
Auto-intoxication and aphasia, 419 ; and insanity, 904-912, 994
Automatism, after epilepsy, 503, 953-954 ; comitial ambulatory, 954 ; in frontal lesions, 49 ; professional, 954
Aversions in vice, crime, and insanity, 864

Babinski's sign and hysteria, 703, 717
Bacillus anthracis in meningitis, 166, 189, *Diplococcus intracellularis* in meningitis, 170-72 ; *Diplococcus rheumaticus* in chorea, 523, 526, 528, 529 ; in meningitis, 166, 188 ; *dysenteriae* in meningitis, 166 ; *enteritidis* in meningitis, 166, 193 ; *influenzae* in meningitis, 166, 187 ; in polioencephalitis, 243 ; leptothrix in meningitis, 166 ; *Micrococcus catarrhalis* in meningococcic meningitis, 168, 171 ; of acute delirium, 900 ; of general paralysis, 357 ; of polioencephalitis, 242-244 ; pneumococcus in meningitis, 165 ; staphylococcus in meningitis, 166, 185 ; streptococcus in meningitis, 166, 185 ; streptothrix in meningitis, 166, 189-191 ; *typhosus* in meningitis, 166, 191-193 ; Weichselbaum's diplococcus, 170-172
Baillarger, the lines of, 948
Barbers' cramp, 680
Bechterew's fluid in epilepsy, 959
Belittlement in general paralysis, 367
Belladonna, and insanity, 910 ; in epilepsy, 516
Bergeron's disease, 631
Bernhardt's disease, 679
Betz-cells, the, in the precentral cortex, 55
Biborate of sodium in epilepsy, 516
" Biliousness " and concussion of the brain, 232, 233 ; and general paralysis, 371
Birth-injuries and idiocy, 876
Birth-palsy, 453-459
Bladder, the, and lesions of the crura cerebri, 118 ; in cerebral haemorrhage, 332
Bleeding in concussion of the brain, 238
Blepharoptosis, 82-83
Blepharospasm, 641 ; and tic, 617 ; hypnotism for, 1010 ; hysterical, 706
Blindness, *see* Amblyopia
Blindness, cortical, 451 ; letter-, 434 ; mind- and object-, 427, 439 ; musical-, 440 ; word-, *see* Word-blindness
Blood-diseases, and cerebral haemorrhage, 309 ; and cerebral thrombosis, 293
Blood-pressure, the, in cerebral haemorrhage, 308, 321, 332 ; means of lowering, 331-332
Brachycephaly in idiocy, 878
Brain, softening of the, 261, 292, 294 ; in idiocy, 885 ; in senile insanity, 938
Brain, the (*see also* Abscess, Cerebral, and Cerebellum), circulation and, 3-37 ; in general paralysis, 352, 354 ; in idiocy, 884-887 ; intoxication of the, 443 ; vasomotor nerves of, 11
Brain-disease and insanity, 837-838
Brain fever, 902
Breast, the hysterical, 711
Bricklayers' cramp, 680
Bristowe's syndrome, 99-100
Broca's aphasia, 385, 395, 397, 402, 408, 410-424
Bromides, the, acne due to, 515 ; in acute delirium, 902 ; in epilepsy, 512-516 ; in head-nodding, 666 ; in insomnia, 797-798 ; in mania, 894, 898 ; in night-terrors, 818
Bromural in insomnia, 799
Bruns' syndrome, 159
Butyl-chloral in insomnia, 798

Calcium-excretion, in tetany, 579, 587-588, 595, 601 ; in tuberculous meningitis, 210
Cancer and insanity, 840, 841
Cannabis indica in insomnia, 800
Carbon di- and mon-oxide and insanity, 907
Carpopedal contractions and tetany, 578-579, 583
Casque neurasthénique, 745
Catalepsy, 987-988
Catatonia, 930, 987-988
Centre, Nerve-, auditory, 123 ; auditory word- 387, left 428-432 ; Broca's, 386, 390-397, 402, 408, 410-424 ; commissural connexions of, 444-445 ; cheiro-kinaesthetic, 387-394, 409-424 ; conceptual, 392-393, 428-429 ; eupraxic, 387, 398, 402, 413, 422-423, 451 ; glosso-kinaesthetic, 387-394, 409-424, 443 ; ideational, 392-394, 429 ; Marie's speech-, 396-398 ; naming, 95 ; perceptive, 393 ; speech-, 386-394, 398 ; visual word- 387, left 432-439 (Figs. 23 and 24) ; Wernicke's, 398 ; word-, *see* auditory- and visual-word ; writing, 391, 412
Cerebellar ataxia, 140, 399 ; hereditary (of Marie), 142
Cerebellum, functions of the, 137-140

Cerebellum, lesions of the, 134-152; abscess, 136, 264; astasia in, 137, 146; atonia in, 138, 144; atrophy, 136, 140-143; attitude in, 143; compensation in, 139; dysdiadochokinesia in, 145; forced movements in, 146-147; gait in, 137-140, 143; gumma, 339; haemorrhage, 134-136, 327; hereditary cerebellar ataxy, 142-143; in general paralysis, 352; in idiocy, 886; in polioencephalitis, 244; nystagmus in, 148; ocular symptoms in, 147; olivo-ponto-cerebellar atrophy, 140-141, 143; pain in, 150-151; polioencephalitis, 247; primary parenchymatous degeneration, 140-141; proprioceptive system and, 139; softening, 136; syphilis of, 339; symptoms, 137-140; tremor in, 146; trophic changes in, 149

Cerebellum, tumour of the, 123, 136, 143-151, 275; deafness in, 276

Cerebral abscess, *see* Abscess of the brain, 250-265

Cerebral circulation, the, *see* Circulation, 3-37

Cerebral disease, the regional diagnosis of, 37-162 (Figs. 3-10), *see also* Areas; angular gyrus, 79-85; centrum ovale and internal capsule, 100-104; cerebellar peduncles, 152-155; cerebellum, 134-152; corpus callosum, 97-100; corpora quadrigemina, 119-125; corpora striata, 104-109; crura cerebri, 116-119; cysticercus in the fourth ventricle, 159-160; destructive lesions, 38-40; falciform lobe, 76-79; frontal lobe, 40-51 (Figs. 5 and 6); hippocampal lobe, 95; hypophysis cerebri, 162; indirect or inhibitory lesions, 40; insula, 96-97; internal capsule and centrum ovale, 100-104; introductory to, 37-40 (Figs. 3 and 4); irritative lesions, 38-40; limbic lobe, 76-79; medulla oblongata, 155-62; occipital lobe, 81, 85-90; optic thalamus, 109, 116; parietal lobe, 79-85; pons Varolii, 125-134, 326, 639; pyriform lobe, 95; quadrate lobe, 78; Rolandic area, 51-76 (Figs. 7-10); supramarginal lobule, 79-85; temporal lobe, 90-97; thrombosis of the posterior cerebellar artery, 160-161; uncus, 96

Cerebral haemorrhage, 307-334; age and, 309; aphasia in, 324, 329; apoplexy and, 320; arterial aneurysms and, 314; associated lesions, 315-316; atheroma and, 313-315; bleeding in, 332; blood-pressure in, 308, 321, 332; causation, 308; cerebral circulation and, 311-313; coma in, 321, 328; conjugate deviation in, 322; convulsions in, 321, 328; diagnosis, 329; duration, 323, 324; gout and, 308; heredity in, 308; hyperpyrexia in, 322; infective endocarditis and, 310, 315; ingravescent, 321; lead and, 308, 309; localising signs and symptoms, 323-329; mechanical effects of, 316-318; meningeal, 319, 327; morbid anatomy, 311-320; onset, 320; orbicularis symptom in, 324; prodromes, 320; prognosis, 328; pulse in, 321; pupils in, 322; reflexes in, 322, 325; rigidity in, 322, 328; secondary degenerations in, 319; sensation in, 324; sex and, 309; site of, 318; sleep and, 293, 310; sphincters in, 322; subdural, 319; symptoms, 320-328; temperature in, 322, 328; treatment, 331; ventricular, 318, 327; weather and, 310

Cerebral paralyses of children, the, *see* Paralyses, Cerebral, 452-467

Cerebral thrombosis, 329

Cerebral vessels, occlusion of the, 290-307; aphasia in, 298; atheroma and, 293; athetosis in, 299; chlorosis and, 293, 305; convulsions in, 302; decubitus acutus in, 300; diphtheria and, 294; embolism, 290-293, 330; headache in, 295, 306; hemianopia in, 297; infarction of lung in, 306; morbid anatomy, 292, 304; pain in, 300; paralysis in, 296; red softening in, 292, 294; reflexes in, 298; rigidity in, 298; symptoms, 293, 304-306; syphilitic 295; thrombosis, arterial, 293-296; thrombosis, venous, 303-307, 329; treatment, 301, 304; yellow softening in, 294

Cerebritis in cerebral abscess, 256

Cerebrospinal fluid, the (*see also* Lumbar puncture), circulation of, 5-9, 33; in atrophy of the brain, 9; in general paralysis, 356; in meningococcic meningitis, 179, 181; pressure of, 14; subarachnoidal, composition of, 8

Cerebrum, the, *see* Brain

Certification, in criminal lunacy, 1037; in lunacy, 1043-1046

Change, benefits of, in melancholia, 980; in paranoia, 1001

Charcot's artery of cerebral haemorrhage, 311

Charcot's blue oedema in hysteria, 712

Cheyne-Stokes respiration, and the cerebral circulation, 25; in cerebral haemorrhage, 321; in general paralysis, 374; in meningococcic meningitis, 178; in tuberculous meningitis, 214; in tumour of the brain, 278

Chloral, and insanity, 911; in chorea, 547; in insomnia, 798

Chloral habit, the, and hypnotism, 1009

Chloralamide in insomnia, 798

Chloroform and cerebral circulation, 18

Chorea, 519-548; age and, 521; ametropia in, 526; arsenic in, 545; chloral in, 547; chorea-corpuscles in, 528; debility and, 524; diagnosis, 540-542, 644; *Diplococcus rheumaticus* in, 523, 526, 528, 529; electrical reactions in, 533; emotions and, 525, 526; etiology, 520-527; eruptions in, 538; heart in, 536-538, 543; heredity and, 520; hypnotism for, 1010; hysteria and, 526, 706; imitation and, 526 ; intoxications and, 524 ; mental condition in, 535, 541, 542, 839 ; morbid anatomy, 527 ; movements in, 530-533 ; ocular phenomena in, 536 ; paralytic, 540, 543 ; pathology, 528 ; pericarditis and, 538 ; pregnancy and, 525, 542, 544 ; prognosis, 542 ; quinine in, 546 ; recurrence in, 539 ; reflex, 527 ; reflexes in, 534 ; rest in, 544 ; rheumatism and, 521-524 ; season and, 521 ; sensation in, 533 ; sex and, 521 ; specific fevers and, 524 ; symptoms, 530-540 ; tic or, 617, 631 ; treatment, 543-548 ; trophic changes in, 535 ; urine in, 538 ; zinc salts in, 546

Chorea Anglorum, *see* Chorea, 519-548

Chorea, chronic progressive *or* chronic adult, 548-552 ; causation, 549 ; morbid anatomy, 549; pathology, 550; symptoms, 551

Chorea, congenital, 553 ; electric, 542, 554-555, 621, 656 ; fibrillar, 651 ; gravidarum, 525, 542, 544 ; habit, 615, 619 ; hemiplegic, 112 ; hereditary *or* Huntington's, 542, 549-552, 656 ; major, 520, 632 ; malleatory, 632 ; minor, *see* Chorea, 518-548 ; mollis *or* paralytic, 540, 543 ; post-hemiplegic, 112-113, 632 ; saltatory, 632 ; senile, 541, 548, 552 ; spastic, 456, 464 ; Sydenham's *or* vulgar, *see* Chorea, 519-548 ; symptomatic, 529

Chorea-corpuscles, 528

Choroid, tubercles in the, 217

Choroid plexuses, the, in idiocy, 886 ; in meningococcic meningitis, 169-170 ; secretion of cerebrospinal fluid by, 7, 12

Chvostek's sign in tetany, 592

Cingulum, the, 79

Circulation, cerebral, experimental pathology of the, 3-37 (Figs. 1 and 2) ; anacrotism and, 3 ; blood-pressure and, 20-23 ; cerebral anaemia and, 23-28 ; symptoms, 30 ; cerebral hyperaemia and, 20-23 ; cerebral venous congestion and, 28-30 ; cerebrospinal fluid and, 5-9, 33 ; Cheyne-Stokes respiration and, 25 ; chloroform and, 18 ; chronic anaemia and, 27-28 ; compression of the brain and, 30-35 ; embolism and, 24 ; expiration and, 4-5 ; hydrocephalus and, 8 ; in sleep, 21 ; infective states and, 7 ; inspiration and, 4-5 ; intracranial pressure and, 3-5, 9-20, 30-35 ; intracranial tumour and, 34 ; ligature of arteries and, 24-27 ; lymphatics and, 5-9 ; position and, 15-19 ; principles of, 9-20 ; pulse-rate and, 18 ; respiration and, 4 ; shock and, 18 ; Traube-Hering blood-pressure curves and, 25 ; veins and, 7, 13 ; venous congestion and, 28-30

Circulation, the, in idiots, 877

Claudication, intermittent, 611-612 ; and aphasia, 418

Claustrophobia, 864, 970 ; hypnotism in, 1011 ; in neurasthenia, 749

Cleptomania, 873

Climacteric, the, and epilepsy, 488 ; and facial spasm, 639 ; and insanity, 933-935, 975, 978 ; and tetany, 580

Clonus and cerebral anaemia, 26

Clownism in hysteria, 696

Coal-gas poisoning and insanity, 907

Cocaine habit, the, and hypnotism, 1009

Cocaine poisoning and insanity, 911

Coenaesthesia, 966

Colitis, mucous, in hysteria, 710

Colonies, epileptic, 959-961

Coma, in cerebral haemorrhage, 321, 328 ; in pontine haemorrhage, 125-126

Commissure, the anterior, 98

Compositors' cramp, 680

Concepts, and aphasia, 391-394 ; centre for, 392-393, 428-429

Concussion of the brain, some milder forms of, 231-240 ; "biliousness" and, 232, 233 ; bleeding advised in, 238 ; due to blows on the cranium, 233 ; glycosuria in, 235, 237 ; indirect, 234 ; loss of consciousness and, 231-233 ; photophobia in, 233 ; prognosis, 236 ; pulse in, 234, 235 ; reflexes in, 235 ; treatment, 237-240

Conduct and insanity, 824, 842, 967

Confusion, hallucinatory, 903

Conium in tic, 635

Consanguinity, parental, and insanity, 825

Consciousness and hypnotism, 1012 ; double, after epilepsy, 953 ; in meningitis, 218

Constipation in tuberculous meningitis, 213

Contracture, hysterical, 701-702, 705 ; in intracranial tumour, 274 ; post-hemiplegic, 75, 303

Convulsions *or* fits, and aphasia, 419 ; hysteroid, 694 ; in abscess of the brain, 259 ; in alcoholic insanity, 915 ; in amaurotic family idiocy, 470 ; in cerebral haemorrhage, 321, 328 ; in cerebral syphilis, 339 ; in cerebral paralyses of children, 457, 461, 463, 466 ; in general paralysis, 359, 370-373, 376, 379 ; in hysteria, 688, 693-697, 724 ; in idiocy, 876, 882 ; in meningococcic meningitis, 178 ; in epilepsy, 486, 489, 493-504 ; in head-nodding, 663-664 ; in paramyoclonus, 652-655 ; in tetany, 588-592 ; in tic, 620, 622-626 ; in tuberculous meningitis, 212, 215 ; in tumour of the brain, 270, 272, 274 ; uncinate, 96, 264, 277

Coprolalia, in convulsive tic, 621, 624 ; in melancholia, 971

Corona radiata, the, 101 ; haemorrhage into, 324

Corpora quadrigemina, lesions of the, 119-125

Corpora striata, lesions of the, 104-109 ; haemorrhage into, 105 ; pseudo-bulbar paralysis and, 106
Corpus callosum, the, 97-100 ; functions, 98 ; haemorrhage into, 325 ; lesions of, 99, 273
Corpus restiforme, lesions of the, 154
Corpus trapezoides, the, 119 ; acoustic semi-decussation in, 431
Cortex, the, excitability of in cerebral anaemia, 26-27, 30 ; structure of and epilepsy, 942-944, 947-950 (Fig. 27)
Cough and the cerebral circulation, 28-29 ; hysterical, 709
Craft-neuroses, *see* Occupation-neuroses, 667-686
Cramps, the professional, *see* Occupation-neuroses, 667-686
Craniectomy in idiocy, 889
Cranium, the, and the cerebral circulation, 3-5 ; in general paralysis, 353 ; in idiocy, 880, 886 ; tenderness of and intracranial disease, 60, 150
Cretinoid idiocy, 878-881 (Fig. 26)
Criminal Appeal Act (1907), 1039
Criminal lunatics, 1023
Criminalists, so-called, 1020
Criminality and general paralysis, 381 ; and insanity, *see* Vice, crime, and insanity, 842-874 ; instinctive, 872 ; lunacy and, 1017-1040
Crowbar case, the American, 44
Crura cerebri, lesions of the, 116-119, 299 ; haemorrhage, 326 ; paralysis in, 117 ; sensation in, 118
Crusta pontis, the, 125
Custom and crime, 845-851
Cutis anserina in general paralysis, 373
Cyclical vice, 856-857, 874
Cyst, arachnoid, in general paralysis, 351 ; cerebral, in porencephalus, 885
Cysticercus in the fourth ventricle, 159

Dancers' cramp, 681
Day-terrors, 815-817
Deafness, and paranoia, 1004 ; and temporal lesions, 92-95 ; cortical, 93 ; hysterical, 699, 708 ; insanity in, 839 ; in cerebellar disease, 148 ; in meningococcic meningitis, 175 ; in polioencephalitis, 247
Decubitus acutus, in acute delirium, 902 ; in cerebral occlusion, 300
Decubitus, the, in tuberculous meningitis, 212, 220
Decussation of the pyramids and hemiplegia, 62
Deformities in cerebral paralyses of children, 457, 459, 460, 464
Degeneration, stigmata of, and crime, 1022
Deglutition, in meningitis, 173 ; in pontine lesions, 132 ; in tuberculous meningitis, 216
Délire chronique, 1002, 1006
Delirium, acute, 899-904 ; "brain fever," 902 ; etiology, 900 ; fever in, 901 ; morbid anatomy, 900 ; symptoms, 900 ; treatment, 902
Delirium, *e potu*, *see* Insanity, Alcoholic, 909, 912-920 ; *e potu suspenso*, 913 ; in tuberculous meningitis, 211 ; transitory post-operative, 923
Delirium tremens, 918
Delusions, 996 ; and testamentary capacity, 1047 ; in melancholia, 973-974 ; in paranoia, 996-1005
Dementia, 989-995 ; acute, 990 ; acute primary, 903, 984, 986-987 ; alcoholic, 991, 994 ; attitude in, 992 ; chronic, 991, 994 ; in cerebral syphilis, 341 ; in general paralysis, 363, 377 ; in frontal lesions, 48 ; infantile, 989 (*see* Idiocy, 874-891) ; partial, 989 ; primary, 990 ; prognosis, 991 ; secondary, 990 ; terminal, 929 ; treatment, 993
Dementia paralytica, *see* General Paralysis of the Insane, 346-385
Dementia paranoides, 1006
Dementia precox, 927-930, 990, 1006 ; and neurasthenia, 771 ; katatonic, hebephrenic, and paranoid, 929, 988
Dermatography in neurasthenia, 753
Desire and crime, 857, 863-864
Deviation, conjugate, angular gyrus and, 80-83 ; in cerebral haemorrhage, 322 ; in frontal lesions, 43, 47 ; in occipital lesions, 85 ; in pontine lesions, 127, 129
Diabetes mellitus and insanity, 840, 908, 976
Diaphragm, paralysis of the, in polioencephalitis, 248
Diarrhoea and cerebral anaemia, 28
Diarrhoea, church, 757
Diaschisis, 38, 73, 395, 411
Diphtheria and cerebral occlusion, 294
Diplegia in children, 459
Dipsomania, hypnotic treatment of, 1009
Disease, Bergeron's, 631 ; Bernhardt's, 679 ; Dubini's, 542, 554, 621, 631, 656 ; Friedreich's (Paramyoclonus), 649-659 ; Gilles de la Tourette's, 619, 621, 657 ; Heine-Medin's, 241 ; Hirschsprung's, 583 ; Little's, 456, 598 ; Parkinson's, 473-482
Disseminated sclerosis, and general paralysis, 379 ; and hysteria, 687, 717 ; and insanity, 839 ; and neurasthenia, 771
Dolichocephaly in idiocy, 879
Dotage, 935-939
Dreams and night-terrors, 807
Dropsy in the brain, 202
Drowsiness and disease of the hypophysis, 162 ; in tuberculous meningitis, 211
Drug-habits, hypnotic treatment of, 1009
Dubini's disease, 542, 554-555, 621, 631, 656
Dura mater, irritation of the, and epileptiform convulsions, 58

Dust, tuberculous, 207
Dysarthria, 399-405; aphthongia, 400; idioglossia, 400; lalling, 400; spastic, 403; stammering, 400; test phrases for, 401
Dyschromatopsia in alcoholic insanity, 916
Dysdiadochokinesia in cerebellar disease, 145
Dysmetria in cerebellar disease, 145
Dysnoia, 996
Dysphagia in medullary lesions, 158
Dysuria in neurasthenia, 762, 770

Ears, the, disease of, and abscess of the brain, 250-254, 256, 259, 263, 265; disease of, and tuberculous meningitis, 221; inflammation of, in meningitis, 170
Eccentricity and insanity, 826
Echo, the epileptic, 952
Echokinesis in convulsive tic, 621, 625
Echolalia, in amnesia, 429; in convulsive tic, 621, 624
Eclampsia in idiocy, 882
Eclampsia nutans, 659
Eclampsia rotans, *see* Head-nodding, 661-667
Education and insanity, 834; and night-terrors, 814; and tuberculous meningitis, 206
Ego-centric insanity, 997
Electricity in insomnia, 801
Embolism, cerebral, 290-293, 330; and aphasia, 418; paradoxical, 291
Emotion, facial movements, and the optic thalami, 111
Emotions, the, and chorea, 525-526; and epilepsy, 491; and facial spasm, 639; and hysteria, 690, 691; and paralysis agitans, 475; and paranoia, 1006; in melancholia, 964-965, 968, 979
Encephalitis (*see also* Polioencephalitis), acute haemorrhagic, 261; in tuberculous meningitis, 202
Endocarditis and latent cerebral abscess, 254; and cerebral haemorrhage, 315
Enophthalmos in thrombosis of the posterior cerebellar artery, 161
Enteric fever and insanity, 838, 841; and meningitis, 191-193
Enteroptosis and neurasthenia, 754-759
Enterospasm in neurasthenia, 758
Ento- and epi-peripheral sensations in melancholia, 979
Enuresis and night-terrors, 811; hypnotism in, 1010
Epilepsia media, 500
Epilepsy, Jacksonian, 37, 57-61, 80; in cerebral abscess, 264; in cerebral tumour, 269, 273; in intracranial syphilis, 339; in lesions of the centrum ovale, 102; sensory phenomena in, 60; types of, 59; visual, 276
Epilepsy, Major *and* Minor, 483-519, 941-961; age in, 488; alcohol and, 508; and cerebral anaemia, 26; and cerebral syphilis, 339-340; and criminal lunacy, 1036; and idiocy, 883; arrest of attacks in, 504; arsenic in, 515, 516; aura in, 494, 495-500, 947-952; automatism after, 503; belladonna in, 516; biborate of sodium in, 516; birth-palsy and, 490; bromides in, 512-516; causes, 487-492; cerebral congestion in, 29; climacteric and, 488; convulsions in, 486, 489, 493-504; course, 505; cry in, 952; death in, 507; definition, 483-485; diagnosis, 330, 379, 509; dreamy types of, 96, 264, 277, 500; emotions and, 491; general management of, 517; heredity in, 487, 488; hypnotism in, 1011, 1015; hysteria or, 509; hysteroid convulsions after, 503, 510; idiopathic, 485; in eclampsia nutans, 660; infantile convulsions and, 464-465, 489-490; injury and, 490; insanity and, 839, 941-961; intermittent mental, 954; local onset of, 496, 502; marriage and, 518; masked, 941; mental state in, 507; minor, 500-503, 509; night-terrors and, 817; organic, 508-509; pathology, 492; post-epileptic symptoms, 502; prodromes, 495, 952; prognosis, 511; psychical, 941; puberty and, 505; recurring tendency in, 485; reflex, 486, 492, 508; secondary forms, 507; sex and, 487-489; sleep and, 505; status epilepticus, 506, 517; symptoms, 493; syncope or, 510; teething and, 489; tetany or, 598; toxic states and, 490, 508; treatment, 512-519; trephining in, 517; uncinate, 264, 277; vertigo and, 496, 501, 510; warnings in, 494-500, 947-952; zinc salts in, 516
Epilepsy, larval, 941, 952; masked, 941, 952; nodding, *see* Eclampsia nutans, 609; spinal, *see* Paramyoclonus, 649-659
Erb's sign in tetany, 592, 598
Ergotism and tetany, 579, 581
Eunuchism, pituitary, 162
Exaltation, in criminal lunacy, 1035; in general paralysis, 360, 366-367; in paranoia, 1001
Exophthalmos in cerebral thrombosis, 304
Eye, circulation in the, 12
Eyeball, movements of the, and the angular gyrus, 81-83

Face, hemispasm *or* histrionic spasm of the, *see* Spasm, facial, 638-649
Facial paralysis, recurrent, 608
Facility in general paralysis, 361, 368
Family history, in paramyoclonus, 650; in pseudo-bulbar paralysis, 107-108
Family periodic paralysis, recurrent, 604
Fatigue in neurasthenia, 732
Fear in melancholia, 970
Fever, in cerebral abscess, 255; in general

paralysis, 373 ; in hysteria, 713 ; in intracranial tumour, 278 ; in neurasthenia, 763 ; in night-terrors, 810 ; in tuberculous meningitis, 210, 217
Fevers, the, and cerebellar lesions, 142 ; and idiocy, 883 ; and insanity, 838, 841
Fillet, the mesial, and cutaneous sensibility, 79
Fit, 501, *see* Convulsions
Flexibilitas cerea, 988 ; in melancholia, 970
Flexner-Jobling antimeningococcic serum, 180
Flora's sign and malingering, 774
Folie circulaire, 962 ; and acute mania, 893 ; in general paralysis, 359, 363 ; testamentary capacity in, 1049
Folie à deux, 974
Folie à forme double, 962, 975
Folie du doute, 625, 628 ; hypnotism in, 1011 ; in general paralysis, 368
Folie raisonnante mélancholique, 972, 997
Fontanelle, the, in meningitis, 173, 219
Forcible feeding in insanity, 982
Fortification figures in migraine, 563 (Plate II.)
Friedreich's disease, *see* Paramyoclonus, 649-659
Frontal lobe, lesions of the, 40-51 (Figs. 5 and 6) ; abscess, 45 ; experimental pathology, 40-44 ; haemorrhage, 44 ; mental symptoms in, 48 ; trauma, 44-45 ; tumour, 45-50
Fundus oculi, the, in amaurotic family idiocy, 470
Furor transitorius in epilepsy, 953

Gait, the, in alcoholic insanity, 915 ; in cerebellar disease, 137-140, 143 ; in cerebral paralyses of children, 456, 460 ; in hydrocephalus, 120 ; in lesions of the corpora quadrigemina, 120 ; in lesions of the corpora striata, 107-108 ; in paralysis agitans, 479
Garrotting and the cerebral circulation, 28
General Paralysis of the insane, 346-385 ; acute, 360 ; adolescent, 364 ; alcoholism and, 350, 377-378 ; bacteriology, 357 ; brain in, 352, 354 ; cerebral haemorrhage or, 331 ; circular, 363 ; convulsions in, 359, 370-373, 376, 379 ; criminality or, 381 ; demented, 363, 377 ; disseminated sclerosis and, 379 ; duration, 365-366 ; emotionality in, 367 ; epilepsy and, 956-958 ; etiology, 348-351 ; exaltation in, 360, 366 ; facility in, 361, 368 ; forms of, 360-365 ; frequency of, 348 ; general description, 346-347 ; hallucinations in, 369 ; hysteria and, 371, 376 ; in women, 364 ; lead poisoning and, 378 ; maniacal, 360, 376 ; megalomania in, 367 ; melancholic, 362 ; memory in, 368 ; micromania in, 367 ; moral perversions in, 359 ; morbid anatomy, 351-356 ; neurasthenia and, 770 ; nutrition in, 373 ; paralysis in, 369 ; pathogeny, 356-358 ; prodromes, 358-360 ; prognosis, 382 ; pulse in, 374 ; pupils in, 368 ; reflexes in, 370 ; remissions in, 363, 374 ; rest in, 383 ; senile, 364 ; sensation in, 359, 368-369 ; spinal, 364 ; sunstroke and, 351, 379 ; symptoms, 366-375 ; syphilis and, 349-351, 378, 832 ; termination, 375 ; treatment, 383 ; venery and, 349 ; writing in, 370
General paralysis, juvenile, 364 ; and syphilitic meningitis, 193
Genius, and insanity, 826 ; and the cerebral circulation, 21-22
Gennari, the line of, 86, 88
Geophagy and insanity, 866
Gilles de la Tourette's disease, 619, 621, 657
Globus hystericus, 694
Glycosuria, in cerebellar disease, 149 ; in concussion of the brain, 235, 237 ; in medullary lesions, 159 ; in pontine lesions, 134
Goldbeaters' cramp, 679
Gonococcus, the, and meningitis, 191
Gout, and cerebral haemorrhage, 308 ; and insanity, 840, 908, 975
Graves' disease and insanity, 841
Gumma, cerebellar, 339 ; cerebral, 339, 343 ; meningeal, 339
Gyrospasm of the head, 661-667
Gyrus (*see also* Areas *and* Lobe), angular, 79-85, 93, 387, 425 ; anterior transverse of Heschl, 92 ; Broca's, 427 ; central, 53 ; frontal, and aphasia, 387-393 ; falciform, 76 ; fornicatus, 76, 93 ; fusiform, 88 ; hippocampal, 76, 95 ; limbic, 76 ; lingual, 86, 88 ; marginal, 78 ; paracentral, 51, 63, 78, 93 ; postcentral, 53-55, 71, 73 ; postero-parietal, 73 ; precentral, 53-55, 63, 71 ; supramarginal, 93, 387 ; temporal, 90-97

Habit in forming tics, 616, 617
"Habit-chorea," 615, 619
"Habit-spasm" and tic, 615, 619
Haematoma, subdural, 319, 327
Haemorrhage, arachnoid, 317, 319 ; cerebellar, 134-136 ; cerebral (*q.v.*), 105-106, 307-334, 339, 454-455 ; corpus callosal, 99 ; corpus striatal, 104 ; frontal, 44 ; in hysteria, 712 ; intestinal, and lesion of the optic thalamus, 115 ; intracranial, physics of, 33 ; medullary, 155-156 ; meningeal, 319, 327 ; optic thalamic, 110, 115 ; pontine, 125 ; subcortical, 102 ; subdural, 319 ; temporal, 93 ; ventricular, 318, 327
Hallucinations, in general paralysis, 369 ; in paranoia, 999-1000
Hammermen's cramp, 679
Headache, in abscess of the brain, 255 ; in cerebral occlusion, 295, 306 ; in cerebral

syphilis, 337 ; in meningism, 199 ; in meningitis, 193, 198, 213 ; in neurasthenia, 745, 763, 785 ; in tumour of the brain, 270
Headache, bilious *or* sick, *see* Migraine, 558-569
Headache, occipital, 745
Head-nodding, 661-667 ; bromides in, 666 ; dentition and, 662 ; diagnosis, 566 ; etiology, 661 ; eyes in, 662, 665 ; nodding in, 663 ; nystagmus in, 663-665 ; pathology, 662 ; symptoms, 663
Head-retraction, 181 ; in meningococcic meningitis, 173 (Figs. 12-16) ; in tuberculous meningitis, 214
Hearing, the, in lesions of the corpora quadrigemina, 122 ; in lesions of the optic thalamus, 115 ; in meningitis, 175 ; in polioencephalitis, 247 ; in temporal lesions, 90-96 ; in tumour of the brain, 276
Heart, the, in chorea, 536-538, 543 ; in neurasthenia, 752 ; movable, in neurasthenia, 754
Heart-disease and insanity, 839, 975
Heat-rheumatism, *see* Tetany, 578-604
Heine-Medin disease, the, 241
Hemiachromatopsia, in pure word-blindness, 435
Hemianaesthesia, and hemiplegia, 60, 66-75 ; and hysteria, 84, 104, 698 ; and lesions of the gyrus fornicatus, 76 ; and lesions of the internal capsule, 104 ; functional, 104
Hemianopia, Hemianopsia, *or* Hemianopsy, bitemporal, 162 ; calcarine area and, 86-89 ; in angular lesions, 81, 84 ; in cerebral abscess, 264 ; in cerebral haemorrhage, 297, 325 ; in frontal lesions, 43 ; in hysteria, 707 ; in lesions of the crura cerebri, 118 ; in lesions of the hypophysis, 162 ; in lesions of the internal capsule, 104 ; in lesions of the optic thalamus, 114 ; in occipital lesions, 85-90 ; in parietal lesions, 81, 84 ; in ophthalmic migraine, 605 ; in pure word-blindness, 435
Hemianopia, cerebral, 88-90 ; homonymous, 88-90, 104
Hemiaprazia, 449
Hemiataxia in lesions of the crura cerebri, 118
Hemichromatopsy, homonymous, 89
Hemicrania, *see* Migraine, 558-569
Hemiopia, *see* Hemianopia
Hemiparesis and lesions of the optic thalami, 110-111
Hemiplegia, alternate, 127, 128 ; and carotid occlusion, 26 ; and the optic thalamus, 110 ; and the Rolandic area, 61-63 ; astereognosis in, 71 ; in cerebral syphilis, 336, 338 ; in cerebral thrombosis, 296 ; in cerebral tumour, 273 ; in general paralysis, 371 ; in hysteria, 702 ; incomplete, 336 ; infantile, 463-467 ; post-epileptiform, 57 ; recurrent, 606 ; syphilitic, 295
Heredity, in alcoholic insanity, 913 ; in cerebellar ataxia, 142 ; in cerebral haemorrhage, 308 ; in chorea, 520, 548 ; in dementia precox, 927 ; in epilepsy, 487, 488, 518-519, 946 ; in facial spasm, 638 ; in hysteria, 689 ; in idiocy, 874-877 ; in insanity, 825, 833, 921, 927, 930, 978 ; in neurasthenia, 737, 741, 753 ; in occupation-neuroses, 672 ; in paramyoclonus, 650 ; in senile chorea, 552 ; in stammering, 572 ; in tic, 616, 629
Hernia cerebri, pathology of, 34
Hernia, in cretinism, 879 ; in mongolism, 878
Hippocampus, the, 76
Hippus, in chorea, 536 ; in tuberculous meningitis, 216
Hirschsprung's disease and tetany, 583
Homén's syndrome, 107, 109
Hydrocephaloid disease, 220
Hydrocephalus, and intracranial tumour, 280 ; gait in, 120 ; in idiocy, 881 ; in syphilitic meningitis, 196 ; in tuberculous meningitis, 202, 208, 227 ; pathology of, 35
Hydrocephalus, acute, 202, *see* Meningitis, Tuberculous, 201-228
Hydrotherapeutics, in hysteria, 723 ; in neurasthenia, 782
Hyoscine, in epilepsy, 517 ; in insomnia, 800 ; in mania, 898
Hyoscyamine in insomnia, 800
Hyperacusis, neurasthenic, 750
Hyperaemia, cerebral, 20-23
Hyperaesthesia, in hysteria, 700 ; in tuberculous meningitis, 216
Hypermetropia and intracranial tumour, 279
Hyperostosis, cranial, in general paralysis, 351
Hyperpiesis and neurasthenia, 772
Hyperpyrexia, hysterical, 713-714, 763 ; in cerebral haemorrhage, 322 ; in meningococcic meningitis, 178 ; in pontine haemorrhage, 126
Hypnotics, in neurasthenia, 785 ; in sleeplessness, 797-801
Hypnotism in insanity, 1007-1017 ; and aphasia, 419 ; and stupor, 987 ; dangers of in alcoholic insanity, 920 ; in dipsomania, 1009 ; in drug habits, 1009 ; in epilepsy, 1011, 1015 ; in hysteria, 691, 724, 1011 ; in insanity, 1007, 1014 ; in insomnia, 802 ; in morphinism, 1009 ; in neurasthenia, 783, 1011 ; in obsessions, 1011 ; in vice, 1010 ; in writers' cramp, 686 ; methods of, 1013 ; results of, 1014-1017 ; theories of, 1012
Hypochondriasis, 972, *see also* Melancholia 964-984 ; and hysteria, 717
Hypophysis cerebri, lesions of the, 162

Hypotonia in cerebellar tumour, 144

Hysteria, 687-727 ; age and, 689 ; anaesthesia in, 697-699 ; ankle-clonus in, 701, 704 ; anorexia nervosa, 710 ; anuria in, 713 ; aphonia in, 420-422, 708 ; arteries in, 711 ; aura in, 694 ; Babinski's sign in, 703, 717 ; breast in, 711 ; Charcot's blue oedema in, 712 ; chorea and, 526, 706 ; contractures in, 701-702, 725 ; cortical lesions and, 67, 74 ; cough in, 709 ; course, 718 ; death due to, 719 ; definition, 687-689 ; diagnosis, 716-718, from cerebral abscess 264, from disseminated sclerosis 687, 717, from epilepsy 509, from facial spasm 644, from general paralysis 371, 376, from intracranial tumour 280, from myelitis 702-703, from neurasthenia 768, from paramyoclonus 650, 657, from tetany 598, from tic 621, 626, 632, from tuberculous meningitis 220 ; emotions and, 690, 691 ; epidemic, 690 ; etiology, 689 ; fever in, 713 ; fits in, 688, 693-697, 724 ; globus in, 694 ; *grande hystérie*, 695 ; haemorrhages in, 712 ; hearing in, 708 ; heart in, 711 ; hemianaesthesia in, 698 ; hemiplegia in, 702 ; heredity in, 689 ; hydrotherapy in, 723 ; hyperaesthesia in, 700 ; hypnotism in, 691, 724, 1011 ; hypotheses of, 690-693 ; hysteroid fits, 694 ; incomplete attacks in, 697 ; insanity and, 715 ; joint-troubles in, 710 ; mental state in, 714-715 ; mucous colitis and, 710 ; neurasthenia and, 717 ; nutrition in, 712 ; ocular symptoms, 706-708 ; oedema in, 712 ; paralysis in, 700-704, 725 ; paroxysms in, 688, 693-697 ; post-epileptic, 503, 510, 696 ; prevention, 719 ; prognosis, 718 ; psychical origin of, 687, 691, 704 ; reflexes in, 701, 704 ; sex and, 689 ; spine in, 710 ; stigmata of, 688, 698, 718 ; subconsciousness and, 692-693 ; suggestion and, 691 ; symptoms, 688-689, 693-716 ; tenderness in, 700 ; treatment, 719-726 ; tremor in, 705 ; urine in, 694, 696, 705, 713 ; vasomotor phenomena, 711-712 ; vomiting in, 709, 725 ; Weir Mitchell treatment in, 722

Hystero-epilepsy, 503-504, 695-697 ; hypnotism in, 1012, 1015 ; traumatic, 764

Hysteroid convulsions, 694 ; after epilepsy, 503, 510, 696

Ideas, imperative, in epilepsy, 952

Idées fixes in tic, 625, 626

Idiocy, and ligature of the carotids, 26-28, 30 ; post-meningitic, 181

Idiocy, amaurotic family, 468-473 ; diagnosis, 472 ; morbid anatomy, 468 ; pathology, 469 ; symptoms, 469 ; treatment, 472

Idiocy and Imbecility, 874-891 (Fig. 26) ; alcoholism and, 875 ; atrophy of the brain in, 884 ; birth injuries and, 876 ; brain in, 884-887 ; cerebellum in, 886 ; chilblains in, 889 ; convulsions and, 876, 882 ; craniectomy in, 889 ; cranium in, 880, 886 ; cretinoid, 878, 881 (Fig. 26) ; developmental, 875 ; diet in, 888 ; epileptic, 883 ; etiology, 874 ; heredity in, 874-877 ; hydrocephalic, 881 ; hypertrophic, 882, 884 ; imitativeness in, 871 ; microcephalic, 881 ; mongolian, 877-881 (Fig. 26) ; natal, 875 ; paralytic, 883 ; pathology, 884-887 ; phthisis and, 875 ; porencephalus in, 885 ; post-febrile, 883 ; sclerosis of the brain in, 885 ; signs of, 878 ; simple congenital, 877 ; softening of the brain in, 885 ; spinal cord in, 886 ; sunstroke and, 876 ; syphilis and, 875 ; traumatic, 882 ; treatment and training in, 887-891 ; tuberculosis in, 885-886 ; tumour of the brain in, 885 ; types of, 877

Idioglossia, 576-577 ; and aphasia, 400

Imitation, and chorea, 526 ; and stammering, 572 ; and tic, 617

Imitativeness in idiocy, 878

Impediments of speech, 569-578 ; adenoids and, 572 ; alphabet and, 570 ; associated movements and sounds in, 573 ; causation, 572 ; examination of case of, 573 ; heredity in, 572 ; idioglossia, 576-577 ; imitation in, 572 ; lisping, 575 ; mechanism of normal articulation, 569 ; pathology, 572 ; prognosis, 574 ; stammering, 569 ; stuttering, 569 ; treatment, 574, 577

Imperception (=apraxia), 447

Impulse and vice, 858

Infantilism, mental, in tic, 616

Infarction of the lung in cerebral occlusion, 306

Influenza, and insanity, 838, 841, 905-907 ; and meningitis, 187 ; and neurasthenia, 769-770

Inquisition in lunacy, 1042

Insanity, Alcoholic, 909, 912-920 ; acute, 918 ; age and, 914 ; causes of alcoholism and, 913-914 ; chronic, 919 ; convulsions in, 915 ; diagnosis, 715, 747 ; forms of, 918-919 ; gait in, 915 ; heredity in, 913 ; hypnosis for, 920 ; Korsakow's syndrome and, 915, 919 ; pathology, 914 ; pseudo-paralysis and, 914-916 ; subacute, 919 ; symptoms, 915 ; treatment, 919

Insanity and Epilepsy, 941-961 (Fig. 27) ; age and, 945 ; altruism in, 956 ; aura in, 947-952 ; automatism, 953-954 ; bromides in, 958-959 ; colonies for, 959-961 ; dementia in, 956 ; diet in, 958-960 ; general paralysis and, 956-958 ; heredity and, 946 ; imbecility in, 955 ; insanity in, forms of, 947 ; irritability in, 955 ; mania in, 953 ; morbid anatomy, 942-944 ; post-paroxysmal, 953 ; pre-paroxysmal, 952 ; ratios of, 945 ; salt in, 958 ; sex and, 945 ; treatment, 958-961

Insanity and Surgical operations, 921-925 ; amnesia in, 925 ; drugs and, 923 ; etiology, 921 ; heredity and, 921 ; prognosis, 924 ; sepsis and, 922 ; symptoms, 923 ; treatment, 925

Insanity, circular, 962, see *Folie circulaire* ; confusional, 903, 989 ; criminal, 1033, *see* Lunacy, Criminal, 1017-1040

Insanity, Crime, and Vice, 842-874 ; alcoholism and, 856-857 ; aversions in, 864 ; cleptomania and, 873 ; conduct and, 842 ; crime, 857 ; custom and, 845 - 851 ; cyclical, 874 ; definitions of, 842 - 851 ; desire and, 857, 863-864 ; impulse and, 858 ; instinctive, 872 ; intention and, 860, 865-869 ; motive and, 861, 865-869 ; obsession and, 865 - 867 ; paroxysms of, 856-857, 874 ; religion and, 846; responsibility in, 869, 872 ; self-indulgence and, 849 - 851 ; self-restraint and, 853-857 ; sexual aberrations and, 851, 853, 856, 863, 866, 873 ; treason and, 844, 849, 851 ; vice, 849 - 851 ; vice and insanity, 851 - 857 ; will and, 858 ; wrong-doing and, 847-851, 857

Insanity, delusional, 996, *see* Paranoia, 995-1007

Insanity, Epochal, 926 - 941 ; climacteric, 933-935 ; heredity in, 927, 930 ; of adolescence and puberty, 927-930 ; of lactation, 933 ; of pregnancy, 930 ; of puberty and adolescence, 927-930 ; puerperal, 930-933 ; senile, 935-939 ; tuberculous, 839, 939-941

Insanity, litigious, 1002-1003 ; maniacal depressive, 893 ; moral, 989 ; of negations, 989 ; partial, 996 ; recurrent 961 - 963, circular insanity and, 962

Insanity, Systematised delusional, 995-1007 ; acquired, 996 ; age and, 997 ; deafness and, 1004 ; delusions in, 997 -1005 ; emotion and, 1006 ; hallucinations in, 999-1000 ; judgment and, 995 - 996 ; memory in, 1003 ; mystic interpretation in, 1003, 1005 ; neologism in, 1005 ; original, 996 ; persecutional, 997 ; primary, 997 ; religion and, 1003 ; secondary, 997, 1005 ; systematisation in, 996, 998 ; voices in, 998-999

Insanity, testamentary capacity in, 1047-1049

Insanity, Toxic, 904-912, 994 ; albuminuria and, 908 ; alcohol and, 909, 912-920 ; anaesthetics and, 907 ; arsenic and, 912 ; belladonna and, 910 ; carbon dioxide and, 907 ; carbon monoxide and, 907 ; chloral and, 911 ; coal-gas poisoning and, 907 ; cocaine and, 911 ; diabetes and, 908 ; fever and, 907 ; glandular defects and, 909 ; gout and, 908, 983 ; habit and, 905 ; influenza and, 905 - 907 ; iodoform and, 909 ; jaundice and, 908 ; lead and, 911 ; malaria and, 905 ; morphine and, 910 ; pellagra and, 910 ; phosphorus and, 912 ; septicaemia and, 909 ; sparteine and, 911

Insomnia, 792-803 ; hypnotism in, 1008, 1010 ; in cerebral syphilis, 337 ; in melancholia, 983 ; in neurasthenia, 733 750, 770, 785-786 ; in senility, 936-937

Instinct and vice, 872

Insula, the, 93, 96-97

Intention and crime, 860, 865-869

Intention-tremor, *see* Tremor, Intention

Internal capsule, the, haemorrhage into, 324 ; lesions of, 100-104

Intracranial pressure, 9-20, 30-35

Iodides, the, in alcoholic dementia, 994 ; in cerebral syphilis, 345 ; in intracranial tumour, 283, 287

Iodipin in meningococcic meningitis, 183

Iodism, 345

Iodoform and insanity, 909

Jaundice and insanity, 908

Jews, the, and amaurotic family idiocy, 468

Joints, the, in hysteria, 710

Judgment, in dementia, 991 ; in paranoia, 995-996

Katatonia, 930, 987-988

Kernig's sign, in meningococcic meningitis, 177, 222 ; in meningism, 199 ; in tuberculous meningitis, 214

Kidney disease and insanity, 840

King's pleasure lunatics, 1026

Knee-jerks, the (*see also* Reflexes), in lesions of the corpora quadrigemina, 123 ; in lesions of the optic thalamus, 113

Korsakow's syndrome, 915, 919 ; in old age, 365

Krause, the lines of, 948

Kronomania, 627

Lactation and insanity, 933

Lalling and aphasia, 400

Latah, 619, 625

Law, the, and criminal lunacy, 1017-1040 ; and lunacy, 1040 - 1049 ; aphasia and, 1048 ; Commissioners' rules, 1043 ; delusions and, 1047 ; forms, 1045 - 1047 ; inquisition, 1042 ; mechanical restraint, 1044 ; medical attendant, 1044 ; medical certificates, 1043 - 1046 ; petition for reception order, 1041 ; reception order, 1042 ; single patients, 1043 ; statement of particulars, 1041 ; testamentary capacity, 1047-1049 ; urgency order, 1040, 1045

Lead-poisoning, and cerebral haemorrhage, 308, 309 ; and general paralysis, 378 ; and insanity, 911

Leptomeningitis in polioencephalitis, 244

Leucocytosis, in abscess of the brain, 258, 260 ; in meningococcic meningitis, 179 ; in tuberculous meningitis, 218

Levator palpebrae superioris, the, and lesions of the angular gyrus, 81

Lip-reading, 390

Lipomas in cretinism, 879

Lisping, 575

Little's disease, 456, 598

"Live blood" in blepharospasm, 640

Lobe *or* lobule (*see also* Areas *and* Gyrus), angular, 79-85 ; falciform, 76-79 ; frontal, 40-51 (Figs. 5 and 6) ; hippocampal, 95 ; Island of Reil, 96-97 ; limbic, 76-79 ; occipital, 81, 85-90 ; parietal, 79-85 ; pyriform, 95 ; quadrate, 78 ; supramarginal, 79-85 ; temporal, 90-97 ; uncus, 96

Love affairs and insanity, 836

Lucid intervals in insanity, 1049

Lumbar Puncture, in general paralysis, 380 ; in influenzal meningitis, 187 ; in meningococcic meningitis, 179, 181 ; in meningism, 199 ; in pneumococcic meningitis, 184 ; in polioencephalitis, 242, 248 ; in serous meningitis, 198 ; in staphylococcic and streptococcic meningitis, 186 ; in syphilitic meningitis, 197, 282 ; in tuberculous meningitis, 218, 226

Lunacy, Criminal, in England, 1017-1040 ; acquisitiveness and, 1019 ; alcoholism and, 1036 ; causes of, 1036 ; certification in, 1037 ; criminal anthropology and, 1020 ; Criminal Appeal Act (1907), 1039 ; criminal lunatics, 1023 ; discharge of prisoners, 1032 ; epilepsy in, 1036 ; exaltation in, 1035 ; insane criminals, 1029 ; insanity in, 1033 ; King's pleasure lunatics, 1026 ; malice and, 1019 ; mania in, 1035 ; melancholia in, 1035 ; murder, 1029-1032 ; pauper lunatics, 1026 ; procedure in, 1037-1040 ; psychology of, 1018-1023 ; Secretary of State's lunatics, 1026 ; sexual offences, 1019

Lunatics, criminal, defined, 1023 ; ill-treatment of, 1043

Lypemania, 964

Macula lutea, innervation of the, 88-89

Main en griffe in tetany, 589

Maladie des tics convulsifs, 619, 621

Malaria and insanity, 905

Malice in criminal lunacy, 1019

Malingering, and criminal lunacy, 1035 ; and neurasthenia, 729, 774

Mania, 892-899 ; *a potu*, 918 ; acute, 892 ; acute delirious, *see* Delirium, acute, 899-904 ; chronic, 991 ; ephemeral, 892 ; gravis, *see* Delirium, acute, 899-904 ; hysterical, 893 ; impulsive, 895 ; epileptic, 953 ; in adolescence, 928 ; in criminal lunacy, 1035 ; in general paralysis, 360-362 ; in hemiplegia, 300 ; in melancholia, 896 ; moral, in epilepsy, 953 ; recurrent, 892 ; religious changes and, 893 ; senile, 892 ; simple, 893 ; simple acute, 895 ; simple hysterical, 893 ; transitory, 921 ; treatment, 894-898

Mannerisms and tic, 616

Marriage, and epilepsy, 518 ; and hysteria, 720 ; and neurasthenia, 761, 789

Mastication, and cortical lesions, 66 ; and the optic thalami, 112

Medulla oblongata, lesions of the, 155-162 ; abscess, 156 ; compression, 157 ; haemorrhage, 155, 327 ; myelitis, 157 ; softening, 156 ; tumour, 156

Megalomania, in alcoholic insanity, 917 ; in general paralysis, 367

Megrim, *see* Migraine, 558-569

Melancholia and Hypochondriasis, 964-984 ; aberrant, 975 ; age and, 965 ; anger in, 970 ; causal characters, 975 ; change in, 980 ; conduct in, 967 ; definition, 965 ; degrees of, 967 ; delusions in, 973-974 ; digestion in, 976, 978 ; emotions and, 964-965, 968, 979 ; fear in, 970 ; feeding in, 982 ; in criminal lunacy, 1035 ; in general paralysis, 362 ; menstruation in, 976 ; neurasthenia and, 773 ; nutrition in, 976 ; occupation and, 965 ; pathology of, 966 ; predisposition to, 965-966 ; prevalence, 965 ; prodromes, 967 ; prognosis, 977 ; progress, 977 ; recurrent, 975 ; religion and, 965, 981 ; remittent, 975 ; rest in, 981 ; stuporose, 986 ; symptoms, 974-977 ; termination, 977 ; treatment, 978 - 983 ; varieties of, *see below*

Melancholia agitans *or* agitata, 971 ; attonita, 970, 984 ; cataleptica, 970 ; convulsiva, 971 ; ecstatica, 971 ; errabunda, 971 ; excitata, 971 ; impulsiva, 971 ; malevolens, 969 ; maniaca, 971 ; misanthropica, 969 ; moralis, 968 ; passiva, 970 ; religiosa, 969 ; resistiva, 970 ; suicidal, 969, 974-975

Memory, the, and testamentary capacity, 1047-1048 ; in general paralysis, 368 ; in neurasthenia, 746 ; in paranoia, 1003

Meningism, 199-201, 221

Meningitis, 165-201 (Figs. 11-19) ; anthrax and, 189 ; classification, 165-166 ; definition, 165 ; gonococcic, 191 ; influenzal, 187 ; typhoid, 191-193

Meningitis, Meningococcic, 166-184 (Figs. 11-16 ; Pl. I.) ; age and, 167 ; agglutination-test in, 172 ; bacteriology, 170 ; blindness in, 174 ; cerebrospinal fluid in, 179 ; clinical features of, 172 ; crisis in, 178 ; deafness in, 175 ; diagnosis, 180-182, 222 ; distinction from epidemic meningitis, 172 ; duration, 180 ; eruptions in, 173, 179 ; etiology, 167 ; fontanelle in, 170 ; head-retraction in, 173 (Figs. 12-16) ; hydrocephalus in, 169, 179, 184 ; leucocytosis in, 179 ; lumbar puncture in, 179, 181 ; method of infection in, 168 ; morbid anatomy, 168 ; mortality, 180 ; ocular signs in, 174 ; opisthotonos in, 176-178 ; opsonic index in, 171 ; paralysis in, 178 ; prognosis, 180 ; spasm in, 176-178 ; tem-

perature in, 178 ; treatment, 182-184 ; vomiting in, 173
Meningitis, mumps and, 189 ; pneumococcic, 184-185 ; posterior-basic, *see* Meningitis, Meningococcic, 166-184 ; purulent, in general paralysis, 353 ; rheumatic, 188 ; secondary pneumococcic, 185 ; serous, 197-199 ; simple, *see* Meningitis, Meningococcic, 166-184 ; staphylococcic and streptococcic, 185 ; streptotrichial, 189-191 ; syphilitic, 193-197 (Figs. 17-19) ; typhoid, 191-193
Meningitis, Tuberculous, 201-228 ; age and, 203 ; associated lesions, 209 ; circumscribed, 209 ; constipation in, 213 ; convulsions in, 212, 215 ; decubitus in, 212, 220 ; definition, 201 ; delirium in, 211 ; diagnosis, 219-224 ; duration, 219 ; ear disease and, 221 ; encephalitis in, 202 ; facies in, 212 ; headache in, 213 ; history, 202 ; hydrocephalic cry in, 212 ; hydrocephalus in, 202, 208, 227 ; hysteria and, 220 ; leucocytosis in, 218 ; lumbar puncture in, 218, 226 ; milk and, 205, 224 ; morbid anatomy, 207-209 ; onset, 210 ; pain in, 213 ; paralysis in, 215 ; paths of infection in, 204-207 ; photophobia in, 212 ; prodromes, 211 ; prognosis, 218 ; prophylaxis, 224 ; pulse in, 213-214 ; rigidity in, 215 ; sex and, 203 ; surgical interference and, 206 ; symptoms, 210-218 ; treatment, 224-227 ; tremor in, 215 ; tuberculin in, 225 ; urine in, 211, 213, 217 ; vertex cases of, 219, 222 ; vomiting in, 211, 213
Meningo-encephalitis, tuberculous, 208
Menopause, the (*see also* Climacteric), and insanity, 933-935
Menstruation and melancholia, 976
Mental diseases, Introduction to, 823-842 ; age and, 829, 838 ; alcoholism and, 833 ; brain diseases and, 837-838 ; causation, 827, 835 ; classification of, 827 ; conduct and, 824 ; consanguinity and, 825 ; crime and, 824-827, 832 ; definition of, 823 ; eccentricity and, 826 ; education and, 834 ; fevers and, 838, 841 ; general diseases and, 838-841 ; genius and, 826 ; heart disease and, 839 ; heredity and, 825, 833 ; increase in, 828 ; injury and, 837 ; kidney disease and, 840; legal test of, 824 ; love-affairs and, 836 ; neurosis and, 825-827, 833-834 ; old age and, 838 ; phthisis and, 839, 939 ; privation and, 838 ; psychoses and, 827 ; religious excitement and, 835 ; sanity and, 824 ; season and, 832 ; sex and, 829 ; sexual aberrations and, 836-837 ; solitary confinement and, 832 ; statistics of, 830-831 ; sunstroke and, 837 ; uterine disorders and, 837 ; venereal disease and, 866 ; whooping-cough and, 839 ; worry and, 835
Mental stupor, 984-988
Mental weakness, *see* Dementia, 989-995 ; in cerebral syphilis, 343 ; in frontal lesions, 48 ; in general paralysis, 382 ; in intracranial tumour, 272; testamentary capacity in, 1047
Meralgia paraesthetica, 679
Meynert's solitary cells, 86
Microcephaly, 467, 881 ; craniectomy in, 467, 889
Micromania in general paralysis, 367
Migraine, 558-569 ; anaesthesia in, 564 ; definition, 558 ; diagnosis, 566 ; etiology, 558 ; ocular phenomena in, 563 ; ophthalmic, 605-608, 707 ; pain in, 561 ; paralysis in, 564 ; pathology, 559 ; seton in, 567 ; symptoms, 564-566 ; treatment, 566 ; vasomotor phenomena in, 562 ; vertigo in, 564
Milk and tuberculous meningitis, 205, 224
Milkers' cramp, 680
Mimetic movements and the optic thalami, 111
Mimicry in idiocy, 878
Mind-blindness a term not to be used, 83 (note)
Mongolism, 877-881 (Fig. 26)
Mono-aesthesia, brachial, 69
Monomania, 996
Monoplegia, brachial, 63, 74 ; brachio-crural, 63 ; brachio-facial, 64, 74 ; centrum ovale and, 102 ; crural, 63, 74 ; facial, 64, 74 ; post-epileptiform, 57
Monospasm, in Jacksonian epilepsy, 57-61
Monro-Kellie doctrine of the cerebral circulation, 12
Moria, 49
Morphine, in insomnia, 799 ; in epilepsy, 503, 517 ; poisoning by, and insanity, 910
Morphinism, hypnotic treatment of, 1009
Motive and crime, 861, 865-869
Mucous disease and night-terrors, 816
Mumps and meningitis, 189
Murder and criminal lunacy, 1029-1032
Mutism, and aphasia, 408 ; hysterical, 420-422, 708 ; pseudo-, 419
Myasthenia, recurrent paralysis in, 610
Mydriasis, and lesions of the corpora quadrigemina, 121 ; and lesions of the crura cerebri, 117
Myelitis, bulbar, 157
Myoclonus, *see* Paramyoclonus, 649-659 ; M. epilepticus, 655
Myokimie, *see* Paramyoclonus, 649-659
Myospasia, *see* Paramyoclonus, 649-659
Myriachit, 619, 622, 625
Mysophobia, 864 ; in neurasthenia, 748 ; in tic, 625, 628
Mysticism in paranoia, 1003

Narcolepsy and night-terrors, 814
Neologism in paranoia, 1005
Nerve, third, in lesions of the corpora

quadrigemina, 120 ; of the crura cerebri, 117
Nerve-fibres and tracts, cerebello-rubral, 123 ; cingulum, 79 ; cortico-thalamic, 110 ; fasciculus longitudinalis superior, 41 ; fronto-pontine, 40 ; geniculate fasciculus, 103, 403 ; in general paralysis, 356, 357 ; inferior longitudinal, 436 ; lateral fillet, 119 ; lenticulo-caudate, 102 ; lenticulo-optic, 103 ; mesial fillet, 79 ; occipital, 101 ; pediculo-frontal, 101 ; pediculo-parietal, 101 ; Pick's, 403 ; posterior longitudinal, 128-129 ; pyramidal, 55-56 ; rubro-spinal, 123 ; short-longitudinal, 79 ; sphenoidal, 101 ; spino-cerebellar, 141 ; thalamo-cortical, 110
Neurasthenia, 727-791 ; age and, 743 ; agoraphobia in, 749 ; ametropia and, 743, 745, 750, 769, 785 ; anaemia and, 769 ; ankle-clonus in, 764 ; arterio-sclerosis and, 772 ; atony in, 756-757, 780 ; cardio-vascular form, 752 ; cerebral form, 744-751 ; claustrophobia in, 749 ; climate for, 780 ; definition, 727 ; diagnosis, 767-775 ; diet in, 786 ; disseminated sclerosis and, 771 ; drugs in, 783 ; dysuria in, 762, 770 ; electricity in, 782 ; etiology, 740-743 ; exercises in, 786 ; fatigue and, 730-732 ; fever in, 763 ; gastric symptoms, 757-759, 769 ; headache in, 745, 763, 785 ; heredity in, 737, 741, 753 ; hydrotherapy in, 782 ; hypnotism in, 783, 1011 ; hypochondriasis and, 772 ; hysteria and, 717, 735, 768 ; latent, 737 ; latent disease or, 773 ; malingering and, 774 ; marriage in, 789 ; melancholia and, 773 ; memory in, 746 ; movable heart in, 754 ; obsessions in, 747-750 ; occupation and, 738-741 ; pathology, 767 ; photophobia in, 751 ; post-operative, 766, 780 ; prognosis, 775 ; psychosis and, 735-736 ; reflexes in, 752, 764 ; sensation in, 751-752, 764 ; sex and, 742 ; sleep and, 733, 750, 785-786 ; spinal form, 751 ; suggestion in, 783 ; symptoms, 743 ; tests for, 729-732 ; theory of, 737 ; toxic causes of, 742 ; trauma and, 728, 762-767 ; treatment, 776-789 ; tropics and, 734 ; types of, 746 ; urine in, 759, 761, 770 ; uro-genital form of, 759-762 ; valerian in, 784 ; vertigo in, 750 ; visceral form of, 754-759 ; Weir Mitchell treatment in, 778-780
Neuritis and occupation-neuroses, 674, 678
Neuritis, optic, in abscess of the brain, 255, 260 ; in chlorotic cerebral thrombosis, 305 ; in lesions of the corpora quadrigemina, 122 ; in tuberculous meningitis, 217 ; in tumour of the brain, 271, 275
Neurocyme, 950
Neuroglia, the, in general paralysis, 354
Neuroma, causing epilepsy, 58
Neuroses, developmental, 928 ; and insanity, 825-827, 833-834
Neuroses, Occupation, *see* Occupation-neuroses, 667-686
Nightmare, 802, 805-806
Night-sweats, 810
Night-terrors, 804-820 ; adenoids and, 809, 816, 817 ; age and, 804, 806 ; asphyxia and, 809 ; aural vertigo and, 809, 816 ; bromides in, 818 ; day-terrors, 815-817 ; dreams and, 807 ; education and, 814 ; enuresis and, 811 ; epilepsy and, 817 ; idiopathic and symptomatic, 811, 819 ; mucous disease and, 816 ; narcolepsy and, 814 ; nightmares and, 805, 806 ; night-sweats and, 810 ; pain and, 810 ; somnambulism and, 819 ; symptoms, 814 ; timidity and, 812 ; treatment, 817 ; worms and, 811
Nodus cursorius, the, 108
Noguchi test, the, in syphilis, 957
Nose, the, disease of, and abscess of the brain, 253, 265
Nosophobia and neurasthenia, 772
Nostalgia, 973
Nucleus, amygdalae, 96 ; caudate, 43, 104-105 ; lenticular, 93, 105-108 ; oculo-motor, 124 ; pontine, 41 ; red, 119, 529
Nurses' contracture, 580, *see* Tetany, 578-604
Nutrition, the, in amaurotic family idiocy, 470 ; in cerebral abscess, 261 ; in cerebral tumour, 268, 278 ; in general paralysis, 373 ; in hysteria, 712 ; in melancholia, 976
Nystagmus, in alcoholic insanity, 915 ; in cerebellar lesions, 141, 148 ; in cerebral anaemia, 25, 30 ; in cerebral compression, 31 ; in head-nodding, 663-665 ; in hysteria, 705 ; in mongolism, 878 ; in polioencephalitis, 246-248 ; in thrombosis of the posterior cerebellar artery, 160 ; in tuberculous meningitis, 216

Obsessions, mental, 865-867 ; hypnotic treatment of, 1011 ; in neurasthenia, 747-750 ; in tic, 625
Occupation and melancholia, 965
Occupation-neuroses, 667-686 ; age and, 672 ; barbers', 680 ; bricklayers', 680 ; compositors', 680 ; counter-irritation in, 683 ; dancers', 681 ; diagnosis, 681 ; electricity in, 684 ; examination of patient in, 668-670 ; general diseases and, 675 ; goldbeaters', 679 ; hammer-men's, 679 ; heredity in, 672 ; massage in, 684 ; mental causes of, 675 ; milkers', 680 ; nerve tenderness in, 674, 680, 683 ; neuritis and, 674, 678 ; pianists', 677, 683 ; prognosis, 682 ; re-education in, 686 ; sawyers', 680, 681 ; sewers', 678 ; sex and, 671 ; skin-dressers', 681 ; tailor's, 679 ; telegraphists', 677 ; tic and, 675-676, 686 ; treatment, 682-686 ; tremor in, 673 ; trumpet-players', 681 ;

typewriters', 676 ; violinists', 678 ; waiters', 680; watchmakers', 681; writers', 670-676, 682, 684

Oedema in hysteria, 712 ; in sinus thrombosis, 304

Ophthalmia, neuro-paralytic, in extra-cerebellar tumour, 148; in pontine lesions, 129

Ophthalmoplegia, in cerebellar disease, 147 ; in cerebral palsies of children, 457, 460 ; in cerebral syphilis, 336, 340 ; in head-nodding, 665 ; in hysteria, 706-708 ; in lesions of the angular gyrus, 82-83 ; in lesions of the cerebellar peduncles, 152; in lesions of the corpora quadrigemina, 120, 124 ; in lesions of the crura cerebri, 117 ; in meningococcic meningitis, 174 ; in mongolism, 878 ; in polioencephalitis, 246 ; in torticollis, 629 ; in tuberculous meningitis, 215, 216 ; pseudo-nuclear, 83

Opisthotonos, and lesions of the corpora quadrigemina, 123 ; in meningitis, 176 (Fig. 14)

Opium in insomnia, 799

Opium-poisoning, diagnosis from cerebral haemorrhage, 330

Opsonic index, the, in meningococcic meningitis, 171 ; in tuberculous infections, 225

Optic radiations, the, 85-88, 103, 436

Optic thalamus, *see* Thalamus, the optic

Orbicularis symptom, the, 324

Orthopnoea and the circulation, 17

Otitis media and tuberculous meningitis, 221

Otorrhoea and abscess of the brain, 250-254 ; in meningitis, 175

Otorrhoea, cerebral, 256

Oxycephaly in idiocy, 881

Pachymeningitis in chronic progressive chorea, 549

Pachymeningitis haemorrhagica, 319 ; in general paralysis, 352 ; in cerebral paralyses of children, 455

Palsy, *see* Paralysis

Parageusia, 916

Paragraphia, 94, 441-443

Paraldehyde in acute delirium, 902 ; in insomnia, 798

Paralysis, acute epidemic, *see* Polioencephalitis, 241

Paralysis agitans, 473-482 ; age and, 474 ; aspect in, 479 ; course, 480 ; diagnosis, 480 ; duration, 480 ; emotions and, 475 ; etiology, 474 ; gait in, 479 ; hot flushes in, 478-480 ; mental changes in, 480 ; morbid anatomy, 475 ; parathyroid glands in, 475 ; pathology, 475-477 ; prognosis, 481 ; rigidity in, 478 ; senility and, 477 ; speech in, 477 ; treatment, 481 ; tremor in, 477-479

Paralysis, apoplectiform bulbar, 159 ; bulbar, distinction from pseudo-bulbar, 106 ; bulbar, in cerebral syphilis, 341 ; cortical motor, 57, 61-75 ; dissociated, 74 ; hephaestic, 679 ; in cerebellar disease, 148 ; in cerebral abscess, 257 ; in cerebral haemorrhage, 320-321 ; in cerebral occlusion, 296 ; in chorea, 540, 543 ; in general paralysis, 369 ; in hysteria, 700-704, 725 ; in intracranial tumour, 270, 273 ; in migraine, 564 ; in pontine haemorrhage, 126-130 ; in tuberculous meningitis, 215 ; labio-glosso-laryngeal, 157 ; oculomotor, *see* Ophthalmoplegia; pseudo-bulbar, 65, 106-108, 297, 400, 402-405 ; sudden nuclear, 124 ; supranuclear, 400, 402-405

Paralysis, Cerebral, in children, 452-467 ; abnormal labour and, 453 ; acquired, 459-467 ; athetosis in, 456, 460, 464 ; birth-palsy, 453-459 ; congenital, 453-459 ; convulsions in, 457, 461, 463, 466 ; deformities in, 457, 459, 460, 464 ; diagnosis, 457, 462, 466 ; diplegic, 459 ; epilepsy and, 464-465 ; etiology, 453, 461 ; gait in, 456, 460 ; haemorrhage and, 454-455 ; hemiplegic, 463 ; Little's disease and, 456 ; mental defect in, 454, 457, 458, 467 ; microcephaly, 467 ; pachymeningitis in, 455 ; porencephalus in, 455, 462 ; prognosis, 458, 462, 466 ; reflexes in, 456 ; rigidity in, 463 ; sex and, 453 ; specific fevers and, 459, 465 ; speech in, 457 ; symptoms, 455-457, 460; treatment, 458, 462 ; tremor in, 456

Paralysis, Recurrent, 604-614 ; aphasia in, 605 ; facial, 608 ; family periodic, 604 ; hemiplegia in, 606 ; in arterial disease, 609-610 ; in intermittent claudication, 611-612 ; in pseudotumor cerebri, 612 ; in Raynaud's disease, 609-611 ; in uraemia, 609 ; myasthenic, 610 ; oculomotor, 607, 610 ; ophthalmic migraine and, 605-608 ; sleep-ptosis, 608 - 609 ; syphilis and, 609, 612 ; third nerve, 607-608 ; treatment, 612

Paramnesia, alcoholic, 918

Paramyoclonus multiplex, 649-659 ; chorea and, 542, 651, 656 ; convulsions in, 652-655 ; cortical origin denied, 652 ; diagnosis, 656 ; epilepsy and, 650, 652, 655 ; etiology, 650 ; familial, 650 ; fibrillar contractions in, 655 ; heredity in, 650 ; history of, 649 ; hysteria and, 650-651, 657 ; pathology, 650 ; prognosis, 657 ; reflexes in, 653, 656 ; sensation in, 656 ; symptoms, 652 ; thyroid gland and, 651 ; tic and, 632, 656 ; treatment, 657

Paranoia, *see* Insanity, Systematised delusional, 995-1007 ; in epilepsy, 957

Paraphasia, 94, 427, 441-443

Parapraxia, 442-443, 449

Parathyroid glands, the, and tetany, 579, 580, 584-587 ; tuberculosis of, and tetany, 209-210

Parkinson's disease, *see* Paralysis agitans, 473-482

Pauper lunatics, 348, 1026
Peduncles, cerebellar, the, 123, 125 ; lesions of, 152-155
Pellagra and insanity, 910
Pericarditis and chorea, 538
Persecutés persecuteurs, 1001, 1003
Perseveration, 443, 451
Petit mal, epileptic, 500, *see also* Epilepsy ; hysterical, 697
Phobia, barbers', 680
Phosphenes, in migraine, 563 ; in tumour of the brain, 276
Phosphorus poisoning and insanity, 912
Photophobia, hysterical, 706 ; in concussion of the brain, 233, 235 ; in tuberculous meningitis, 212 ; neurasthenic, 751
Phrenalgia, 964
Phrenitis, *see* Delirium, acute, 899-904
Phrenocardia, 753
Phthisis, and insanity, 839, 939-941 ; *see also* Tuberculosis
Pianists' cramp, 677, 683
Pithiatisme, 776
Pituitary body, diseases of the, 162 ; urine in, 277 ; extract of, in tetany, 599
Plasma-cells in general paralysis, 354
Pli courbe (angular gyrus), 81, 87
Polioencephalitis superior acuta, 124, 241-250, 465 ; age and, 241 ; bacteriology, 242 ; cerebellar form of, 245, 247 ; diagnosis, 248 ; morbid anatomy, 244 ; prognosis, 249 ; symptoms, 245-248
Polioencephalo-myelitis, 242
Poliomyelitis, relation to polioencephalitis, 241-244
Polyopia, hysterical, 706
Pons Varolii, lesions of the, 125 - 134 ; haemorrhage, 326 ; tumour, and facial spasm, 639
Porencephaly, in children, 455, 462 ; in idiocy, 885 ; of the Rolandic area, 61
Pregnancy, and chorea, 525, 542, 544 ; and insanity, 930
Primiparity and insanity, 931-933
Protospasm, 57 - 61 ; brachial, 59 ; crural, 60 ; facial, 59
Psammoma in idiocy, 886
Pseudo-angina and -asthma in neurasthenia, 754
Pseudo-apoplexy in cerebral tumour, 273
Pseudo-general paralysis, 341 - 342, 378 · alcoholic, 914
Pseudo-glioma and meningitis, 175
Pseudo-mutism, 419
Pseudo-ptosis, 563
Pseudo-tetanus, *see* Tetany, 578-604
Pseudo-tetany, 579, 585
Pseudotumor cerebri, 612
Psychasthenia, 735
Psychology of crime, the, 1018-1023
Psychosis, and insanity, 827 ; and neurasthenia, 735
Psychosis polyneuritica, 365, 915, 919
Psychotherapy in neurasthenia, 776, 788
Ptosis, hysterical, 706 ; in lesions of the angular gyrus, 81 - 82 ; in lesions of the crura cerebri, 117 ; sleep-, 608, 609, 802
Puerperium, the, and insanity, 930-933
Pulse, the, in abscess of the brain, 255 ; in cerebral haemorrhage, 321 ; in meningococcic meningitis, 178 ; in tuberculous meningitis, 213-214 ; in general paralysis, 374
Pulvinar, lesions of the, 109-116
Pupils, the, in alcoholic insanity, 915 ; in cerebellar disease, 148 ; in chorea, 536 ; in general paralysis, 368 ; in head-nodding, 665 ; in hysteria, 707 ; in lesions of the angular gyrus, 80 ; in lesions of the corpora quadrigemina, 121 ; in lesions of the crura cerebri, 117 ; in meningococcic meningitis, 173 - 175 ; in neurasthenia, 745 ; in pontine haemorrhage, 126 ; in tuberculous meningitis, 216
Pyaemia and abscess of the brain, 251, 254, 262

Quadrilateral, Marie's, 397
Quinine in chorea, 546

Radiations, the optic, 85-88, 103
Raynaud's disease, recurrent paralysis in, 609-611
Réaction à distance, 55
Reception order, lunatic, 1042
Reflexes, the, in alcoholic insanity, 915 ; in birth-palsy, 456 ; in cerebellar tumour, 144 ; in cerebral haemorrhage, 298, 322, 325 ; in concussion of the brain, 235 ; in cerebral paralyses of infants, 456, 460 ; in chorea, 534 ; in cretinism, 879 ; in general paralysis, 370 ; in hemiplegia, 298 ; in hysteria, 701, 704 ; in lesions of the corpora quadrigemina, 123 ; in lesions of the optic thalamus, 113 ; in lesions of the pulvinar, 113 ; in neurasthenia, 752, 764 ; in paramyoclonus, 653, 656 ; in tuberculous meningitis, 214 ; in tumour of the brain, 268, 273
Religion and insanity, 835, 846, 1003
Respiration and the cerebral circulation, 16-17
Responsibility, in criminal lunacy, 1033-1040 ; in insanity, 869, 872
Rest in melancholia, 981
Restraint, mechanical, of lunatics, 1044
Rheumatism, and chorea, 521-524 ; and insanity, 840
Rhinorrhoea, cerebrospinal, 8
Rigidity, decerebrate, 123
Rigidity, hemiplegic, 298, 322, 328, 463 ; and irritation of the fronto-parietal fibres, 102 ; in intracranial tumour, 274 ; in paralysis agitans, 478 ; in tuberculous meningitis, 215

Risus caninus *vel* sardonicus in facial spasm, 642
Rolandic area, disease of the, 51-76 (Figs. 7-10); corpus callosum and, 98; destructive lesions of, 56, 61-75; irritative lesions of, 57-61; Jacksonian epilepsy and, 57-61; sensation and, 60, 66-75
Rolando, tubercle of, 158

St. Vitus's dance, *see* Chorea, 519-548
Salaam convulsions, in eclampsia nutans, 659; in tic, 627
Salt-free diet in epilepsy, 958
Sanity and insanity, 824
Satyriasis and general paralysis, 380
Sawyers' cramp, 680, 681
Scaphocephaly in idiocy, 884
Schlesinger's sign in tetany, 592
Scotoma, in epilepsy, 499; in hysteria, 707-708; in migraine, 563
Scrofulides and tuberculous meningitis, 206, 223
Secretary of State's lunatics, 1026
Self-indulgence and insanity, 849-851
Self-restraint and insanity, 853-857
Senility and insanity, 935-939
Sensation, Impairment of, absent in cerebellar disease, 149; in alcoholic insanity, 916; in bulbar paralysis, 106; in cerebral haemorrhage, 324; in cerebral tumour, 270, 275; in corpora quadrigeminal lesions, 123; in chorea, 533; in facial spasm, 644; in falciform lesions, 76-79; in frontal lesions, 43; in general paralysis, 359, 368-369; in gyrus fornicatus lesions, 76-79; in internal capsule lesions, 103-104; in hereditary chorea, 552; in neurasthenia, 751-752, 764; in hysteria, 697-699; in optic thalamus lesions, 113; in paramyoclonus, 656; in parietal lesions, 79-84; in pontine lesions, 127-129, 131-132; in pseudo-bulbar paralysis, 106; in Rolandic lesions, 66-75; in temporal lesions, 93; in tuberculous meningitis, 216; in tumour of the brain, 270, 275
Sense, spatial, 71
Senses, the special, in lesions of the crura cerebri, 118; in lesions of the optic thalamus, 114
Sensibility, muscular, in lesions of the parietal lobe, 80, 84; in lesions of the Rolandic area, 70, 74; in pontine lesions, 132
Sensibility to pain, 76
Sensibility, stereognostic (*see also* Astereognosis), in lesions of the parietal lobe, 80; in lesions of the Rolandic area, 67, 70-74
Sensibility, tactile, 76; in lesions of the Rolandic area, 60, 66-75
Sensibility, thermal, in cortical lesions, 73, 77
Sensori-motor functions of the Rolandic area, 66-75
Septicaemia and insanity, 909, 922, 931
Serum-treatment of meningitis, the, 183
Sewing cramp, 678
Sexual aberrations, and criminal lunacy, 1019; and general paralysis, 349; and idiocy, 883; and insanity, 836-837, 929; and mania, 893, 894, 896; and senility, 937; and stupor, 985; and vice and crime, 851, 853, 856, 863, 866, 873; hypnotism in, 1011
Shoemakers' spasm, *see* Tetany, 578-604
Sign *or* Symptom, Babinski's, 703, 717; Bruns', 159; Chvostek's, 592; Erb's, 592, 598; Flora's, 774; Kernig's, 177, 199, 214, 222; Schlesinger's, 592; Stocker's, 220; Trousseau's, 591, 596
Sinus phlebitis and cerebral abscess, 259
Sinus thrombosis, 303-307
Skin-dressers' cramp, 681
Sleep (*see also* Insomnia), and the cerebral circulation, 21; and cerebral compression, 30-31; cerebral haemorrhage in, 293, 310; and epilepsy, 505; hypnotic, 1012; and general paralysis, 374
Sleep, disorders of, 791-804; amount of sleep required, 792; bromides in, 797-798; causes of, 793; diet in, 794-796; drugs in, 796-801; electricity in, 801; habits and, 794; hypnotics in, 797-801; hypnotism in, 802; insomnia, 792-803; irritation and, 793; nightmare and, 802; pathology, 794; physiology, 791; somnolence and, 802; toxic causes of, 793; treatment, 794-803
Sleep-ptosis, 608, 609, 802
Smell, sensation of, 95, 103; in general paralysis, 369; in lesions of the optic thalamus, 115
Softening of the brain, in general paralysis, 352; red, 292, 294; yellow, 294
Solitary confinement and insanity, 832
Sommeil provoqué in hypnotism, 1012
Somnambulism, 692, 803, 819
Somniloquy, 802
Somnolence, 802, 814
Sparteine poisoning and insanity, 911
Spa-treatment of cerebral syphilis, 345
Spasm, and cerebral anaemia, 24-26; cynic, 642; histrionic (facial), 638; mimic (facial), 638; nodding, *see* Eclampsia nutans, 659, *and* Head-nodding, 661-667
Spasm, Facial, 638-649; chorea or, 644; definition, 638; diagnosis, 644; electricity in, 647; emotions and, 639; etiology, 638-640; facial nucleus and, 640; heredity in, 638; hysteria or, 644; organic disease in, 645; pathology, 640; prognosis, 645; reflex irritation and, 639, 640; sensation in, 644; sex and, 638; stapedius in, 643; symptoms, 640-644; tic and, 619, 639-640; treatment, 646-648
Spasm, mobile, 112, 299, 456, 460

Speech (*see also* Aphasia), in alcoholic insanity, 915; impediments of, 569-578; in cerebellar disease, 148-149; in cerebral paralyses of children, 457; in dysarthria, 401; in general paralysis, 370; in paralysis agitans, 477
Speech-apraxia, 386
Speech-centres, *see* Centres
Spes phthisica, 939, 975
Sphincters, the, in cerebellar lesions, 141; in cerebral haemorrhage, 322
Spider-cells in general paralysis, 354
Spinal cord, the, in general paralysis, 353, 355; in meningococcic meningitis, 169
Spine, the hysterical, 710
Splanchnic area, the, and the cerebral circulation, 11-20
Spring-halt tic, 620
Stammering, 569; and aphasia, 400
Stapedius, the, in facial spasm, 643
Status epilepticus, the, 506, 517; cerebral congestion in, 29; in general paralysis, 371, 384
Status hemicranialis, the, 567
Status hystericus, the, 695
Stereognosis, 67, 70-74, 80, 275
Stertor in cerebral haemorrhage, 321
Stigmata, criminal, 1022; hysterical, 688, 698, 718; saintly, 712
Stocker's sign, 220
Strophanthin and the cerebral circulation, 11
Stupidity, in lesions of the corpus collosum, 100; in syphilis of the brain, 343; in pseudo-bulbar paralysis, 106
Stupor, 984-987, 990; anergic, 903, 990; delusional, 990; in gas-poisoning, 907; katatonic, 986
Stuttering, 569
Subarachnoid space, the, 5
Subdural space, the, 5
Substantia nigra, the, 116
Suggestion, 1007-1017; dangers of, 1014; in dipsomania, 1009; in drug habits, 1009; in hysteria, 691, 1011; in insomnia, 1008; in morphinism, 1009; in neurasthenia, 783, 1011; methods, 1013; results, 1014-1017; theories, 1012
Suicide, in melancholia, 969, 974-975; in paranoia, 1001; in puerperal insanity, 931
Sulphonal in insomnia, 798
Sunstroke and general paralysis, 351, 379; and idiocy, 876; and insanity, 837
Suprarenal extract and the cerebral circulation, 11-12
Sydenham's chorea, 519-548
Syncope, and the cerebral circulation, 16-20; epilepsy or, 510
Syndrome, Bristowe's, 99-100; Bruns', 159; of the corpus striatum, 109; Homén's, 107, 109; Korsakow's, 365, 915, 919; thalamic, 116, 325; Weber's, 117
Synostosis, premature cranial, in idiocy, 864, 887
Syphilis, and cerebral occlusion, 295; and cerebral paralyses in children, 453, 460; and Jacksonian epilepsy, 58; and general paralysis, 349-351, 378, 957; and idiocy, 875; and insanity, 836; and meningitis, 193-197 (Figs. 17-19); and recurrent paralysis, 609, 612
Syphilis, Inherited, and adolescent general paralysis, 364; and meningitis, 195
Syphilis, Intracranial, 334-346; age and, 335; aphasia in, 338; arterial disease in, 338; bulbar palsy in, 341; cerebellar, 339; convulsions in, 339; dementia in, 337, 341-343; epilepsy and, 339-340; etiology, 334; gumma in, 339, 343; haemorrhage in, 339; headache in, 337; hemiplegia in, 336, 338; injury and, 335; insomnia in, 337; iodides in, 345; meningeal lesions in, 193-197 (Figs. 17-19), 340; mental changes in, 337, 342-343; mercury in, 344; nerve palsies in, 341; onset, 334; ophthalmoplegia in, 336, 340; prodromes, 336-337; prognosis, 343; pseudo-general paralysis and, 341-342; ptosis in, 342; stupor in, 343; symptoms, 336; treatment, 344-346; vertigo in, 337; Wassermann's reaction in, 342
Syphilophobia and general paralysis, 350
Systematisation in paranoia, 996, 998

Tache cérébrale in general paralysis, 374; in meningism, 199; in tuberculous meningitis, 212
Tailors' cramp, 679
Taste, sensation of, 96, 97, 103; in pontine lesions, 129; in tumour of the brain, 277
Tegmentum cruris, lesions of the, 116-119
Tegmentum pontis, 119, 125
Teichopsia in migraine, 563 (Pl. II.)
Telegraphists' cramp, 677
Temperature, regulation of, and the corpus striatum, 108; and optic thalamus, 115
Tentorium cerebelli, the, 6; intracranial pressure and, 32
Testamentary capacity in lunacy, 1047-1049
Tetanilla, 585, *see* Tetany
Tetanus, intermittent, pseudo-, remittent, or rheumatic, *see* Tetany
Tetany, 578-604; acute fevers and, 581; age and, 580; calcium metabolism in, 210, 579, 587, 595, 601; carpopedal contractions and, 578-579, 583; death in, 598; diagnosis, 595-598; dilated stomach and, 580, 591, 598-600; distribution, 582; electrical reactions in, 592; epidemic, 581; ergotism and, 579, 581; etiology, 580; hysteria or, 598; idiopathic, 581, 582; in adults, 580-582; in children, 583; meningitis or, 598; mental changes in, 595; morbid anatomy, 583; muscular spasm in, 588-592; occupation and, 581; pain in, 593; para-

thyroids and, 209-210, 579, 580, 584-587 ; paroxysmal nature of, 590 ; pathogeny, 584-588 ; pituitary extract in, 599 ; pregnancy and, 580, 587 ; prognosis, 598 ; pseudo-tetany, 579, 585 ; reflexes in, 594 ; rickets and, 579, 588 ; sensation in, 588, 593 ; symptoms, 588-595 ; synonyms, 578 ; tetanus or, 597 ; thyroid gland and, 579, 581, 585 ; toxins and, 585-588 ; treatment, 599-602 ; true, 579 ; urine in, 585, 588, 595 ; vasomotor changes in, 594

Thalamus, optic, 93, 98, 109-116 ; haemorrhage into, 110, 115, 325 ; is inexcitable, 104 ; mimetic movements and, 111 ; motion and, 110 ; post-hemiplegic chorea and, 112 ; sensation and, 113 ; special senses and, 114 ; thermotaxis and, 115

Thermotaxis and the corpus striatum, 108 ; and the optic thalamus, 115

Thrombosis, cerebral, 329 ; of the posterior cerebellar artery, 160 ; pontine, 126

Thrombosis, cerebral, arterial, 293-296 ; chlorotic, 305 ; infective, 305 ; of sinuses, 303-307 ; venous, 303-307

Thyroid gland, in cretinism, 880 ; in paramyoclonus, 651 ; in tetany, 579, 581, 585

Thyroidectomy and tetany, 581

Tic douloureux not a tic, 614

Tics, the, 614-638 ; age and, 617 ; arsenic in, 634 ; atropine in, 635 ; blepharospasm, 617 ; classification of, 618-619 ; conium in, 635 ; convulsive, 619, 621-626 ; co-ordinated, 619, 626, 633 ; definition, 614 ; diagnosis, 631-633 ; etiology, 616, 618 ; exclamatory, 624 ; facial spasm and, 619, 638-649 ; habit in, 616, 617 ; habit-spasm and, 615, 619 ; heredity in, 616, 629 ; hysteria and, 621, 626, 632 ; *idées fixes* in, 625, 626 ; imitation and, 617 ; in sleep, 624 ; latah, 619, 625 ; mannerisms and, 616 ; mental infantilism and, 616 ; mental obsessions in, 625 ; myriachit, 622, 625 ; non-douloureux, *see* Spasm, facial, 638-649 ; paramyoclonus and, 632, 656 ; pathology, 618 ; prognosis, 633 ; psychical, 619, 627 ; psychosis in, 618 ; salaam, 659, saltatory, 619, 630 ; sex and, 616 ; simple, 619-621 ; spasm or, 614 ; spring-halt, 620 ; Sydenham's chorea or, 617, 631 ; symptoms, 618-631 ; tic douloureux and, 614 ; torticollis, 619, 629, 635 ; treatment, 630, 634-636 ; writers' cramp and, 675-676

Tinnitus in cerebellar disease, 148, 277 ; in intracranial tumour, 276-277

Tone-deafness, 94

Torcular Herophili, the, blood-pressure in, 4, 10

Torticollis tic, 619, 629, 635

Training of idiots and imbeciles, 887-891

Traube-Hering blood-pressure curves and the cerebral circulation, 25

Trephining, in epilepsy, 517 ; in intracranial tumour, 286-288

Treponema pallidum, 195

Trional in insomnia, 799

Trismus, and lesions of the cortex, 66 ; in medullary lesions, 159 ; in tetany, 590

Tremor, in birth-palsy, 456 ; in bulbar paralysis, 106 ; in cerebellar lesions, 141 ; in frontal tumour, 47 ; in Homén's syndrome, 108 ; in hysteria, 705 ; in infantile hemiplegia, 464 ; in lesions of the corpora quadrigemina, 123 ; in lesions of the crura cerebri, 118 ; in lesions of the optic thalami, 112 ; in paralysis agitans, 477-479 ; in pontine lesions, 132 ; in tuberculous meningitis, 215 ; in tumour of the brain, 274

Tremor, convulsive, *see* Paramyoclonus, 749-759

Tremor, intention, in birth-palsy, 456 ; in cerebellar lesions, 141, 142 ; in dysarthria, 402 ; in hysteria, 705 ; in infantile hemiplegia, 464 ; in lesions of the corpora quadrigemina, 123 ; in lesions of the optic thalamus, 112 ; in neurasthenia, 763

Trousseau's sign in tetany, 591, 596

Tuber cinereum, the, and thermotaxis, 115

Tuberculides in tuberculous meningitis, 206, 223

Tuberculin in tuberculous meningitis, 225

Tuberculosis, and idiocy, 875, 878, 885-886 ; and insanity, 204, 839, 939-941, 975 ; choroidal, 217 ; meningeal, *see* Meningitis, Tuberculous, 201-228 ; of the crura cerebri, 117 ; of the brain, calcified, 266 ; of the paracentral lobule, 63

Tumour, cerebellar, 123, 136, 143-151 ; extracerebellar, 143 ; intracerebellar, 143 ; medullary, 146

Tumour, Intracranial, 266-290 ; age and, 267 ; and general paralysis, 379 ; apoplectiform attacks in, 273 ; ataxia in, 275 ; convulsions in, 270, 272, 274 ; diagnosis, 223, 279-282 ; etiology, 266 ; headache in, 270 ; hearing in, 276 ; hemianopsia in, 276 ; localisation of, 268-270, 274 ; mental changes in, 272 ; morbid anatomy, 266 ; nutrition in, 278 ; optic neuritis in, 271, 275 ; paralysis in, 273 ; pathological physiology, 267 ; perimetry in, 271 ; phosphenes in, 276 ; prognosis, 282 ; reflexes in, 268, 273 ; secondary deposits from, 266 ; sensation in, 270, 275 ; sex and, 267 ; sphincters in, 274 ; subtentorial, 273 ; symptoms, 268 ; taste and smell in, 277 ; tentorium and, 268 ; treatment, 282-289 ; tremor in, 274 ; types of, 278 ; urine in, 277 ; vertigo in, 272 ; vomiting in, 272

"Turns" or "sensations" in minor epilepsy, 500

Typewriters' cramp, 676

Uncinate fits, 96, 264, 277
Uncus, disease of the, 96 ; epilepsy and, 264, 277
Uraemia, diagnosis from cerebral haemorrhage, 330 ; recurrent paralysis in, 609
Urgency order in lunacy, 1040, 1045
Urine, the, in chorea, 538 ; in general paralysis, 374 ; in mania, 895, 897 ; in hysteria, 694, 696, 705, 713 ; in neurasthenia, 759, 761, 770 ; in pituitary disease, 277 ; in tetany, 585, 588, 595 ; in tumour of the brain, 277

Vaccine treatment in meningitis, 183
Valerian in neurasthenia, 784
Vasomotor changes in lesions of the optic thalamus, 115
Venery and general paralysis, 349
Verbigeration, 987 ; post-epileptic, 955
Veronal in insomnia, 799
Vertigo, in cerebellar disease, 147, 150, 277 ; in cerebral syphilis, 337 ; in epilepsy, 496, 501, 510 ; in migraine, 564 ; in neurasthenia, 750 ; in night-terrors, 809, 816 ; in tumour of the brain, 272
Vice, Crime, and Insanity, 842-874 ; alcoholism and, 856-857 ; aversions in, 864 ; cleptomania and, 873 ; conduct and, 842 ; crime, 857 ; custom and, 845-851 ; cyclical, 874 ; definitions of, 842-851 ; desire and, 857, 863-864 ; impulse and, 858 ; intention and, 860, 865-869 ; motive and, 861, 865-869 ; obsession and, 865-867 ; paroxysms of, 856-857, 874 ; religion and, 846 ; responsibility in, 869 ; self-indulgence and, 849-851 ; self-restraint and, 853-857 ; sexual aberrations and, 851, 853, 856, 863, 866, 873 ; treason and, 844, 849, 851 ; vice, 849-851 ; vice and insanity, 851-857 ; will and, 858 ; wrong-doing and, 847-851 ; wrong-doing and insanity, 857
Violinists' cramp, 678
Visceroptosis and neurasthenia, 754-759
Vision (*see also* Amblyopia), in alcoholic insanity, 916 ; in hysteria, 706-708 ; in lesions of the corpora quadrigemina, 121 ; in lesions of the optic thalamus, 114
Visuals and auditives, 389, 391, 426, 430, 434
Vocal cords, the, in aphasia, 65-66; in medullary lesions, 158
Voices in paranoia, 998-999
Vomiting, in cerebellar haemorrhage, 135 ; in cerebellar tumour, 150 ; in hysteria, 709, 725 ; in meningitis, 173, 213 ; in tumour of the brain, 272

Waiters' hand, 680
"Walnut brain," 461-462
Warnings in epilepsy, 494-500
Wassermann's reaction, in cerebral syphilis, 342 ; in general paralysis, 381, 957
Watchmakers' cramp, 681
Weather, influence of, on cerebral haemorrhage, 310
Weber's syndrome, 117
Weichselbaum's diplococcus, 171
Weir Mitchell treatment, the, 722, 778-780
Wernicke's aphasia, 385 ; hemiopic pupillary reaction, 89 ; zone, 397, 411, 415, 451
Whooping-cough, and idiocy, 875 ; and insanity, 839
Will, the, and insanity, 858
Willis, the circle of, and the cerebral circulation, 10, 25, 26
Wills and testamentary capacity, 1047-1049
Witzelsucht, 49
Word-blindness, 90, 412, 424-443, 450 ; and lesions of the angular gyrus, 83, 84 ; and lesions of the occipital lobe, 87, 89, 450-451 ; cortical, 451 ; in cerebral abscess, 264 ; Marie's views on, 397 ; pure or subcortical, 388, 431
Word-centres, *see* Centre ; defects of, and aphasia, 424-443
Word-deafness, 84, 412, 424-443, 450 ; and lesions of the temporal lobe, 90-97 ; Marie's views on, 397 ; pure, 437
Word-images and aphasia, 391
Word-memory and aphasia, 387 (Fig. 20), 396
Worms and night-terrors, 811
Worry and insanity, 835
Writers' cramp, 670-676, 682, 684
Writers'-sand tubercles, 207
Writing, defects of, and word-centre lesions, 424-443
Writing centre, 412
Wrong-doing, vice, crime, and insanity, 847-851, 857

x-rays, the, in intracranial tumour, 283

Zinc salts, in chorea, 546 ; in epilepsy, 516
Zone, Wernicke's, 397, 411, 415, 451
Zones, epileptogenous, 951 ; hystero-genetic, 700
Zoopsia, in hysteria, 696 ; in neurasthenia, 763

END OF VOL. VIII

Printed by R. & R. Clark, Limited, *Edinburgh*.

PRESS NOTICES OF THE SECOND EDITION OF A SYSTEM OF MEDICINE

VOLUME I

LANCET.—"Considerable alterations have been made in the arrangement of the subjects dealt with in this volume, several new articles have been added, others have been transferred to later volumes, and yet others have been modified or extended, with the result that it is larger than its predecessor by over two hundred pages. . . . As a whole, the work is one of which the profession of medicine in this country may well be proud, and one which, like its predecessor, will be of great value to the investigator and the practitioner who desires to keep himself informed of the advances in medical knowledge and to have the current state of that knowledge conveniently summarised."

NATURE.—"Altogether this volume commands admiration, and if its high standard be maintained, as it doubtless will be, in the succeeding volumes, this *System of Medicine* will form a lasting monument of the high place which British medicine holds at the present time."

VOLUME II.—PART I

BRITISH MEDICAL JOURNAL.—"Some of the articles have only been revised and brought up to date by their authors, others have been entirely rewritten, while a certain number are quite new. The same thoroughness of treatment characterises them all. . . The new instalment of the *System* will thus be seen to be as full and complete a work of reference as the most expert writers and the most discriminating of editors can make it. . . . A work which presents a trustworthy compendium of all that is really known upon the subjects with which it deals."

MEDICAL CHRONICLE.—"The whole volume is most excellent."

VOLUME II.—PART II

BRITISH MEDICAL JOURNAL.—"The list of contributors, thirty-three in number, contains the names of so many eminent authorities that the different articles should represent the standard of our knowledge of them at the present day. . . . We should have liked to mention some of the articles throughout the volume in detail, but space will not permit; it is sufficient to say that the hope of the editors that the volume will serve as a complete work on tropical medicine has been realised; it should, in fact, become the standard of the subject in the English language, both as a book of reference and for study."

EDINBURGH MEDICAL JOURNAL.—"The editors are to be congratulated on the production of an excellent work, which, containing in a compact form such a mass of information with copious references to the literature on the various subjects, should prove invaluable not only to students of tropical medicine, but to practitioners at home and abroad."

PRESS NOTICES—*continued.*

VOLUME III

LANCET.—"The present volume well maintains the credit gained by those which have previously appeared, and there is every indication that the new *System* will retain the place secured by the former edition as the most popular standard medical text-book in this country."

BRITISH MEDICAL JOURNAL.—"The volume in every way maintains the high standard which has characterised this publication throughout."

VOLUME IV.—PART I

LANCET.—"We once more congratulate the editors on the success of their labours. The present volume is a valuable addition to the *System of Medicine.* It contains much new matter, and will serve as a most excellent work of reference."

BRITISH MEDICAL JOURNAL.—"Limits of space prevent our mentioning all the new articles, but we would refer our readers to this volume with great satisfaction as being a perfectly adequate presentment of all the subjects with which it deals."

VOLUME IV.—PART II

LANCET.—"An extremely creditable volume. . . . Well up to the high standard set by the rest of the work."

VOLUME V

LANCET.—"The whole volume maintains the high standard which we are accustomed to expect in our leading medical text-books. In almost all instances a careful and thorough survey of medical literature, both of our own and of foreign countries, has been made, and valuable bibliographies are appended to the articles."

BRITISH MEDICAL JOURNAL.—"A volume which cannot but enhance the high reputation deservedly earned by the previously-issued portions of the new edition of the *System.*"

VOLUME VI

BRITISH MEDICAL JOURNAL.—"A volume which is in all respects representative of the latest knowledge and experience at home and abroad."

LANCET.—"The present volume contains a vast amount of information in connection with diseases of the heart and blood-vessels. As a separate work of reference it is one of great value, while it forms a worthy addition to the *System,* which now holds a high place in medical literature upon which Sir Clifford Allbutt and Dr. Rolleston may be heartily congratulated."

VOLUME VII.

MEDICAL TIMES.—"With such an array of contributors, all of the front rank in their several spheres of work, it goes without saying that the volume before us worthily represents this particular branch of British Medicine."

MACMILLAN AND CO., Ltd., LONDON.

KING'S COLLEGE LONDON

KING'S COLLEGE HOSPITAL
LIBRARY
Medical School